Dipole Neurology: An electromagnetic multipole solution to brain structure, function and abnormality.

Felix Lanzalaco (a), **Wajid Zia.** (a,b)

(a) Psychology Cares Scotland, 142 Brunton Gardens, Edinburgh EH7 5ET, Scotland
(b) Dept of Clinical Psychology, University of Edinburgh, Scotland.

Correspondence to: Felix Lanzalaco, Psychology Cares Scotland, *142 Brunton Gardens, Edinburgh EH7 5ET, Scotland.*
Tel: +44 (0) 141 639 9722
E-mail address: Lanzalaco@Dipoleneurology.org.uk

This is an exploratory paper which proposes that natural selection optimizes the evolution of mechanisms to facilitate brain wide electromagetic coherence in development. The emphasis is on attempting to understand the composite brain structure and their interactions rather than any of its single parts in isolation. An Electromagnetic Multipole Solution (EMS) model then proposes an explanation for why the cortex has features such as cortical folds and hemispheres, and the reverse lateralization of processing, which occurs in these hemispheres. The mammalian brain is conceptualised in terms of a dipole structure for the cortex (Cortical EMS) and a linear quadrupole for the limbic system (Limbic EMS). Cortical grey matter is reviewed and it is determined that to have chemical distributions consistent with the structure of a dipole system. It is hypothesised that magnetic interactions at the cortex surface form stripe domains which explain its sulci and gryi. The limbic system is similarly explored and proposed to possess the structure of a harmonically oscillating linear quadrupole. At the point of interaction between the poles of the Cortical and Limbic EMS an S-shaped twist can theoretically explain the form of the hippocampus. From a systems view Limbic EMS tends towards collapse, entwined inside a radiating cortex dipole which tends towards expansion. To elaborate on this interaction further, a 'collapse-expansion' paradox and the recurrent cycle between these two systems is contended to an engine for mammalian consciousness. We propose that a CorticoLimbic EMS model will aid with understanding the brain's overall structure, neurodevelopmental stages and the evolution of its present electromagnetic functions.

Keywords
Systems neuroscience
Corticogenesis
EMS
Neurodevelopment

Sections

ABBREVIATIONS

5HT	5-hydroxytryptamine
AA	Arachidonic acid
AC	Alternating current
apoE	Alipoprotein E
BOLD	Blood-oxygen-level dependent
Cl-	chlorine
cPLA2	Cystolic Phospholipase platelet A2
CSF	Cerebrospinal fluid
Cx	Connexin
DAT	Dopamine active transporter
DHA	Docosahexaenoic acid
EEG	Electroencephalography
EMS	Electromagnetic Multipole Solution
EPA	Eicosapentaenoic acid
EPSP	Excitatory Post Synaptic Potential
ERD	Event related desynchronization
ERP	Event related potentials
fMRI	Functional magnetic resonance imaging
GABA	gamma-Aminobutyric acid
GCP	Glutamate carboxypeptidase
iPLA2	Calcium independent Phospholipase platelet A2
IPSP	Inhibitory Post Synaptic Potential
LCH	Left Cerebral Hemisphere
LC PUFA	long-chain polyunsaturated fatty acids
LTP	Long-term potentiation
Na+	Sodium
NAAG	N-acetyl-alpha L-aspartyl-L-glutamate
NAAMF	Neuronal Activity Associated Magnetic field
NMDA	N-methyl-D-aspartic acid
PGE2	prostaglandin E2
PIP2	phosphatidylinositol-4,5-bisphosphate
PKC	phospholipid-dependent protein kinase
PLA2	phospholipase platelet A2
RCH	Right Cerebral Hemisphere

INTRODUCTION

This review sets out to amalgamate two mysteries in neuroscience and contends that they may have one common explanation. It offers a novel theoretical and conceptual framework from which the brain structure, function and abnormality can be examined and perhaps understood. Given the advances in the study of the brain structure over the last century the question of *what* (definitions of functions) *where* (neuro-anatomical localisation) and *how* (the processes by which a cause exert itself) have been examined methodically and systematically, yielding huge advances in recent years (Birren and Schaie, 2006). In contrast the question of *why* (causal determinates of the brain structure) has been largely left unexplored. This very question is the starting point from which the composite brain structure and their interactions are evaluated and conceptually organised.

1. Why does the brain look the way it does, with its cortical surface features, symmetry, split hemispheres and semi complete discs of the limbic system?

2. Why do, the cortex hemispheres give rise to reverse processing and behaviour patterns, and why are these consistent across humans and other mammals?

Autopsy data (Williams and Gluhbegovic 1992) shows striking observable similarities between cortex field lines and images from dipole simulators. (Figure 1) A dipole is a separation of two opposite electromagnetic charges that converge towards poles at either end. For example a bar magnet, a water molecule, and even celestial bodies are shown to be an oscillating electrical dipole emitting electromagnetic energy (Ferronsky, 1981). Similarly the entire cortex radiates an electromagnetic field, (McFadden, 2002) and its functions are defined primarily in electromagnetic terms such as spikes, EEG and Wave propagation. Arguably this gives raise to a critical question. Are the cortical visual similarities to a dipole reflected in these electromagnetic processes? In section 1 of this paper evidence will be considered which suggests that cortical hemispheres give rise to reversals in processing, behaviour patterns, and neurochemical distributions that can be explained by the opposite charges of a dipole. Further sections (2,3 and 4) concentrate on using this approach to examine other brain structures. For example the limbic system will be examined as an inverted variation of a dipole, termed a 'linear quadrupole'; the cortex sulci and gyri in terms of magnetic domains, and finally whether the interactions of these structures sheds light on current mysteries in conscious processing.

Background and History

Considering that a dipole like field line structure is apparent from autopsy data (figure 1) there is surprisingly no documented precedent for a cortex dipole hypothesis. Multiple factors which may have contributed to this omission. Arguably chief among them is that the current approaches in the research of brain structure have increasingly been underpinned by questions of what, where and how rather than why. This has produced huge advances in the medical sciences and led to highly effective but invariably fragmented approaches and methods. To propose the hypothesis that these cortically apparent field lines could be linked to the opposite charges or poles of a dipole structure required a link to neural processing. This link has only been available since Roger Sperry brought the opposites of lateralized brain processing to light in the 1970's (Levy and Sperry, 1971). That discovery resulted in a popular explosion of research into lateralization of brain processing. e.g. (Finke and Bettle, 1996; Springer and Deutsch, 1993; Fink et al., 1997; Vallortigara et al., 2004). At this time the paradox of opposite processing from either hemisphere could have been linked with the cortex's observable dipole features. It could have been conceived that these processing opposites were equivalent to some kind of positive and negatively charged distribution of processing re-constituted in the structure of either hemisphere. However this would have been a difficult concept to propose in terms of evidence. Now that thousands of studies describe almost every physiological process of grey matter content within the hemispheres at the neuron level, (and where in the brain hemispheres these processes occur) it is possible to attempt a re-assembling of these pieces into a larger electromagnetic framework for such a proposal.

Central Hypothesis

It is proposed that the left and right cortical hemispheres correlate with the negative and positive poles of a biomolecular dipole and these poles occur at the temporal regions. A Negative pole at the left temporal regions and a positive pole at the right temporal region. This model will be reffered to as "Cortical EMS". (Electromagnetic Multipole solution) Other significant structures of the brain such as the limbic system, cortex surface as well as the entire content within these structures will also be simplified with this electromagnetic approach to brain structure. The limbic system model will be referred to as "Limbic EMS" model. The entire model will be refered to as "CorticoLimbic EMS".

It is important to bear in mind then that many parts of this paper attempt to understand the brain by starting with physics based observations of the brain structures to generate some of the hypotheses, as there is simply a lack of known mechanisms to explain these observable phenomena. These are intended purely as hypothetical starting points rather than definite conclusions. Images will be used in the parts of the paper where an observational premise is necessary. With the amount of neuroscientific data now available it is possible to then proceed to consider if these physical observations are consistent with existing data or not. In Section 1: Cortex as a Dipole, a series of features (some visual) that are commonly known to dipoles will be compared with cortical features and functions known from neuroscience research. Such a cortical dipole hypothesis will propose that the entire cortical grey matter content is consistent with a dipole structure. Neurotransmitters, receptors, ions, neuromodulators and associated processes lateralize into opposite hemispheres, in a manner consistent with the separation of charges in a dipole. It should be borne in mind that the images in figure 1, are of magnetic dipoles. The Cortical EMS model proposes that magnetic dipole field lines arise from cortical white matter and electric dipole processes occur in cortical grey matter. These points are discussed at the end of Section 1.

To summarize what is attempted here in terms of these electromagnetic models.

- Determine if the theory is consistent with known research in the area of brain function such as EEG.
- Review the research in neurochemical and grey matter distributions to see if they are consistent with the proposed, cortical (EMS) model.
- To discover what does not agree with this model, and see what this tells us.
- To visually remodel the brain structure using the basic components of electro-magnetic structures.
- To provide some new concepts for how the brains electromagnetic field is generated and maintained.
- To explore if viewing the brain this way can give new insights into its larger complex system interactions.

Section 1: Cortex as a Dipole.

1.1 Method to simplify brain structure analysis

The proposed cortical dipole differs from standard dipoles in many ways. The brain has an electromagnetic structure that is different from any known multipole thus far explored. For example the corpus callosum possesses static rather than changeable field lines (figure 1) reconstituted in proteins and neurochemicals. The whole structure is sealed and compressed within a confined space, and developed over non-mammalian brains (Bear et al., 2001). These old brain parts do not appear to have the more obvious electromagnetic structures highlighted in this paper. Cortical Brain structure also differs from a dipole by being a semi-complete sheared and twisted sphere convoluted with non-dipole related appendages such as the brain stem and cerebellum (Bear et al., 2001). For simplification of analysis, removal of the non-mammalian brain stem and cerebellum is carried out in the sections that attempt remodelling the brain in dipole/multipole terms. This is carried out on the basis that the brain dipole/multipole structures are not fully complete due to the brainstem and skull's facial intrusion. During hominid evolution the brain has progressed towards a more spherical shape, (Smith, 1998) which is consistent with the spherical shapes dipoles tend to form if not constrained (Sewell, K.K., 1999). To fully analyse the brain as a multipole structure, the brain structure is looked at as semi-compete electromagnetic sphere. The cortex and limbic system are often mirrored in the images in this project to give an impression of what the brain would look like if multipole shapes of the brain were complete. This process is carried out for the purpose of pattern matching with common electromagnetic structures, the majority of which are spherical or spin completely round a central axis.

1.2 Analysis of Cortex as a Dipole structure

In table 1 the cerebral cortex can correlate with ten major features of dipoles. Each point from this table will be expounded upon in the following sections.

Table 1: Ten primary features of dipoles and their correlates in cortical brain structure.

DIPOLE FEATURES	CORTEX
1. Field lines	Callosum axons
2. Midline Symmetry	Corpus Callosum
3. Domain wall	Longitudinal fissure
4. Toroidal structures	sagittal sinus , induseum griseum , Callosum cavity
5. Divergence / Convergence	Neuropsychology opposite in hemispheres
6. Polar asymmetry	Music + language, Functional asymmetries
7. Charge/ Field reversal in each side	Neurons, receptors and neurochemicals are reversed
8. Polarity at dipole poles	Na+ (sodium) depolarizes right temporal lobe, Cl- (chlorine) Hyperpolarizes left.
9. Frequency reduction from mid axis to pole (radiating dipole)	Audio at poles, vision at midline.
10. Torque / Spin	Yakovlevian , Cartoid supply, Cortical folds

Each of these points is expanded on within section 1.

The analysis outlined in the table above concerns the correlation between cortex and dipoles. This structure can be broken down into shapes, patterns and properties. The evidence throughout this paper will be suited to either. For example some of the tables will clarify patterns of lateralization from data such as neurochemical distributions. These will show what neurochemicals are predominant in the Right and Left Cerebral Hemispheres. (referred to as RCH and LCH) Images will be used to understanding the shape of brain areas. Observational data is a key premise to find an easy entry into this subject. So first of all as few observable features are briefly covered to introduce the concept, while the most important features concerned with highlighting dipole field and polarity in the distribution of brain content, (points 6,7 and 8, table 1) will be largely data driven reviews of the current data on neurochemicals and receptors. (Section 1.3 – 1.7) What follows from here, and constitutes the entire Section 1, is a more in-depth description of each dipole feature outlined in table 1. With the aim being to show that each of the ten features of a dipole has its basis in the cortex.

1: Field lines are observable and evident when comparing cortical dissections with dipoles. In this graphic a ring electromagnet dipole simulation (Belcher, J., 2005) correlates well with cortical axons.

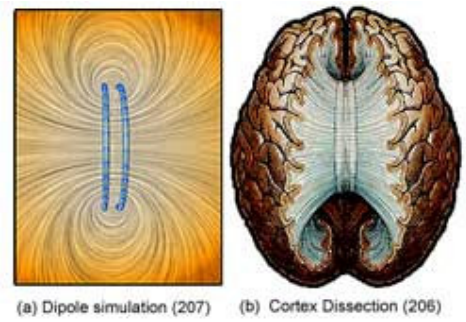

Figure 1: Brain dissection visually correlates with dipole. (a) (Belcher, J., 2005) (b) left (Adapted from Hubel, D. 1998.)

2: Midline Symmetry. The Corpus Callosum is symmetrical along its longitudinal axis. In the data presented for dipole features 6 and 7, neural processing and neurochemical distribution is more symmetrical at the cortex midline than any other brain area.

3: The Domain wall runs along the midline axis of a dipole and represents an area with no electromagnetic force. This correlates with the gap at the longitudinal fissure, although this gap is quite broad. In Section 2: Cortex surface is examined and proposed to have ferroelectric properties which are disordered. In ferroelectric dipoles, the domain wall is usually broader than in ordered dipoles. (Kirichenko et al., 2003)

4: Toroidal structures, (donut style tubes) at the midline are a feature of almost every dipole. These are evident from computed dissections of corpus Callosum. (Figure 2a,b) The induseum griseum running round the corpus callosum is a tube like structure. tube like. As is the sagittal sinus, which runs round the previously mentioned domain wall, the longitudinal fissure. These tube-like structures are also reflected in the axons of the longitudinal fasciculus which run all the way round the corpus callosum.

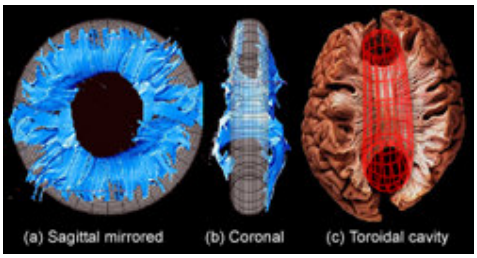

Figure 2: Toroidal cavity in brain structure. Left, cavity is mirrored to from a full toroidal tube. (Adapted from Basser et al., 2000) Middle, front view. (Adapted from Basser et al., 2000) Right, top view, showing a Toroidal graphic which has been placed into the cortex dissection. (Adapted from Williams and Gluhbegovic 1992)

5. Divergence / **Convergence :** Sperry and Gazzaniga's well known epilepsy research with split brain and WADA Procedure's (closing of blood flow to one brain hemisphere) revealed previously unseen and often opposite human neurological cognitions from the left and right sides of the brain. (Levy and Sperry, 1971) Given the task of drawing a navon, which are large visual shapes compromised of smaller features (i.e. a cup drawn from composing together smaller elements such as the letter D), the patients with right hemisphere damage (left hemisphere working) only drew the components of the navons. When the left hemisphere is damaged (right hemisphere working) patients drew the overall shape of the navon, but missed the components. (Carter, 1999) This spurred a lot of subsequent research into brain laterality at every conceivable level. This research evolved from understanding behavioral tendencies (Finke and Bettle, 1996; Springer and Deutsch, 1993) to cognitive processes, (Kucharska-Pietura et al., 2003; Fink et al., 1997; Vallortigara et al., 2004) revealing that many lateral functions appeared to be the complete antithesis of each other. (Table 2)

Many of these qualities would appear to be opposites. Researchers and theorists have used a number of these terms to define opposing factors in complexity theory (Capra, F., 1996.) : Order is a terms associated with convergent/pull processes (Kauffman, 1993;Fuller, 1979) and the opposite of Chaotic, a term associated with divergent/push processes. (Kauffman, 1993; Fuller, 1979) An important point that ties in electric dipoles with this systems analysis of the cortex, is that an electric dipole field is also defined by divergence at the positive pole and convergence at the negative pole. (Lowrie, 1997) Lowrie argues that divergence is the spreading out of the charge from the positive pole where as Convergence is the moving inwards of charge toward the negative pole.

The evidence from epilepsy research is gathered with the cortical callosum connections being split completely - giving a revealing but incomplete view of hemisphere function. (Levy and Sperry, 1971) A more accurate view of how the cortical hemispheres operate to separate processing emerges in the 1980's, summarized in table 2, which highlights the lateral functions of most major cortical regions. These are categorised as convergent for the LCH (Left Cerebral Hemisphere) and divergent for the RCH. (Right Cerebral Hemisphere) This is because, when given a specific cortical region of study the left hemisphere processing moves towards a more narrowly defined point of localised cortical processing (e.g. visual landmarks, shapes, voice) in comparison to the right hemisphere. When looking at the same cortical region studied in the RCH, there is a wider coverage of processing within a similar space of cortical area that can encompass larger or global features. (e.g. Entire visual scenes and faces)

6. Polar asymmetry: Table 2 shows that the cortical regions which are located along the midline fissure of the cortex, (i.e. the visual cortex) have less marked processing differences than the auditory areas which are located at the farthest separated temporal regions. It will be proposed as there is increasing distance between cortical regions on either hemisphere that divergence (global) and convergence (local) processing increases respectively for the RCH/LCH. So local processing for the LCH and global processing for the RCH increase from cortical midline to temporal regions.

For example, the midline V1 visual regions are co-operating to build up a stereo image differed only by the space between our eyes. (Bear et al., 2001) As processing moves out to V2 and V3 there is divergence and convergence in the respective hemispheres, marked by the move to process local information in the LCH and global visual information in the RCH. For example processing of navons, which are shapes constructed of large visual shapes compromised of smaller features show that the visual cortex starts to separate local and global features at area V2 (Fink et al., 1997). Where as Area V1 is focussed on the separation of similar visual information between the entire scene from each eye, known as binocular disparity (Bear et al., 2001). Moving out from visual areas to hippocampus where spatial visual information is separated into landmarks and entire scenic features (Vallortigara et al., 2004; Maguire et al., 1997). Again moving out from hippocampus regions to auditory cortex are the planum temporal areas. (Which are marked out as the furthest separated poles in the Cortical EMS model) The differences in processing here are so marked that the context of information is itself altered, with language on the LCH and music at the RCH (Carter, 1999).

It is proposed that processing at these temporal regions has reached its maximum point of asymmetry. fMRI reviews also highlight these areas as the most asymmetrical to each other in volumetric terms, (Toga and Thompson, 2003) although this asymmetry could be due to human specialization for language (Toga and Thompson, 2003). Research evidence suggests that Language develops in a logical and self-referring manner (Chomsky N., 1969). Language can also be considered a mono dialogue as it operates in a start/stop manner between speakers. On the same lobe on the right side is music which by contrast to language can be proposed as divergent, continuous and multi-layered, without the constraints of logic and stop/start protocols. With the right cortex known in general as being spatially orientated, (Carter, 1999) music is proposed to require a more divergent network to layer its spatially continuous auditory soundscapes. The auditory regions of right and left hemispheres, do not process local or global features in a complementary manner, like the closer together visual regions that are higher in cross hemisphere connectivity (Peters et al., 1990). They appear to process far more disconnected, asymmetrical kinds of information. Music and language have little resemblance to each other, except to say they are diametrically different processes of audio information.

Table 2: Summary of processing in either hemisphere.

Hemisphere	Cortical area	Observed Processing	Reference
LCH		Analytical	(Springer and Deutsch, 1993)
RCH		Holistic	(Springer and Deutsch, 1993)
LCH		Verbal	(Carter, 1999)
RCH		Musical	(Carter, 1999)
LCH		Logical	(Finke and Bettle, 1996.)
RCH		Random	(Finke and Bettle, 1996.)
LCH		Ordered	(Finke and Bettle, 1996.)
RCH		Chaotic	(Finke and Bettle, 1996.)
LCH	V2, V3 Left visual cortex	Local shapes	(Fink et al., 1997)
RCH	V2, V3 Left visual cortex	Global picture	(Fink et al., 1997)
LCH	Left frontal lobe	Perceives vocal expression	(Kucharska-Pietura et al., 2003)
LCH	Left frontal lobe	Processes analytically	(Carter, 1999)
RCH	Right frontal lobe	Perceives facial expression	(Kucharska-Pietura et al., 2003)
RCH	Right frontal lobe	Processes conceptually	(Carter, 1999)
LCH	Left Hippocampus	Landmark information	(Vallortigara et al., 2004)
RCH	Right hippocampus	Whole scene information	(Vallortigara et al., 2004; Maguire et al., 1997)
LCH	Left auditory cortex	Language	(Carter, 1999)
RCH	Right auditory cortex	Music	(Carter, 1999)

Rows with cortical area descriptions (lower half of table) concentrate on neuropsychology while those above are more popular descriptions.

differences in lateral processing may be attributed to neuronal assemblies. For example the left hemisphere has more closely packed, tightly connected neurons, giving quicker co-operation between similarly dedicated brain cells (Carter, 1999). In comparison to the right hemisphere auditory cortex, the left hemisphere spacing of the interconnected clusters of neurons is significantly larger, with a greater number of “selectively” interconnected columns (Hustler and Galuske, 2003). Greater separation of connectivity in cortical columns gives improved extraction of information from auditory inputs and separation of processing streams (Hustler and Galuske, 2003). In the right hemisphere auditory cortex there is greater number of interconnected columns (Hustler and Galuske, 2003). Dendrite spreading (Carter, 1999) in lower order dendrites from spiny stellate cells, which link together information within sensory areas, (Kalpouzos et al., 2005) are longer in the right hemisphere (Shiu and Nemoto, 1981). In other words there are more concentrated separations of neuron assemblies in the left hemisphere which are proposed to converge processing of sensory information. By diamteric contrast the extended connections in the right hemisphere, diverge sensory processing. The cortical EMS model proposes that the opposites of divergence for positive charge, and convergence for negative charge in dipoles (Lowrie, 1997) are responsible for these diametric differences and related neuronal assemblies. In other words if the LCH and RCH temporal regions are the brains poles, then this is the overarching structure which should predict the distribution of all primary grey matter content and larger neuron assemblies.

1.3 Neurochemical evidence for dipole type charge distribution in Cortex.

This section covers in great depth these dipole features:

6. Charge/ Field reversal in each side
7. Polarity at dipole poles

If hypothesising that visual, neuropsychological, and systems observation of the whole cortex implies dipole structures, then the entire cortical grey matter content should be consistent with a dipole structure. Neurotransmitters, receptors, ions, neuromodulators and associated processes must lateralize into opposite hemispheres, in a manner consistent with the separation of charges in a dipole. It may also be predicted that an increase of the components, which represent charge lateralization, will be found in greater concentrations towards the proposed poles at the temporal lobes. This does not lead to a mechanism for how a Cortical Dipole is formed, (which is covered in section 2.6) but is the key to pointing out the distribution of neurochemistry in grey matter is consistent with the predictions for separations of charged ions. Even if these ions are completely isolated from each other in terms of net electromagnetic coherence, however not all researchers disagree with this hypothesis (Hameroff, S., 2007,2009.)) it will then be important to consider what their overall effect is on the function and structure of the areas where they are found. In respect to this, section 2 also reviews mechanisms by which the resultant effect of these ions can be sustained in a larger polarized magnetic field.

The poles of a dipole, electric or magnetic have opposite polarity (Kane and Sternheim, 1988). Since the brain function known so far in grey matter is mostly electrical, it is presumed that we are looking for electric dipole chemical distributions that remain in the grey matter. Positive and negative or its counterpart in complex physiology which could be defining these poles in many other ways: Acid/base, hyperpolarized/depolarized etc. As long as there are the structural features you would expect with a separation of charges that occurs across hemispheres in development. These charges would converge/diverge towards specific points, which are at opposite ends of the cortex located at the temporal lobes. This temporal lobe axis polar distribution would be consistent with the observations of field line branching and midline toroidal structure (figure 1,2). The purpose of this section is to consider supporting and apposing evidence for such hypotheses. This involves a comprehensive review of the available scientific literature on the cortex small scale content: neurotransmitters, receptors and neurochemical distributions. There is available a large amount of disparate evidence describing cross-hemisphere reversals of brain content, which assist in line with the charge requirements of the dipole model.

The distribution of primary ionic excitatory and inhibitory neurotransmitters gamma-Aminobutyric acid (GABA) and glutamate, their voltage gated receptors and ions, have an opposite symmetry. (Figure 3) The primary ions involved in left/right brain lateralization are negatively charged chlorine and positively charged sodium, each of which are gated into their respective receptors. Chlorine into GABA and Sodium into Glutamate. By synthesising the available research on this cortical content into tables of lateralization patterns, a comprehensive analysis will highlight that GABA is lateralized to the left hemisphere and glutamate in the right hemisphere. Both more so at their temporal regions. GABA forms mainly convergent neuron networks and fires negatively charged chlorine ions which inhibit neurons. Higher concentrations of GABA would be spatially summarized as a net Inhibitory Post Synaptic Potential (IPSP) activity. Glutamate forms mainly divergent neuron networks and fires positively charged sodium ions which excite neurons. Higher concentrations of Glutmate would be spatially summarized as a net Excitatory Post Synaptic Potential (EPSP) activity. The evolution of Gaba and Glutamate ionotropic receptors not surprisingly co-incides with the movement of earliest plant life forms into NaCl water environments. (Ryan and Grant, 2009) at similar time to the emergence of bilaterian evolution of symmetry in the nervous system such as Eumetazoan Sea Anemone. (Putnam et al., 2009). If this separation of charges approach is taken in line with the evolution towards the dipole features of the human cortex, it is then possible to propose that the physical interplay between ionotropic Gaba and Glutamate receptors could be a consistent developmental factor responsible for symmetrical structure throughout the greater part of nervous system evolution.

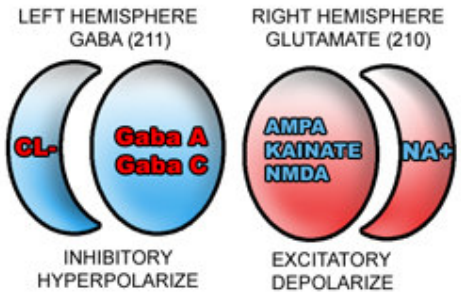

Figure 3: Diagram points out reverse symmetry for ionic receptor structure and the ionic key for polarization at each proposed pole. Cl- for gaba (Moss, 2006) at left temporal pole and Na+ for Glutamate (Doherty, 2001) at right temporal pole. In this sense it would not be a surprise that the nervous system has evolved its primary separation of charges from the balanced and common structure of sodium Chloride. NaCL. (Author image)

1.4 An Overview of Primary Cortical Content

The ratio of GABA to Glutamate neurons is in favour of glutamate 70/30 (Johansson and Lansner, 2007) and do not fit a 50/50 balance of inhibition/excitation expected initially for a balance of charges in a cortical dipole model. This is based on the evidence that the cortex appears somewhat symmetrical, although dipoles can be asymmetrical. A cortex dipole model does not demand symmetry at the poles but may only require a certain degree of symmetry, which occurs at the midline of a dipole and would be expected based on the observational data (figure 1). There is asymmetry at the proposed poles, the temporal lobes, which will be shown in the following section *1.5.4.* The following points show how the unequal ratio of excitatory/inhibitory neuronal numbers may not represent the ratio of electrotonic inhibitory/excitation in the cortex as 70 (excitation)/30 (inhibition) ratio exists.

1. GABA has a higher fidelity of information transmittal than that of other known major neurotransmitters such as Glutamate (Roberts and Sherman, 1993) so may require a smaller number of units for equal effect.

2. The numbers of dopamine neurons and pathways in the brain is small compared to the number of serotonin neurons, yet they still compete equally for chemical resources and brain territory, with dopamine controlling the left hemisphere and serotonin the right. (Table 5,6) Adding serotonin decreases the formation of dopamine and vice versa (Manoucher and Messripour 1992). GABA and Glutamate also exist on such an axis: Acidification decreases postsynaptic efficacy of glutamate, and alkalinization increases it while alkalinization decreases GABAergic inhibitory function and acidification decreases it (Roberts and Sherman, 1993). GABA/glutamate system exists in some kind of balance, even if how they express themselves in bare numbers of components does not appear balanced.

3. The cortex does tend towards excitatory processes, but this need not rely on Glutamate. GABA neurons can also exert disinhibtion. (Roberts and Sherman, 1993)

4. The ratio of cortical GABA synapses is estimated at 40%. (Ashton, 2002; Rosack, 2002) GABA synapses can exert a lot of control with a minority numbers of synapses, pointing to more inhibition than previously thought. (Roberts and Sherman, 1993)

5. Glutamate receptors are highly diverse. Glutamate is more likely to form divergent network. The same transmitter can activate many kinds of receptors and responses (Siegel et al., 1999). GABA receptors are mostly homogenous. GABA has a diverse number of transmitters directed at a single chloride channel (Siegel et al., 1999). Convergent networks activate many types of response from a single source, so need fewer numbers of components to match a divergent network. This is illustrated by the many functions of dopamine D2 receptors in comparison to their 5HT counterparts. (table 6) The ratio of GABA to glutamate synapses is 40/60 perhaps because Convergent aspects of a system require less firing components, and more control components. GABA receptors are homogenous and attract other types of inhibitory neurotransmitter systems to work for them. An increase in systems power which does not belong to the Glutamate receptors. Glutamate receptors are heterogeneous. They tend towards proliferating many types of connections.

In comparison to each other, the left hemisphere is a convergent system and the right hemisphere a divergent one. It is no surprise that the inhibitory GABA receptors are lateralized in the left hemisphere (Morand et al., 2001; Cernácek, 1989; Guarneri et al., 1988). Something that has come up in this research is that other researchers have a harder time pinpointing cortical distributions of right hemisphere excitatory Glutamate, or Serotonin receptors in comparison to their left hemisphere inhibitory counterparts GABA or dopamine (Lester et al., 1996).]. It may be that divergent networks are harder to pin down due to their heterogeneous proliferations.

It is necessary to show that not only is GABA left lateralized and Glutamate right lateralized, but for a dipole that there will be more GABA systems at the brains left pole, and more Glutamate systems at the right pole. There is a large amount of research in lateralization from 1970 to 1990, for most major components in the brain. Allowing analysis of existing information to highlight that the glutamate system is lateralized in the Right Cerebral hemisphere (RCH) and GABA system is lateralized in the Left Cerebral Hemisphere (LCH). This includes their enzyme, marker, lipid and signalling processes. These will be broken down into 3 sections outlined below where the results of the analysis are put into a table. (Table 3) NOTE : LCH is Left Cerebral Hemisphere, RCH is Right Cerebral Hemisphere

As the following sections are technical in depth, it will be summarised in table 3 and 7 which analyses all current data on neurochemical distributions affecting GABA/Glutamate and cortical neuromodulation systems. Section 3 of the table shows that highest asymmetry and density for GABA/Glutamate occurs at temporal regions. Extensive reviews of schizophrenia take place as this condition is marked by temporal region asymmetry as a result of an alteration of GABA. In other words Schizophrenia is used as a guide to examine how alterations in the GABA/glutamate systems affect the lateral neurochemical distributions predicted by the Cortical EMS model.

1.5 Data for Lateralized Neurochemical distribution

1.5.1. Transcollasal effects of Progesterone and LCH/RCH effects of Glutamate administration.

a) Glutamate administrated to just the LCH of chicken forebrain has marked excitatory effect, increase in behavioural aggression and retards sensory processing. Glutamate treatment of both LCH and RCH retards both hemispheres sensory processing but with no LCH increase in aggression. Glutamate administrated to just the RCH has no effect. (Howard et al., 1980)

In chickbrain, why is the LCH the most severely affected and the RCH not at all ? The proposal would be that RCH is naturally structured to handle high glutamate levels, as it is glutamate dominated in comparison to the LCH.

b) Progesterone enhances the GABA receptor (Smith et al., 1987; Callachan et al., 1987) and decreases the effect of glutamate (Smith et al., 1997). LCH performance has been found to increase under progesterone, which is due to a decoupling of the RCH. (Hausmann and Güntürkün, 2000)

Transcollasal transmission (across corpus callosum) is dependent on a glutamate induced initial excitatory postsynaptic potential (EPSP) activating pyramidal neurons which receive transcallosal input (Hausmann and Güntürkün, 2000). This EPSP is followed by an inhibitory postsynaptic potential (IPSP) which involves the activation of GABA-ergic interneurons (Kumar and Huguenard et al., 2001). If GABA receptors are enhanced by progesterone, then transcollasal decoupling of the RCH due to progesterone implies that there is greater concentrations of GABA in the LCH.

1.5.2. Indirect markers for LCH GABA and RCH Glutamate.

a) RCH Glutamate: Isoprenoid Pathway and Glutamate in RCH

Quinolininc acid, serotonin and elevated HMG CoA reductase activity are found to add greater excitement to glutamate transmission. All these glutamate exciting biochemical factors have been found to be lateralized in RCH. (Kurup, K., and Kurup A., 2002, 2003a, 2003b, 2003c)

b) LCH GABA: LCH Enzymes Limit Glutamate and Modulate GABA

Several enzymes involved in lipid metabolism and Ca2+/ phospholipid-dependent protein kinase (PKC) activation are lateralized to the LCH (Pediconi and Barrantes, 1990). The LCH displayed about 50% more PKC activity in synapses than the RCH and (Ginobili de Martinez and Barrantes, 1988) this PKC activity modulates GABA transporter function (Macek and Schaffhauser, 1998; Beckman et al., 1998).

Table 3: showing lateralization of the glutamate / GABA system

Section	RCH/ LCH	Agonist, Antagonists, Modulators	Resultant process or lateral activity	Reference
1.4	LCH		GABA receptors are lateralized	(Morand et al., 2001; Cernácek, 1989; Guarneri et al., 1988))
1.5.1a	RCH	Glutamate treatment of RCH	Retards processing, increases rat aggression	(Howard et al.,1980)
1.5.1a	LCH	Glutamate treatment of LCH	No problems	(Howard et al.,1980)
1.5.1b	LCH	Progesterone	Increased LCH performance	(Smith et al., 1987
1.5.1b	iLCH	Progesterone	Enhances GABAA receptor	Callachan et al., 1987)
1.5.1b	iRCH	Progesterone	Attenuates the effect of glutamate	(Smith et al., 1987)
1.5.2a	RCH	Quinolininc acid, HMG CoA reductase, serotonin.	1. Lateralized in the RCH. 2. Glutamate agonists.	(Kurup RK and Kurup PA; 2002, 2003a,b,c)
1.5.2b	LCH	Ca2+/phospholipid PKC	LCH lateralized.	(Pediconi and Barrantes, 1990,1993; Strachan and Read, 1999)
1.5.2b	LCH	Ca2+/phospholipid PKC	50% more activity in LCH synapses	(Pediconi and Barrantes, 1990,1993)
1.5.2b	iLCH	PKC	Modulate GABA	(Macek et al., 1998, Beckman et al., 1998)
1.5.2b	iRCH	PKC	Inhibit Glutamate	(Macek et al., 1998)
1.5.2c	iRCH	Arachidonic acid (AA)	Implicated in NMDA (LTP) Long term potentiation	(Miller et al.,1992)
1.5.2c	RCH	Arachidonic acid (AA)	RCH lateralized	(Pediconi, 1984; Ginobili de Martinez et al., 1985)
1.5.2c	iRCH	Arachidonic acid (AA)	Amplifies glutamate	(Miller et al., 1992)
1.5.2c	RCH	Glutamate LTP in cats	Led by right amygdalo–hippocampus	(Adamec, 1999)
1.5.2d	iRCH	PLA2 - AA,(omega 6) -PGE2	(inhibited) by GABA	(Shiu and Nemoto, 1981; Colin et al., 2003)
	iRCH	AA-PGE2	Inhibited by Omega 3 lipids.	(Colin et al., 2003; Berger et al., 2006; Babcock et al., 2000; Hong et al., 2001)
1.5.2d	iRCH	GABA receptors	Attenuate AA	(Shiu and Nemoto, 1981)
1.5.2e	LCH	Increasing omega 3	Reduces LCH asymmetry in Schizophrenia.	(Bell et al., 2004; Richardson et al., 1999)
1.5.2e	RCH	Schizophrenia high omega 6 AA	Reduces RCH due to oxidative stress	(Ross et al., 1999)
1.5.2e	LCH	Dysfunctional NMDA	LCH asymmetry in Schizophrenia.	(Thompson et al., 2001)
1.5.3	LCH	Schizophrenia	Damage to GABA function in the left hippocampus	(Begany, 2004) (Jeon and Polich, 2003)
1.5.3	RCH	Schizophrenia	iPLA2 activity increased by 45%	(Ross et al., 1999)
1.5.3	RCH	Schizophrenia	Increasingly RCH hypofunction asymmetrically to RCH temporal lobes	(Thompson et al., 2001)
1.5.3	LCH	GABA receptors	Asymmetry at left auditory cortex	(Morand et al., 2001)
1.5.3	LCH	Anxiety states	GABA reduced at left temporal pole	(Tiihonen et al., 1997)

The table represents the existing data on lateralization of primary ionotropic receptors and associated processes. The overall picture showing how they distribute cleanly to the LCH (Gaba) / RCH (glutamate). Lower down the table at section 1.5.3 data is applied which highlights temporal regions lateralization of LCH (Gaba) / RCH (glutamate). iRCH or iLCH means Indirect marker for RCH/LCH, in that an RCH association for agonist, antagonist or modulator can take place when in conjunction with another reference. For example in 1.5.2d AA can be inferred to be in the RCH as increasing omega 3 reduced LCH asymmetry (increased omega 3 in 1.5.2e). The Section refers to the sections in the text which discuss the data.

1.5.1. Transcollasal effects of Progesterone and LCH/RCH effects of Glutamate administration: This section covers two studies of direct RCH/LCH changes which occur due to neurochemical administration affecting Glutamate and GABA.
a) Glutamate administered to either and both front hemispheres of chick brain, shows that only the LCH has problems.
b) LCH performance increases under progesterone which enhances GABA function

1.5.2. Indirect markers for LCH and RCH distribution of GABA/Glutamate.

a) RCH Glutamate: Isoprenoid Pathway and Glutamate in RCH: Some research from India that suggests the Right lateralization of glutamate can be inferred from other biochemical markers, which they have selected.
b) LCH Gaba: LCH Enzymes Limit Glutamate and Modulate GABA: Some research which shows that enzymes in the LCH inhibit Glutamate and modulate GABA
c) RCH Glutamate: Modulation of Glutamate in RCH by Arachidonic Acid. Glutamate LTP is modulated by RCH lateralized fats Arachidonic acid (AA and their precursors) prostaglandin E2 (PGE2) and Phospholipase platelet A2 (PLA2)
d) LCH Gaba: Inhibition of AA,PLA2 and PGE2 in LCH by GABA and Omega 3, which is a reverse of the above in the LCH. AA, PLA2 and PGE2 are all inhibited either by GABA or Omega 3 lipids. Omega 3 and 6 appear to be lateralized to the LCH and RCH respectively.
e) Shows how the RCH/LCH can be altered by schizophrenia.

1.5.3. Polar Lateralization in Schizophrenia: An important evidence section for a dipole model here is polarization. Here a case is made that GABA is lateralized extremely at the brains left temporal regions, with the reverse true for Glutamate (right temporal regions). Both GABA and Glutamate are polarized as well as being distributed at their highest density at the temporal lobes.

c) RCH Glutamate: Modulation of Glutamate in RCH by Arachidonic Acid (AA)

Lateralization of AA (an omega 6 lipid) is higher in the RCH (Ginobili de Martinez and Barrantes, 1988; Pediconi and Barrantes, 1990). AA is derived from long-chain polyunsaturated fatty acids (LC PUFA) and is a precursor of prostaglandin E2 (PGE2) (Ginobili de Martinez and Barrantes, 1988; colin et al., 2003) which modulates postsynaptic membrane excitability and long-term synaptic plasticity (Bazan, 2003). AA originates at postsynaptic glutamate site in response to Phospholipase platelet A2 (PLA2) activation. PLA2 activation is a retrograde messenger of long-term potentiation (LTP), a modulator of glutamate release, and an upregulator of memory formation (Bazan, 2003). To summarize the pathway is PLA2 – AA - PGE2, all being essential to brain function, and marking out glutamate as their target.

Mechanisms within the glutamate system have been found which facilitate amplification of glutamate (Miller et al., 1992) by AA. Furthermore these mechanisms are thought to be implicated in glutamate receptor LTP (Miller et al., 1992; Pellerin et al., 1991). For example cats behavioral changes are lead by LTP in the RCH amygdalo–hippocampus (Adamec, 1999). To summarize AA is higher in RCH and LTP also lateralized in the RCH when assisted by the AA precursor PLA2 and its metabolite PGE2. Unless there are similar but undiscovered modulation processes in the LCH facilitating glutamate LTP, the evidence suggests that key glutamate processes are more predominant in the RCH.

d) LCH Gaba: Inhibition of PLA2 AA, and PGE2 in LCH by GABA and Omega 3.

In the LCH, GABA and omega 3 lipids appear linked in a similar way to glutamate and AA (omega 6) in the RCH (Ginobili de Martinez and Barrantes, 1988; Pediconi and Barrantes, 1990). Omega 3 Docosahexaenoic acid (DHA) reduces AA (Omega 6) metabolite PGE2 (Colin, 2003) that modulates NMDA (glutamate receptor) postsynaptic membrane excitability and long-term synaptic plasticity (Bazan, 2003) in the RCH. (Tiihonen et al., 1997) So Omega 3 appears to inhibit key Glutamate processes. GABA receptors activated by barbituates also decrease AA (omega 6) during global ischemia (Shiu and Nemoto, 1981). Both Omega 3 (DHA) and activation of Gaba receptors then inhibit the key lipid processes of glutamate function. Omega 3 (DHA) also serves to modulate GABA (Nabekura et al., 1998) Pointing to the idea that Omega 3 could be linked to GABA function and Omega 6 to glutamate function.

If there is a competing axis between GABA/omega 3/ and Glutamate/omega 6 to to RCH and LCH, the attenuation of AA by LCH GABA receptor agonists (GABA being LCH lateralized (Morand et al., 2001; Cernácek, 1989; Guarneri et al., 1988)) could explain why the lateralization of AA is significantly higher in the RCH. This section points out there may be an axis with increases in LCH components reducing the operation of PLA2, AA, and PGE2 which are all involved in modulating glutamate and facilitating LTP in the RCH. This fits with a reversal axis model for the hemispheres. As one set of modulators increase function in one hemisphere a decrease occurs in the other hemisphere. As it is already clear that GABA is LCH lateralized, (Morand et al., 2001; Cernácek, 1989; Guarneri et al., 1988) the picture is emerging that glutamate processes are strongly distributed to the RCH.

e) Schizophrenia alterations to lateralization

It is possible to integrate section 1.5.2(c) RCH and 1.5.3(d) LCH lipid processes, by looking at schizophrenia research. Schizophrenia is marked by glutamate/ N-methyl-D-aspartate (NMDA) receptor dysfunction (Nyiri et al., 2003; Abi-Saab et al., 1998; Korn, 2000) which leads to LCH asymmetry (reduced RCH function) (Thompson et al., 2001). This LCH asymmetry was shifted back to balanced LCH/RCH symmetry following treatment with omega 3 Eicosapentaenoic acid (EPA) acid (Richardson et al., 1999). As mentioned omega 3 in DHA form increases modulation of GABA which is LCH lateralized and reduces glutamate RCH processes. So why does an increase in omega 3 reduce LCH asymmetry back to balanced LCH/RCH symmetry ?

Omega 3 (EPA) inhibits the AA triggering enzyme PLA2 (Berger et al., 2006) which hydrolyzes fatty acids like AA from membrane liberated omega-6 PUFAs (Haag, 2003) and so reduces synthesis of AA (Babcock et al., 2000). So not surprisingly Omega 3 also inhibits the AA metabolite PGE2. (Colin, 2003). PLA2 activity (Calcium-independent) which triggers AA production is elevated in schizophrenia leading to abnormal AA metabolism and oxidative stress, (Ross, 1999) This presumably plays a role in the glutamate/NMDA hypofunction of schizophrenia (Nyiri et al., 2003; Abi-Saab et al., 1998; Korn, 2000). It is this hypofunction which marks out the decrease in RCH lateralization and increase to LCH asymmetry. As omega 3 (EPA) competes with AA (Omega 6) for incorporation into phospholipids, (Hong, 2001) and AA is elevated in schizophrenia - increasing omega 3 (EPA) reduces AA (Omega 6) and alters the axis of brain back from LCH to RCH.

To summarize, a review of the research points at lateralization of omega 3,6 neurolipids, (Pediconi and Barrantes, 1990, 1993; Palestini et al., 1997; Pediconi and Rodriguez, 1984; Ginobili de Martinez et al., 1985) indicating a LCH/RCH axis based round Omega 3/Omega 6, with Omega 3 linked to modulate GABA in the RCH and Omega 6 linked to modulate Glutamate in the LCH.

Table 4 : Lateralization of neuronal lipids.

RCH/ LCH	Agonist, Antagonists, Modulators	Resultant process	Reference
LCH	Omega 3 (EPA)	Reduces AA (Omega 6 precursor) Phospholipase A2	(Abi-Saab et., 1998)
LCH	Omega 3 (DHA)	Modulates GABA	(Morand et al., 2001; Cernácek, 1989; Guarneri et al., 1988)
LCH	Omega 3	Reduces PGE2 (AA Omega 6 metabolite)	(Colin, 2003)
LCH	GABA / barbiturate	reduces AA (Omega 6)	(Shiu and Nemoto, 1981; Hattori et al., 1986)
RCH	AA (Omega 6)	Modulates Glutamate LTP	(Ginobili de Martinez et al., 1986; Ginobili de Martinez and Barrantes, 1988; Pediconi and Barrantes, 1990)

1.5.3. Schizophrenia as a guide to highlighting temporal lobe lateralization of NMDA/GABA

It is now emerging that the brains primary content GABA/Glutamate has some kind of RCH/LCH distribution, which is so far consistent with the theory. A cortical dipole model requires the highest density of GABA/Glutamate distribution at the proposed poles, the brains temporal regions. As has been shown from schizoprenia research, laterality can be linked to imbalances of lipids that are associated with either brain hemisphere. Schizophrenia is also considered to be a dysfunction of dysregulated NMDA (glutamate) receptors operating at reduced activity (Nyiri et al., 2003; Abi-Saab et al., 1998; Korn, 2000). Glutamate carboxypeptidase (GCP) II is an enzyme that degrades the NMDA receptor antagonist N-acetyl-alpha L-aspartyl-L-glutamate (NAAG). This enzyme is reduced in the hippocampus, prefrontal cortex, and temporal cortex of patients with schizophrenia (Begany, 2004). In other words reduced GCP is implicated in dysfunction of NDMA receptors. The loss of grey matter in these areas can be seen in figure 4a. (Red areas)

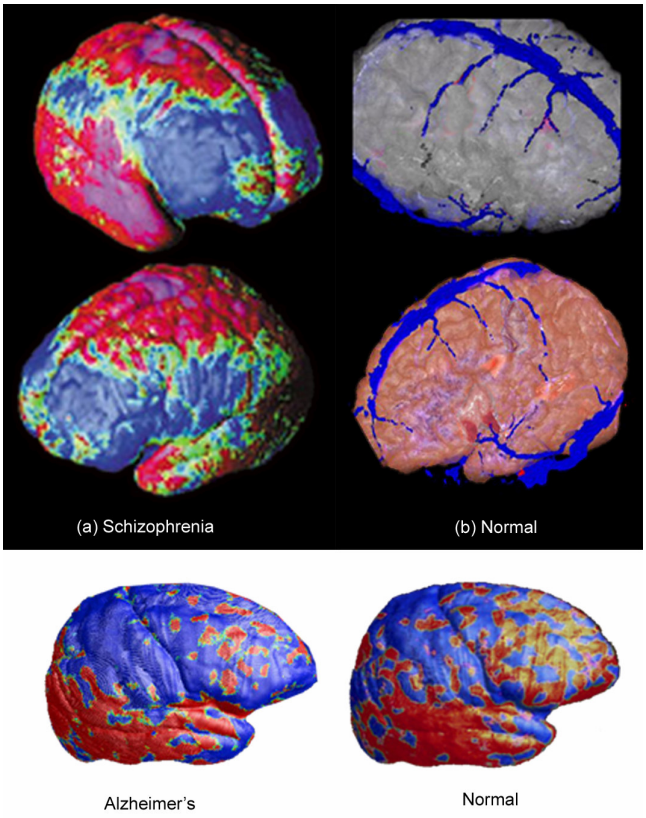

Figure 4a Left:, asymmetrical destruction of grey matter due to NMDA receptor dysfunction (Thompson et al., 2001). 4b Right: areas affected correlate to symmetrically supplied veinous returns (Hardenbergh, 2004). Bottom: Two images taken from the same paper. (Thompson et al., 2003) Bottom Left: in other grey matter neurodegenerative diseases which do not singly target GABA or Glutamate such as Alzheimer's neuronal loss is symmetrical. Bottom Right: These areas affected are also those with the highest neuron densities at the temporal lobes.

As can be seen in figure 4a, the grey matter loss is not symmetrical between hemispheres, at the temporal regions. Aside from the temporal regions, the loss does not correlate with areas of highest grey matter density as it does in Alzheimer's. (red areas in figure 4 bottom) Grey matter loss does however correlate well with the veinous return system (right in figure 4b, veins are blue) which is symmetrically distributed. This means that an almost symmetrical distribution of grey matter is being affected asymmetrically to a high degree. As neuron density is equal amongst hemispheres and even slightly greater in the left temporal lobe, (Toga, 2003) it is proposed that these asymmetrical patterns of loss (especially at the temporal lobes), represent a more extremely lateralized distribution of NMDA to GABA receptors in the cortex (the proposed Cortical EMS poles). There is LCH asymmetry of GABA receptors and neurons in left auditory cortex, (Morand et al, 2001) while reduction of GABA concentration in the left temporal lobe but not the right is correlated with extreme anxiety (Tiihonen et al., 1997). This implies that GABA loss in the left temporal lobe has more severe consequences than GABA loss at the right temporal lobe. This is consistent with the concept that the left temporal lobe operates its functions to a higher degree on GABA.

There is a slight decreased function of GABAA receptors in the hippocampus as schizophrenia moves towards first positive sympoms (Begany, 2004). P300 ERP (Event related potential) generation (for which Hippocampal function is considered essential (Jeon and Polich, 2003)) is reduced more in the LCH than RCH temporal lobe (Jeon and Polich, 2003). This is because NMDA-mediated glutamatergic activity impinges on GABAA interneurons (Woo et al., 2004) and so NMDA-mediated GABA release is markedly decreased (Woo et al., 2004). However this is a sub symptom of NMDA hypofunction (Begany, 2004) resulting in disinhibition that impairs cortical-hippocampal processing (Begany, 2004). Only a specific minority of GABAA neurons are thus affected (Hashimoto et al., 2008; Akbarian and Huang, 2006; Hashimoto et al., 2003). This asymmetrical NMDA hypofunction induced damage to GABA function in the left hippocampus highlights that there is an asymmetrical distribution of GABAA receptors in the left hippocampus. As mentioned previously (section 1.4) unlike glutamate receptors, GABAA receptors are globally homogeneous, so if they exist equally in the right hippocampus it would be expected that there would be no dysfunctional asymmetry, as their function is similar. As was shown in figure 4a, GABAA loss at the left but not right temporal lobe is known to be correlated with a move to the secondary symptoms of schizophrenia. To summarize, the above evidence suggests that GABAA receptors are strongly leftwards lateralized to the brain's proposed poles, the hippocampal, temporal, auditory areas.

1.5.4 RCH temporal lobe as highest density of glutamate/NMDA function.

In regards to highlighting a RCH temporal lobe lateralization of glutamate, it was shown in the above section (1.5.2e) that in schizophrenia iPLA2 (Calcium independent PLA2) activity is elevated and that this elevation leads to an increase in the AA (Ross, 1999) which is implicated in RCH glutamate memory formation processes. In this study iPLA2 activity was increased by 45% in the temporal cortex of patients with schizophrenia but was not significantly altered in other brain areas. No data was given in regards to iPLA2 lateralization though and parietal regions (seen affected in figure 4a) were also not studied either (Ross et al., 1999). In schizophrenia, altered PLA2G6A function (the gene which produces iPLA2) leads to significant reductions in the amount of AA incorporated into the cell membrane and decreases memory formation in rats (Law et al., 2006). This suggest that iPLA2 is directly implicated with the previously mentioned (section 1.5.2c) RCH glutamate LTP processes. In other words it can be proposed that a significant increase in iPLA2 in either RCH or LCH temporal cortex would lead to a greater impact on RCH temporal cortex glutamate/NMDA processes. (even if dysfunctional) It is proposed that this iPLA2 increase is the result of a disease (schizophrenia) which shifts brain structure from RCH to LCH. Is it possible to use schizophrenia and temporal lobe increase of iPLA2, as a guide to normal brain structure?

In normals, oxidative stress due to NMDA activation releases reactive oxygen species (ROS), increasing AA which in turn increases astrocyte operation (Volterra et al., 1994). Astrocytes then release cPLA2 (cystolic PLA2) and iPLA2 (Bazan, 2003; Volterra et al., 1994). Astrocyte processes are also implicated in schizophrenia at hippocampus/frontal/and temporal lobes (Begany, 2004; Han, 2007). The product cPLA2 is reduced due to excess dopamine, (Ross, 1999) resulting in large iPLA2 (Bazan, 2003) release in the temporal lobe (Ross, 1999). This increased iPLA2 also increases DHA (Green, 2008) which then inhibits GABAA (Nabekura et al., 1998) reducing PGE2 (Colin, 2003) as well as AA incorporation (Law, 2006). It is important to bear in mind that both AA and PEGE2 are RCH lateralized, (Section 1.5.2c) so the higher density of iPLA2 would affect the previously described RCH temporal lobe glutamate processes such as memory, (section 1.5.2c) and reduce any GABAA inhibition in that area. Does this explain the asymmetry of loss at temporal lobes seen in figure 4a? As can be observed there is markedly less grey matter loss at LCH area, where as the highest grey matter loss is at the RCH temporal region.

In schizophrenia there is decreased NMDA receptor co-expression on GABAergic interneurons proposed to be due to increases in temporal/hippocampal region NAAG as the hippocampus has abnormally high levels of neuronal activity (Begany, 2004). This together with iPLA2 reduction of GABAA, implicates two mechanism for reduced GABA function in the RCH hippocampus/temporal lobe, inducing NMDA hyperexcitability. As discussed previously cortical neuronal content is most dense at the temporal regions (figure 4 bottom) while GABAA receptors can be said to be most densely located at the LCH hippocampus and temporal region. This evidence points to reduced GABA inhibition of NMDA receptors playing a major role in this secondary phase of schizophrenia. That there is asymmetry of schizophrenia hypofunction at the RCH temporal region ,(Thompson et al., 2001) points to a greater concentration of glutamate/NMDA excitation (and resulting hypofunction) at the RCH temporal region than either the midline parietal or LCH temporal regions. There is less hypofunction at the LCH parietal,(Thompson et al., 2001) hence implicating greater RCH NMDA activity at the RCH temporal lobe and less LCH NMDA temporal lobe activity. If there is such laterality at the temporal poles but not at the cortical midline, then this explains why there is a more symmetrical distribution of NMDA/GABA hypofunction at the midline parietal region (Thompson et al., 2001).

To summarize, there is a proposed asymmetry of primary NMDA and GABAA receptors at the brains temporal lobes. These are also the areas with the highest neuron densities (figure 4 bottom). The temporal lobes could then be said to contain the brains greatest density of Glutamate/NMDA(RCH) and GABA/GABAA (LCH) processing. If this review of evidence is accepted, then the Cortical EMS model with its clarification of an asymmetry of GABA/Glutamate located at the temporal lobes may also contribute to models of schizophrenia. It does this by placing an emphasis on the fact that the LCH temporal lobe with its higher concentration of inhibition and lower levels of excitatory neurons, is the least affected by this disorder of excitatory hypfunction.

The Cortical EMS model points out that the cortex has a greater concentration of excitatory NMDA related function in the RCH than the LCH. As well as this there is greater GABA related concentration in the LCH. It is also proposed that the concentrations of either GABA or NMDA function are increasing towards their highest known density at the cortical temporal regions. This lateralization also applies to many of its related lipid, marker, signalling and enzyme processes. At the left and right temporal poles there is a greater degree of Asymmetry between GABA and NMDA respectively, These areas contain the brains highest separation of the negative (CL-)and positive (NA+) charges active at any time in the processes of Hyperpolarization (Cl-) and Depolarization (NA+) respectively. It is then not surprising that similar lateralization patterns also appear to be emerging for enzyme, modulation, marker, signalling and lipid processes associated with the cortex primary grey matter content.

1.6 Evidence for Lateralized neuromodulator distribution

1.6.1 Neuron Modulators

The section tackles the brains primary submodulators such as dopamine, serotonin, their receptors and associated chemical groups in a similar manner to the previous section, reviewing research on lateralization and their chemical properties. E.g. (Kurup RK and Kurup PA, 2002, 2003a; Flor-Henry, 1986; Bruder et al.,, 2001; Demeter et al., 1986) As mentioned previously, (section 1.4) GABA and Glutamate exist on an acid/base axis : Acidification decreases postsynaptic efficacy of glutamate, and alkalinization increases it while alkalinization decreases GABAergic inhibitory function and acidification decreases it (Roberts and Sherman, 1993). The following discusses the evidence that Dopamine and Serotonin can be considered similar to GABA/Glutamate, both operating on acid/base competition axis in a similar manner.

1.6.2 Base/Cation properties of Serotonin

Optimal pH for serotonin synthesis is about 7.2. As the pH of the suspension medium decreased below 7 the rate of serotonin formation declines (Rorig, 1996). The substrate form for the serotonin transporter is called the cation (Rorig, 1996; Thompson AJ, Lummis, 2003). Over one-fourth of all tryptophans in the protein data bank experience an energetically significant cation-pi interaction (Gallivan and Dougherty, 1999). Studies of the receptors for trytophan derived neurotransmitters nicotine and Serotonin find they have cationic binding properties, named Cation-p interactions (Zhong et al., 1998; Andrade, 1998; Beene, 2002; Parihar and Kirschbaum, 2002).

1.6.2b Acid/Anion properties of Dopamine

Dopamine operates in lower local Ph environments (Manoucher and Messripour, 1992). Optimal pH for dopamine production is 6.1. At pH 7.4 the rate of dopamine formation decreased to about 25% of that at pH 6.1 (Manoucher and Messripour, 1992). Dopmine or any of the tyrosine derived catecholamines neurotransmitters has no electrical (gap) junctions, (Collingridge et al., 2009) and decouples any local gap junctions (Piccolino et al.,1984; Rörig et al., 1995). Is this due to dopamine increasing acidity ? Intracellular acidification reduces gap junction coupling (Rorig, 1996). Serotonin has electrical junctions and these release sodium cations. Also Receptor held dopamine is an electron deficient anion Pi species, which would increase the acidity of its environment. Finally dopamine active transporter (DAT) potentiate a chlorine anion conductance associated with the dopamine transporter (Meinild et al., 2004).

1.6.3 Dopamine/Serotonin Acid/Base Axis

It is known that the level of free tryptophan (from which serotonin is derived) in the blood can influence the transport of tyrosine (from which dopamine is derived) across the blood brain barrier into the brain and vice versa, (Ravikumar et al., 2000) since both these amino acids share the same transport systems and compete with each other (Ravikumar et al., 2000). The rate of dopamine or serotonin synthesis in rat brain synaptosomes is determined as a function of pH (Rorig et al., 1996). Adding serotonin decreases the formation of dopamine and vice versa. These are Ph dependant with dopamine production at a lower Ph than serotonin (Manoucher and Messripour et al., 1992). In other words Dopamine/Serotonin production operate on a see/saw axis. This is also modified dependent on the acid/alkaline nature of their environment.

Table 5: Lateralization of modulating neuro-transmiiters and their chemical groups.

RCH/ LCH	Neurotransmitter	Chemical group	Reference
LCH	Dopamine	Tyrosine Derived	(Cernácek, 1989; A and Kurup, 2003; Glick et al., 1982; Toga and Thompson, 2003; Flor-Henry, 1986)
LCH	Norepinephrine	Tyrosine Derived	(Cernácek, 1989; Kurup RK and Kurup PA, 2002, 2003a)
LCH	Morphine	Tyrosine Derived	(Kurup RK and Kurup PA, 2002, 2003a)
RCH	Serotonin	Tryptophan Derived	(Kurup RK and Kurup PA, 2002, 2003a; Flor-Henry, 1986; Bruder et al.,, 2001; Demeter et al., 1986)
RCH	nicotine	Tryptophan Derived	(Kurup RK and Kurup PA, 2002, 2003a; Gilbert, 2004; Gilbert, 1989)
RCH	quinolinic acid	Tryptophan Derived	(Kurup RK and Kurup PA, 2002, 2003a)

1.6.4. Reversals of properties for Serotonin/Dopamine receptors

Serotonin receptors proliferate in variety of types, while they trigger very basic g-proteins signals. Conversely there are few dopamine receptors which trigger many g-protein signals.

Also the reversal is apparent within their g-protein mechanisms. (Table 6)

D1 receptors : Stimulation of adenylyl cyclase,

5HT1 (5-hydroxytryptamine) receptors : Inhibition of adenylyl cyclase

D2 receptors : Inhibition of phosphoinositide-specific phospholipase C

5HT2 receptors : Stimulation of phosphoinositide-specific phospholipase C

Table 6: Types of primary neuromodulation receptors.

RECEPTOR	EFFECTOR (5-HT - Siegel et al., 1999) (D1-5 - Tarazi, 2001)
5-HT1A	Inhibition of adenylyl cyclase, opening of K+ channels
5-HT1B	Inhibition of adenylyl cyclase
5-HT1Da	Inhibition of adenylyl cyclase
5-HT1Db	Inhibition of adenylyl cyclase
5-ht1E	Inhibition of adenylyl cyclase
5-ht1F	Inhibition of adenylyl cyclase
5-HT2A	Stimulation of phosphoinositide-specific phospholipase C, closing of K+ channels
5-HT2B	Stimulation of phosphoinositide-specific phospholipase C
5-HT2C	Stimulation of phosphoinositide-specific phospholipase C
5-HT3	Ligand-gated cation channel
5-HT4	Stimulation of adenylyl cyclase
5-ht5A	Inhibition of adenylyl cyclase
5-HT5B	?
5-HT6	Stimulation of adenylyl cyclase
5-HT7	Stimulation of adenylyl cyclase,
D1	Stimulation of adenylyl cyclase, Stimulation of phosphoinositide-specific phospholipase C
D2	Inhibition of adenylyl cyclase Inhibition of phosphoinositide-specific phospholipase C Stimulation of Arachidonic acid Opening of K+ channels Closing of Ca2+ channels
D3	Inhibition of adenylyl cyclase
D4	Inhibition of adenylyl cyclase
D5	Stimulation of adenylyl cyclase Stimulation of Arachidonic acid

Dopamine receptors converge themselves to basic types with more functions (see D2), while serotonin receptors proliferate in many types with few functions. Also D1 has reverse effects of 5HT1 and D2 has reverse effects of 5HT2.

1.6.5 Lateralization and properties of Tyrosine and Tryptophan groups

Dopamine and its groups are lateralized in the left hemisphere, Serotonin and its groups are lateralized in the right hemisphere. (Table 5) Dopamine and serotonin receptors types are reversals of each other and have convergent (dopamine) / divergent (serotonin) properties. (Table 6) Dopamine has cation properties and serotonin has anion properties. As Cations are associated with negative charges then dopamine can be proposed to be convergent in system properties. As anions are associated with positive charges then serotonin can be proposed to be divergent in systems properties. To summarize Dopamine neurotransmitters and receptors have convergent systems properties. Serotonin neurotransmitters and receptors have divergent systems properties. As is pointed out (section 1.2_6,7)) the LCH is proposed to have convergent systems properties and the RCH is proposed to have divergent systems properties. So Dopamine neurotransmitters and receptors which are LCH lateralized fit with the predicted

convergent properties of the LCH. Serotonin neurotransmitters and receptors which are RCH lateralized fit with the predicted divergent properties of the RCH.

1.7 Summary of evidence for neuronal content distributions.

- Medical databases contain a wealth of data to illustrate that entire chemical groups and their primary subtypes like Dopamine can be grouped with GABA in the LCH and Serotonin with Glutamate in the RCH. NMDA receptors are strongly lateralized to the right temporal lobe, and GABAA receptors to the left temporal lobe. The analysis in table 5 highlights the secondary cortical content; Neuromodulators, dopamine, serotonin and their related chemical groups are also lateralized. Dopamine for the LCH and Serotonin for the RCH.

- It is proposed that all of these groups within each hemisphere exist on a LCH acid/RCH base axis system. GABA and dopamine are not only RCH lateralized but also tend towards acidic processes while Glutamate and Serotonin are not only LCH lateralized but also tend towards base processes. The acid / base lateralized systems for both primary neurotransmitters and neuromodulators exist in an axis. (as one increases in the brain the other decreases and vice versa)

- In systems theory terms, LCH neurochemicals and receptors have convergent homogenous properties while RCH neurochemicals and receptors have divergent heterogenous properties. This can be linked together on many levels.

- Convergent=Acid/inhibitory/hyperpolarizing Divergent=Base/excitatory/depolarizing.

Table 7: Summary of section 1.4,1.5,1.6

Primary cortical grey matter content	LCH Negative charge (convergent)	RCH Positive charge (divergent)
	Gaba	**Glutamate**
Primary Ionotropic neurotransmitters	Ionotropic Gaba receptors (Acid inhibitory, convergent, hyperpolarizing)	Ionotropic Glutamate receptors (Base, divergent,excitatory,depolarizing.)
	Polarized to temporal regions	Polarized to temporal regions
	Gaba modulators: Progesterone, LCH enzymes (phosphatidate, PIP2)	Glutamate agonists, Quinolininc acid, HMG CoA reductase, serotonin
Neuronal membrane for ionotropic neurotransmiiters	Gaba modulators (w3, DHA, EPA)	Glutamate modulators (w6, AA)
Modulating neurotransmitters	**Tyrosine Derived neurotransmitters**	**Tryptophan Derived neurotransmitters**
	Dopamine, Norepinephrine, Morphine	Serotonin, nicotine, quinolinic acid
	Dopamine receptors (Convergent)	Serotonin receptors (Divergent)
	Dopamine neurotransmitter (Acid)	Serotonin Neurotransmitter (Base)

Summary of the previous sections illustrate that cortical grey matter content is lateralized in line with what would be expected of a separation of charges. It should be borne in mind that these charges or acid/base distributions are still isolated within cells and neuronal membranes and not proposed as net charge or pH. The receptor types of both primary ionotropic neurotransmitters and neuromodulators also have divergent or convergent subtypes in accordance with the systems properties of convergent for a net hyperpolarization and divergent for a net depolarization action.

1.8 Dipole feature 9/ Frequency reduction and acid/base distribution towards the poles

The above summary indicates a complete dipole model of cortical grey matter content can be put together based on neurochemical acid/base properties. Known physiology points out that pH is kept at an overall balance throughout the brain. There is still a greater concentration of GABA and NMDA receptors at the brain's pole, so the accumulation of ionic gradients will tend to be more acidic at the left pole, and alkaline at the right pole. These larger amounts of acid/base pH are aggregated but isolated in the same manner that negative/positive gradients are in neuronal membranes. In that respect this is proposed to be the structural remains of a separation of charges that occurred in neurodevelopment. More importantly the systems properties of these charges as a net action still remain on neuronal function. The dipole separation of charges can be based on several levels. Polar separation of negative and positive ions used in neurotransmission (Cl-,Na+) and polar separation of acid/ base reactions. Both of these have an effect on the polar separation of Hyper/depolarizing processes which to have a mechanism for net coherence which will be discussed in section 2. This can be generally summed up in systems terms as convergent LCH/ divergent RCH increasing towards the cortex proposed poles at the temporal lobes. While constructing such a complete cortical dipole model it is also important to point out, that in an electric dipole as two charges move apart from the midline and distance increases their wavelength and wave energy decreases (Kane and Sternheim, 1988). In a similar way that the dipole was able to simplify small scale brain content, this frequency reduction aspect could have ramifications for the entire distribution of cortical processing

The cortical grey matter is proposed to be an electric dipole defined primarily by the separation of its charges at the temporal regions. Each ionotropic neuron synapse in the cortical layer is isolated as a series of point like charges into neuron assemblies connected by axons to its opposite charge counterpart across hemispheres. (this is a simplification of brain wiring of course). At the midline of this separation the corpus callosum, the brain processes visual sensory information, which has high electromagnetic frequency. Moving out from the midline the receptive fields which process vision increase with size to process larger aspects of visual information with lower frequency information. (Section 1.2(6)), (Bear et al., 2001) Moving

out further to the auditory cortex at the temporal lobes, the processing of frequencies for sensory input dips sharply to 20,000 hz. This higher frequency auditory information is processed first, then moving out further decreases in steps to just 15hz (Fox, 1987). In other words a complete and gradual reduction of the processing of electromagnetic frequency consistent with an electric dipole. Is this reduction consistent across other cortical regions ?

Moving forward in the cortex, the distribution of high to low frequency processing from midline out repeats. (high frequency, midline, lower frequencies to temporal regions) The motor strip deals with feet, then further out processes sensory information higher up the body, finally stopping at the face (Fox, 1987). This could imply the fastest cortex midline processing is needed for flight and fight reactions and a slower processing for communication. Forward again to the frontal lobes. At the midline is fast reacting emotional, spatial and memory pathways, while moving out from the midline some executive motor processing occurs in the farthest regions from the midline, again the slower requirements of speech and social interaction (Bear et al., 2001). This is not an in depth review of course, but so far appears consistent with the Cortical EMS concept of dipole frequency reduction from cortical midline out to temporal region.

1.9 Dipole feature 10/ Torque and polarity.

Electric dipoles undergo torque or precession, which means they spin at an angle within a magnetic field. In the following section (3), the cortical surface will be expanded into that of a bioelectric dipole dominated by magnetic fields. These magnetic fields can provide a solution to many mysteries about brain surface structure and function. Brain torque is feasible under the condition of a magnetic field at the cortex surface. The cortex would be an electric dipole sealed within a magnetic field. Brains have two known torque structures formed in childhood known as Yakovlevian and volume torque (Toga and Thompson, 2003). Yakovlevian torque is if the entire cortex is trying to twist round on itself. (figure 2c) Another is called volume torque, which looks like the left temporal lobe is shifting to the posterior in comparison to the right temporal lobe . The Cortical EMS model proposes possible solutions for both.

The proposed cortical surface magnetic model Neuronal Activity Associated Magnetic field (NAAMF) (Banaclocha, 2007) would develops magnetic field coherence when neurons fire their electromagnetic field to it. (Details expanded on in section 3) Under those conditions there is a theoretical explanation for whole brain Yakovlevian torque due to precession in neurodevelopment, (see section 2.6) as an electrical field is operating within a separate magnetic field.

In regards to Volume torque, the Cortical EMS model proposes that two opposite kinds of charge are present at either temporal lobe. Negative at the left and positive at the right, due to the ions Cl- and Na+ which predominate in either side primary ionic receptors. These are proposed to increase the amount of LCH GABA hyperpolarization (Cl-) and RCH Glutamate Depolarization (Na+).

In schizophrenia volume torque increases. Maximal left hemisphere volume was more posterior placed than on the right hemisphere and this negatively correlated with left superior temporal gyrus (STG) volume (Lewis et al., 2003). In schizophrenia the glutamate/sodium/NMDA part of the dipole in the right temporal lobe breaks down much faster than the left temporal lobe. (Section 1.5.3) This increased leftward asymmetry in schizophrenia can be read from the ionic field itself in EEG (Edgar et al., 2008, Flynn et al., 2008) with greater than normal gamma synchrony occurring at the LCH temporal lobe (Flynn et al., 2008). Such increased synchrony could perhaps be interpreted as a dysfunction in the hyperpolarized processes of the LCH temporal lobe. Schizophrenia is predicted by the Cortical EMS model, to be a breakdown of the brains dipole structure to such a degree that cortex wide electromagnetic torque is increased. Is volume torque near the LCH temporal lobes an asymmetry of collective charge, (net hyperpolarized processes) due to a bias towards hyperpolarized processes in the LCH temporal region?

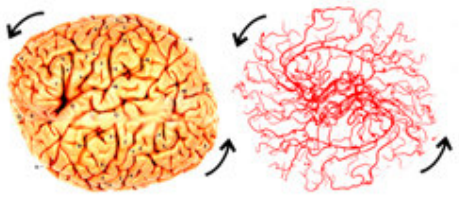

Figure 5: Left: Cortex mirrored (Adapted from Ellis et al., 1991) appears to have a direction of spin, which matches that of the blood supply. (right)) (Adapted from URMC 2009) Do these spin structures indicate that the entire cortex is undergoing torque which is dipole related ?

Key Points from section 1:

- All major features of dipoles have correlations with the cortex
- The cortex primary electromagnetic grey matter content : ionotropic neurons have lateralized distribution contents which accord with the charge distribution requirements for a dipole. That charge is distributed across an axis and separates at the furthermost poles.
- Associated processes to the grey matter: Neuromodulators, lipids and enzymes also lateralized in line with the ionotropic content, so most cortical grey matter content is lateralized in line with a dipole distribution.
- This distribution of ionotropic content and neuromodulators operates on an acid/base axis.
- Frequency reduction appears to occur in terms of neural processing across the cortex dipoles axis.
- Whole brain structure has spin or torque which can be altered by diseases like schizophrenia affecting the brains primary ionic function.
- As predicted by the summary of neuropsychology (table 2) the cortex primary grey matter content is consistent with the hemispheres systems properties of Convergent (LCH) / Divergent (RCH).

Section 2: *"Magnet's on the brain"* : Magnetic influence on brain structure, abnormality and function ?

2.1 Magnetic correlates for brain structure

Cortical EMS (Electromagnetic Multipole Solution) model attempts to provide a generalised electromagnetic solution to mammalian brain structure. Cortex surface structures and their underlying cortical columns have structural features commonly found in magnetic sciences. An example is magnetic layers such as ferrofluids *(A nanoscale magnetic compound mixed with surfactant such as lipids or an ion solution)* which produced stripe domain when under the influence of electric currents or magnetic fields. (Figure 6) Researchers have commented on the similarity of stripe domains to cortical folds (Stevens, P.,S. 1974 ; Ogawa, 1983).

The production of magnetic stripe domains on thin ferromagnetic layers have also been observed to produce similar observable appearance to the perpendicular anisotropic (upwards direction) domain ordering, seen at the surface of the brain. (figure 6a) When looking side on at these stripe domains, there are columns which provide an interesting analogy to the cortical columns themselves. (figure 6b) Sandwiching a ferrofluid between two plates and applying an alternating current also produces the same stripe domain result. (figure 3c) Cortical folds not only scale with size through all mammalian species, but manifest uniquely even between identical twins, suggesting that they could be related to the manifestation of a physical property. For example a common set of particles, proteins etc producing magnetic forces across species.

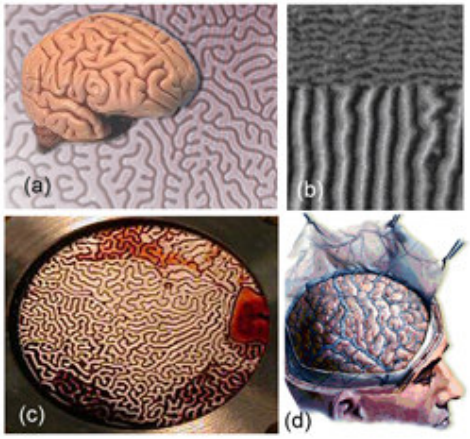

Figure 6: (a and c) shows thin layer ferromagnetic and fluid patterns are similar to cortical folds. Underneath the thin layer patterns columns form similar to cortical columns.(c) The brain veins are precisely aligned to these folds, more so than arteries. (d) As veins carry paramagnetic blood are these aligned due to magnetic forces ? (see section 2.5)

Figure references (Zani, G., 2000) (Cord and Westwood, 2001) (Cooper, 2003.) (Rubin and Safdieh 2007)

Magnetic compounds in Neuroscience

Magnetite and ferrihydrite are superparamagnetic iron compounds found throughout the brain, sometimes bathed in lipid's as hemosiderin (Quintana et al., 2006) with approx 3,420,000,000 magnetite particles at cortical surface, (Kobayashi et al.,,1995; Lindboe, 2003) Although the healthy brain is covered in these magnetic / lipid mixtures, currently they are thought to be a waste by product which accumulates, eventually leading to neurodegeneration. They are found mostly at the brain surface, astrocytes and regions associated with memory, such as hippocampus and cerebellum (Quintana et al., 2004, 2006; Brem et al., 2006; Dobson 2001). What ties all these brain region's together is that they are near the ventricular system which has access to neuronal astrocytes that can fill with CSF, (Guyton, 2000) magnetite and hemosiderin (Quintana et al., 2006). These are some of many possible components required for a ferrofluid. CSF has abundance of free lipids suitable for ferrofluids, linoleic, myristic and palmitic (105-638 microG/L) (Prasad et al., 1998). although astrocytes themselves are also production and exchange centres for ferrofluid components, being a site of magnetite precursors ferritin, and excess iron accumulation (Dringen, 2007). as well as apolipoprotein E. (apoE) apoE is one of the main apolipoproteins in the central nervous system which play an important role in lipid metabolism (Gong et al., 2007). CSF apoE is synthesized and secreted mainly by astrocytes (Gong et al., 2007). The lipoproteins contain mainly phospholipid (Gong et al., 2007; Williard at al., 2001) such as AA and DHA which as mentioned previously plays a key role in brain function and memory. (section 1.5.2c) Astrocytes can then be proposed to produce the required components for a ferrofluid. The problem with a purely ferrofluid based model for the cortex is that there is not a known sufficient density of magnetic particles for a typical ferrofluid. The reason it is being explored is that the memory properties of ferrofluids are a good place to derive a starting point in terms of the properties that such magnetic material or its variants in biology can supply to neural functioning. In this regard there will also be an attempt in this section to look at other possible magnetic models for cortex function.

2.2 Are the development of cortical columns and folds linked to magnetic fields ?

Memory speed, (Banaclocha, 2001) short term span and binding (Hameroff, 2007; Harrison, 2009) are not explainable by known molecular mechanisms (Banaclocha, 2007). These are all very different problems, yet current branches of theory and research tracking each of these aspects down, have mostly climbed from neuronal assemblies up to the brain surface itself. Several different researchers are looking at Astrocyte Gap junctions "hyper-neurons" for recording (Banaclocha, 2001) and binding mechanisms (Edwards, 2005). Banaclocha (Banaclocha, 2001) developed cortical surface electromagnetic models arguing that astroglial magnetic fields also induce neuronal firing, (Banaclocha, 2007) and proposes that there appears to be some kind "tape like" magnetic recording happen at the brain surface. Banachlocha proposes that astroglia produce magnetic fields sustain the information from neuronal firing, by holding it in a magnetic field, and that this plays a role in the formation of cortical columns in which cortical magnetite could be implicated (Banaclocha, 2001, Banachlocha forthcoming paper).

Overarching magnetic fields, such as those provided by astroglial fields are predicted to play a role in cortical column formation. Magnetic materials deposited on to an oily surface which are also under the influence of a larger magnetic field form geometric pinwheel structures (figure 7a,b) consistent with both the NAMMF magnetic model for the cortical columns having geometic structures produced by astroglial fields

(Banaclocha, 2007) and the well known pinwheels associated with hypercolumns. These pinwheels are more prominent in the upper cortical layers of the cortical hypercolumns (McLaughlin, 2000) proposed to be higher in magnetic interactions. (Banaclocha, 2007)

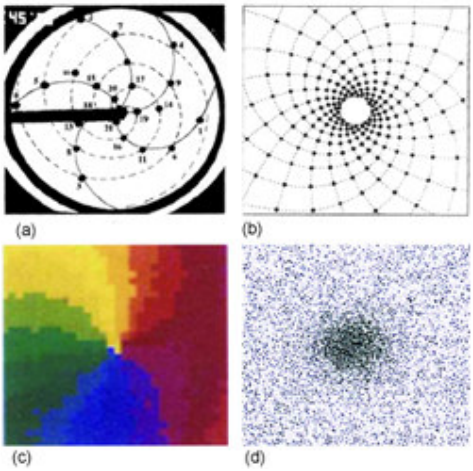

Figure 7: Magnetic compounds under the influence of a magnetic field dropped onto an oily surface (black points in (a)) represent a similar concept to magnetic compounds on lipid membranes under the magnetic field of the veinous system. These magnets spread out into a pinwheel formation similar to that found in cortical hypercolumn formation (c). The spin formations from experiment (a) when analysed converge around a central hole again similar to the central discs found in analysis of hypercolumn pinwheel formations (d)

Figure references (a,b Douady and Couder, 1992) (c Bonhoeffer and Grinvald, 1991) (d McLaughlin et al., 2000)

2.3 Memory and binding properties of Ferrofluids

The Magnetite and ferrihydrite found in the brain are ideal for a "spin glass" ferrofluid (Satinover, 2001). This is a magnetic fluid, which can hold more than one layer of memory. A spin glass material such as a ferrofluid is frustrated, because its ferromagnetic (Magnetite) and antiferromagnetic compounds (Ferrihydrite), are at odds with the strict order its iron atoms require in either of these magnetic compounds. (Satinover, 2001) Such "spin glass" compounds are being researched as the ideal requirements for memory in consciousness, (Satinover, 2001) and for computation (while frozen) (Lahaye et al., 2007; Balcells et al, 1992; Levy, 2002). At physiological temperature ferrofluids have Ferroelectric, (Ayton et al., 1995) spin glass, (Satinover, 2001) and associative memory (Palm and Korenivski, 2007) properties with a 3ms access time (Park et al., 2001). This 3ms latency correlates to neural mental processing limits, (Weiss, 1992; Lago and Kon, 2004) as well as unknown mechanisms for gap junction coherence in the astrocyte networks mentioned, (Womelsdorf, 2007; Weiss, 1992) and high gamma 4ms. It is still not clear however if magnetite/lipids are a requirement for these ferric properties or if an ion/ferritin solution would also produce similar results. However the magnetic properties of either those proposed here, or some kind of similar biological materials are hypothesised as necessary.

Brain function is proposed to require associative processing for object recognition ("superposition catastrophe".(Vianin et al., 2002)) and context updating when probabilities tied to certain outcomes are refreshed (Polich, 2007). Such associative processing requires several short term memories to be held together at once (Vianin et al., 2002). Information associated with these proposed multiple short term memories can be stored in a low frequency sub cycle of cortical Gamma, (Weiss, 1992) although where they could be held is unknown. Banaclocha describes such low frequency cycles (theta) as the activator of cortical memory (Banaclocha, 2007) which he proposes is held in magnetite located in astrocyte cell membranes. (Personal correspondence Banaclocha 2009) This magnetite/membrane configuration for Neuronal activity associated magnetic fields (NAMMF) (Banaclocha, 2001) is consistent with the outlined ferrofluid proposals for short term memory, and the concept that the cortex surface structure is a complex manifestation of magnetic stripe domains.

2.4 What is the mechanism that hits "record" ?

For cortical activity to be sustained as short term memory beyond neuronal spiking requires some kind of specific activation mechanism. Banaclocha proposes that at least 100–300 astrocytes would be magnetically influenced when a neuronal minicolumn is active (Banaclocha, 2007). The electrical coupling between these astrocytes play a role in the synchronization of ripples, high gamma oscillations. (Edwards, 2005) These oscillations may feedback through the corticolimbic system, signalling that there is a large network of firing neurons (Bazhenov, 2006). Cortical ripples are proposed as a possible record mechanism as they are marked by a shift in the functioning of working memory (Bazhenov, 2006). If ripples are coupled to astrocytic activity then this could induce a greater magnetic field in accordance with NAAMF. Any existing magnetic particles will generate heat under a changing magnetic field (Matsuoka et al., 2004). This provides a possible record mechanism as such heat is a required component to alter a ferrite based memory from its base state and allow new information to be written (Palm and Korenivski, 2007).

Evoked signals from sensory input which propagate throughout the cortex are synchronised by a superposition of low frequency activity (Klimesch et al., 2004). This increases the likelihood that thalamic sensory inputs and hippocampal memory can be integrated within these models. Each component of sensory information could be linked to a representation of evoked potential in the gamma range (Klimesch, 1996) where each node within it can be associated with a unique phase (VanRullen and Koch, 2003; Prechtl et al., 1997). As ferroelectric fluids respond to AC (Alternating current) such as Gamma, theta, alpha etc) a model can be constructed whereby multiple memories, or phase components are held within cortical ferrofluids, each being triggered by their respective AC carrier. In other words these oscillations provide tape recording style signal bias for multiple evoked signals. The superposition catastrophe and sustaining of cortical activity patterns could be feasible utilising these proposed cortical magnetic mechanisms.

2.5 Cerebral vein structure as a marker of magnetic activity.

Cortical veins are more tightly aligned to cortical folds (proposed to be stripe domains) than arteries, and large veins themselves were used in section 1.1 as markers of cortical dipole structure. Sagittal sinus was proposed as a midline toroidal structure. (table 1) As well as this the

temporal veins are used to mark out Cortical EMS poles. (figure 4b) These large veins carry paramagnetic deoxygenated blood. Arterial blood gives up oxygen at astrocytes (Heeger and Ress, 2002) producing a shift in the red blood cells magnetic field. This "Hemodynamic" shift is called BOLD (Blood-oxygen-level dependent). Cortical columns are activated as this blood shifts from diamagnetic to paramagnetic in capillaries (Malonek and Grinvald, 1996; Grinvald et al., 2000; Kim at al., 2000).

Astrocytes are involved in the cascade of neural processes underlying the hemodynamic responses, while (Brovelli et al., 2005) hemodynamic shifts are directly linked to astrocyte function and activity (Heeger and Ress, 2002). In addition to unknown mechanism for sustaining attention based visual cortical activity patterns, attentional visual input is proposed as having a perceptual hysteresis. That is sustaining in short term memory more of the image than can be perceived (Oliva and Brady, 2008). Perceptual hysteresis correlates both with the fMRI BOLD signal (Avidan and Behrmann, 2002) and the brain areas marked out by large veins : occipital, frontal, parietal, temporal (Kleinschmidt et al., 2002). As As pointed out veins are more tightly aligned to cortical folds than arteries and prominent at cortical dipole related structures. It could then be proposed that veins are indirect evidence of cortical magnetic activity as paramagnetic materials respond would align to astrocyte magnetic fields.

With this analysis showing clear magnetic structures, the cortical dipole model cannot simply be a bioelectric dipole model. It will be proposed as a bioelectric/magnetic dipole hybrid such as ferroelectric, with the magnetic functions re-enforcing the polarization of otherwise isolated cortical charges. The sustaining of cortical activity patterns by magnetic interactions re-enforces cortical structures and gives a "robustness" to cortical processes. It is these magnetic mechanisms which would have to facilitate the highest re-enforcement of cortical hyperpolarization LCH / depolarization RCH required for a dipole distribution. So aggregation of the mechanisms and components for this re-enforcement at the temporal regions would be a prediction of this model. Already covered (Section *1.5.3., 1.5.4)* the highest density of ionotropic neurons and veinous system, are in these regions, and administration of lipids alters lateralization of brain structure associated with ionotropic content most in these regions (Section *1.5.2d).* Also magnetite and astrocytes would be predicted for a higher distribution in these regions. This may be consistent with the findings that cortical magnetite is higher in Alzheimer's and the areas affected most by Alzheimer's are those with higher neuronal and therefore higher astrocyte densities at the temporal regions (Thompson et al., 2003). To summarize combining magnetite/lipids with NAAMF's can generate possible solutions to several unknown cortex structure, functions and abnormalities.

2.6 How does the dipole structure form ?

Although images of magnetic and electric dipole field lines are similar, the cortical axons appear to have a greater similarity to the images of magnetic dipoles from a ring electromagnet. (figure 1) As has been pointed out for such magnetic structures to arise there would need to be a ring of current round the corpus callosum to produce such a purely magnetic field (Personal correspondence W. Bains, 2009). Although It is beyond the scope of this paper to provide every solution and mechanism for the dipole model it should be pointed out that the corpus callosum does have a semi complete ring of axons wrapping round it and that axons themselves are known to produce perpendicular magnetic fields (Roth, B.J., Wikswo, J.P.Jr., 1985). These are the cingulum bundle as well as the axons of the longitudinal fasciculus. Axonal formation appears to be the final structure to form in neurodevelopment, (bear et al., 2001) so is unlikely to play a major role in the required separation of charges to bring about a Cortical EMS model. The main problem with providing a mechanism for dipole formation is that available neuroscientific data pertains to adult cortex, and the cortical (hence dipole) structure forms in development. For a biological structure to form into a polarized organ there would have to be some kind of magnetic coherence occurring in the neural tube in neurodevelopment. Gap junction connexins that increase electromagnetic coherence (and hence net charges or magnetic interactions) between neurons, astrocytes and axons are often measured by neuronal synchrony. (Hameroff, S., 2007,2009.) These connexins are most prominent in development. (Nadarajah, et al., 1997; Sadowska et al., 2009). One of these connexins (Cx32) also found in the corpus callosum, (Kamasawa, N., et al., 2006) is marked out as playing a role in synchronous oscillations during development. (Nadarajah, et al., 1997) The cortical dipole structure is then predicted to emerge in development as a result of magnetic or electromagnetic coherence which does not exist to the same degree afterwards.

The sequence these Connexins arrive match the sequence required for Cortical EMS. This would be an overall magnetic structure that can facilitate a separation of charges across hemisphere. The connexin Cx43 associated with astrocytes and cortical surface migration (Magnetic coherence in NAAMF's) remain throughout the entire neurodevelopment period of the cortical structure (Nadarajah, et al., 1997). Radial cells are key lines of passage for neurons to move from proliferative zone to cortex layers in the cortical neural tube (Wolpert et al., 2002). The radial cells are extensions of astrocytes and have much the same processes which play a part in the proposed magnetic fields of NAAMF, such as calcium waves mediated through connexin channels (Weissman et al., 2006). Cx43 was present throughout migration associated with astrocytes (cortical surface) (Nadarajah, et al., 1997). Although neuronal migration was not greatly affected when mice had electrical coupling removed from Cx43 (Elias et al., 2007) it should be pointed out that Cx43 is not proposed to play a leading role in electrical coupling between astrocytes (Scemes et al., 1998). There are also other astrocyte connexins involved in electric current coupling such as Cx40, Cx45, Cx46 (Elias et al., 2007). Cx47 also plays a role in mediating electrical coupling of astrocytes to axons (Kamasawa, N., et al., 2005). It is then not possible to provide a complete electrotonic picture of radial glial cell connexins during neurodevelopment except to say that they appear to be consistently present throughout the developmental period. (Nadarajah, et al., 1997). The connexins next to emerge are those with neuronal formation Cx26, followed by those associated with white matter Cx32 (Nadarajah, et al., 1997). Both Cx26 and Cx32 subside while astrocyte connexins remain. (Nadarajah, et al., 1997).

This is entirely consistent with the Cortical EMS model. If neuronal activity is consistently surrounded by a coherent magnetic field (astrocytes, radial glia) the result could be a separation of charged ions across hemispheres with a resultant effect on neuronal structure. Although at this stage its not possible to provide a specific mechanism sequence in terms of development proteins. There is also likely to be magnetic repulsion or a very broad domain wall between hemispheres if there is both magnetic and electric coherence. This is because in ferroelectric dipoles, (the cortex surface being proposed to be ferroelectric) the domain wall is usually far broader than in ordered dipoles such as a magnet (Kirichenko et al., 2003). That white matter development (and hence field lines) then follow the proposed separation of charges is no surprise. Axons, the most visually obvious feature of a cortical dipole formation (figure 1) are then proposed to be the traces of the initial magnetic structure (cortex surface) which separated the neuronal charges across hemispheres. Once matured Connexin gap junction activity has to decrease or else the cortex would constantly be altering its electromagnetic structure. In this respect it is also interesting to note that laterality switches back and forward between hemispheres several times during childhood, finally slowing down and stopping this process towards the age of eight (Morris, 1978).

To summarize, the white matter of the cortex (or entire brain) are proposed as the magnetic aspects of the dipole structure that is noticeable by its magnetic field lines. (figure 1) The grey cortical matter would be proposed to be that of an electric dipole with the separation of charges giving a net polarization of inhibitory or hyperpolarized activity at the LCH pole. Conversely also a net polarization of excitatory or depolarized activity at the RCH pole. Biology being this complex, it's not possible with the current findings to propose a simple electric or magnetic dipole. There would be a complex hybrid form of a dipole structure, as marked out by the differences between the cortical dissection and that from a dipole simulator. (figure 1) The difference being that the cortex field lines are split at the midline as if there was magnetic repulsion occurring there between the hemispheres. As well as this the cortex surface bears no resemblance to any known dipole simulation, but does bear a resemblance to magnetic domains. (figure 6) However investigation of the cortical surface has given rise to magnetic mechanisms by which a net excitatory RCH / Inhibitory LCH activity can sustain the structural activity of a dipole formed in neurodevelopment (Banaclocha, 2007).

Key Points from section 2

- **Structure** : Cortex surface appearance and column structure explained in terms of magnetic interactions.
- **Function** : Some form of magnetic model would give solutions for associative processing and sustaining of short term memory.
- **Abnormality** : Magnetic materials age. This could degrade the finely tuned balance of cortical systems function possibly contributing to the process of neurodegeneration in Alzheimer's.

Section 3: Limbic system Multipole Analysis

3.1 Limbic system as a linear multipole.

What remains of the brain, after explaining away the cortex structure and surface as a complex radiating electric/magnetic dipole is the limbic system, brainstem and cerebellum. To make analysis easier only the limbic system will be concentrated on. The Cortical EMS theory arose primarily from observations on human dissections and behaviour, and so its prime focus is on what the brain has developed into on top of its non mammalian brain roots. How to analyse the limbic system has been more problematic than the cortical dipole, as there is not so much data available on lateralization. The approach taken is similar to that used to generate hypothesis for the cortical dipole. First remodelling how the limbic system would look if was spherically mirrored to fill its incomplete loops. (section 1.1) Then following that build up a database of images from electromagnetic simulators for shape and pattern comparison between them to be considered. With limited data on limbic system lateralization, reviewing EEG research on the limbic system will be the means to establish if there is evidence to propose it possesses the following electromagnetic structures.

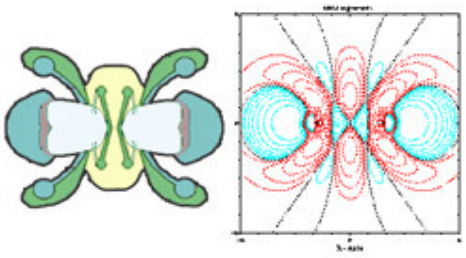

Figure 8: Right, one pattern of linear Quadrupole simulation. (Harrison, 2004) Left, Diagram of limbic system mirrored. (drawn from Review of the Universe, 2009)

As can be seen in figure 8, the limbic system has some rough correlates with patterns produced from linear quadrupole simulations, although at this stage it's not specifically exact - there could be other linear multipoles which fit better. Linear multipoles are defined both mathematically and visually by spherical harmonics, which are functions that produce 3 dimensional variations on toroid's and symmetrical lobes. (figure 9) Although neurological areas are extremely distorted in comparison to spherical harmonics, overall the shapes of each part of the limbic system are surprisingly consistent with these components of linear multipoles.

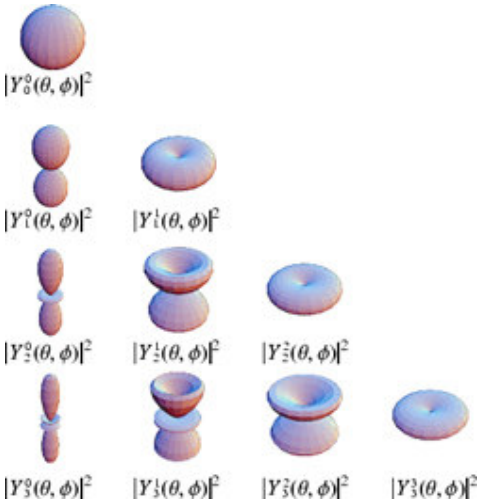

Figure 9: Spherical harmonics (Weisstein, E., 2009) are mathematically variable subcomponents of complex electromagnetic multipoles. The variations revolve around three main shapes and their combinations. Lobes (hourglass), Toroids (doughnut shapes) and Disc pairs.

Table 8: Breakdown of limbic system into spherical harmonic variations.

Brain Segment	Shape	Harmonic
3rd Ventricle	Toroid	Y 0/2
Thalamus	lobes	Y0/1
lateral Ventricles	Discs (2)	Y 1/2
Caudate Nucleus	Discs (2)	Y 1/2
Fornix / hippocampus	Discs (2)	Y 1/2
Globus Pallidus	lobes	Y 0/1

Each part of the limbic system when mirrored has a shape which is a variation of basic multipole wavefunctions..

In table 8, each part of the limbic system when mirrored has correlates with the basic shape components of multipole structures: Lobes and toroid's which depending on their position along the multipole axis can be sheared, flattened or compressed into further variations. (figure 8) Toroids when sheared and flattened become curved discs. The limbic system components, (including the ventricles) when mirrored all tend's towards disc or lobe shapes. Using these the limbic system can be taken apart in a 3d modeller and rebuilt as a linear multipole (figure 10) using spherical harmonics the components of electromagnetic multipoles.

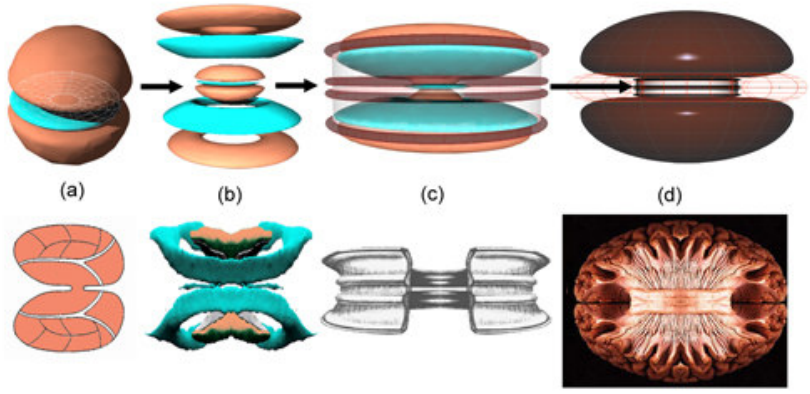

Figure 10: Observable analysis of primary limbic /cortical system regions when mirrored and rebuilt using spherical harmonic components. The entire limbic system can be modelled from variations of spherical harmonics the components of electromagnetic multipole expansions. (top row author image, bottom row adapted literature images: references below. Brain regions are in the bottom row, spherical harmonic equivalents are represented above them) (a) This shows that the thalamus has a basic hourglass lobe form, and has a third ventricle between it, which is a toroid. (b) The third ventricles and caudate nucleus are mirrored produce sheared discs. Hippocampus is similar but has been left out for simplicity. These are added to thalamus third ventricle from (a). (c) the induseum griseum of the corpus callosum, a continuation of the hippocampus has toroidal structures. These are added to (a) and (b). (d) (a,b,c) are enclosed in the cortex which is a large lobe structure. (cortical surface folds left out due to modelling restrictions.) Again a toroidal structure occurs in midline. (see section 1.2) Consistent with the concept that it is only the limbic system which can be modelled in this manner the induseum griseum and cortex although possessing some of the structures (toroid's and lobes) don't appear to possess an overall pattern in line with any of the spherical harmonics. The indiseum griseum has four toroid's and cortex structure overall cannot be approximated.
Figure References (a Adapted from Best, B., 2009) (b Adapted from Sundsten,, 2009) (c Adapted from Nieuwenhuys et al., 1988) (d Adapted from Williams and Gluhbegovic,1980)

In section 2, Understanding the glial system with its magnetic interactions near the subarachnoid space in cortex, has given rise to theoretical solutions for both cortical brain structure and electromagnetic function (section 2). Functionally it is not surprising that glial cells are so important considering that the CSF system is the basis from which the cortical dipole structure is proposed to develop (section 2.6). For this reason a similar solution will be sought for the limbic system by comparing the lateral and third ventricle visually (figure 11) with the very basic components of the simplest linear quadrupole. The ventricles have similarities not only in shape but also correlate with the charge requirements for a linear quadrupole neurodevelopmental model. Such charge requirements would be the alternating current function of a linear Quadrupole.

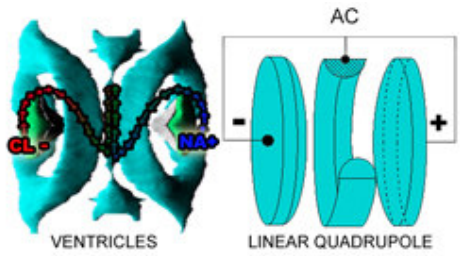

Figure 11: Ventricles (mirrored) are compressed toroid's with the third ventricle being a toroid. (b Adapted from Sundsten,, 2009) These bear observable resemblance to the ion firing structures of linear quadrupoles in both shape and the electrical components which will be analysed in the following sections.

3.2 Limbic system EEG correlations with a Quadrupole structure.

Linear electric multipoles, such as the linear quadrupole are the inverse of radiating dipoles. They activate centrolineally opposite charges from poles at either end, moving towards two more poles at the midline. These midline poles produce tight adjacent alternating currents, due to the swapping of charges in a confined area. The tight oscillations and stability of electromagnetic energy at an electric quadrupole midline creates a central cavity or disc that has a lens effect. This symmetric electrical lens can be used for the generation of atomic clocks (Allan, 2004).

A central cavity exists at the midpoint of the brain – the third ventricle has disc type structure, although it's not proposed here that this is functional in electromagnetic terms. What is interesting is that the main oscillating (alternating current) EEG signals, Alpha, Delta and Theta, which clock the brain (Brazier, 1980) are produced at the midpoint of the proposed linear quadrupole structure. For example alpha rhythm and delta rhythms have been correlated to the third ventricle area (Brazier, 1980; Karson et al., 1988) with alpha being proposed to arise from neurons bordering the third ventricle, (Karson et al., 1988) while the septum, a thin membrane between the lateral ventricles, plays a key role in brain wave generation of the theta rhythms. (Ujfalussy et al., 2007; Sotty et al., 2003; Lawrence et al., 2006; Brazier, 1980) The neurons in this area are known as septal "pacemaker" neurons (Wang, 2002; Kocsis and Li, 2006) for their timing abilities. The main alternating frequencies of EEG appear to originate where the lateral ventricles meet, and the third ventricle but not in either lateral ventricle alone. Is this due to a linear quadrupole midline lens structure, (Allan, 2004) reconstituted in neuronal processes ? Lateral ventricles are involved in Sodium and chloride ion production, producing quantities of sodium, bicarbonate and chlorine for Cerebrospinal fluid (CSF) fluid homeostasis (Rabary, 1994). This CSF enters astrocytes at lateral ventricles choroids plexus,(Guyton, 2000) and so the ionic content of CSF could effect neurons by volume transmission. Because of the amount of dipole consistent lateralized brain content found in the cortex, including the opposite ions of sodium and chlorine at either pole, (see section 1) the Limbic EMS model predicts that neural content of the limbic system be consistent with the electric multipole structure, and that ion content at left and right lateral ventricles choroids plexus could differ between hyper and depolarizing, with a resultant effect on the developing neurochemistry of the area.

3.3 Harmonic oscillation in Linear Multipoles

As was shown in remodelling (section 3.1) the limbic system mirrored and smoothed out transforms observably into structures similar to the spherical harmonics that define linear multipoles. Linear multipoles (such as a linear Quadrupole) fire from poles to midline, and produce natural periodic oscillations which lock phase producing harmonics. This is why the midline of quadrupoles are also used for spectral measurement, as electromagnetic input's to a harmonically oscillating system will naturally resolve themselves into their periodic sub-components. Are these harmonic properties found in the limbic system brain structures ?

Researchers involved in analysis of the computations performed by the thalamus find that it performs measurement of incoming information for compression which have similarities to spectral computation. For example thalamic nuclei targeted by corticothalamic input orthogonalize their inputs to matrix cells (Granger and Hearn, 2008) that is they reduce spatial ordered information to subcomponents (Granger and Hearn, 2008). As a similar analogy, spectral decomposition computers perform orthogonal transformations for eigendecomposition *"whereby the matrix is represented in terms of its eigenvalues and eigenvectors."* (Eiegen : term for harmonic subcomponents) (Horn and Johnson, 1985,1991) The operations to derive these subcomponents are using basic linear operations (Walther, 2000; Dhar and Banerjee,1995) associated with superpositions. As mentioned in section 2, superpositions occur in limbic system EEG (Klimesch et al., 2004). This provides some background to illustrate that the limbic system is primarily a system that generates the linear processes which are part of harmonic systems.

What kind of information breakdown occurs in the thalamus is still a subject researched with differing results- Spatial compression (Pelaez, 2003) and pattern recognition of sensory data (Rodriguez et al., 2004). Both have similarities to spectral decomposition. Conceptual harmonic processing models in neuroscience produce chains or sequences of information derived from sensory input (Weiss, 1989). Thalamocortical derived algorithms based on lab analysis also produce chains of reduced information from sensory input (Granger and Hearn, 2008). There is then reason to conceive that this key area of the limbic system is operating using harmonic processes.

In a harmonic system periodic oscillations lock phase. Continual phase locking amongst the lower EEG signals is a property unique to the limbic system. Phase locking between the EEG of two primary limbic components, hippocampus/theta and thalamus/alpha is responsible for the regularity of conscious perception and its integration with memory (Gruber et al., 2005; Klimesch, 2004). This phase locking is not so prominent in the cortical EEG generated above 29hz, where resonance tends towards amplifying input's from noise (Rodriguez et al., 2004; Ward et al., 2006; Moss et al., 2004; Tiesinga and José, 2000; Ringach and Malone, 2007) and synchrony is generally considered to be sporadic and dominated by noise. (section 4.4) The cortex depends on the limbic systems lower frequencies for its overall functional synchronization (Brazier, 1980; Gruber et al., 2005; Klimesch et al., 2004). By contrast the limbic system could be said to be primarily defined by superpositions of synchrony (Klimesch et al., 2004). It is generally accepted by physicists that superpositions of synchrony produce harmonic interactions. Up to 29hz (limbic system) EEG activity has been linked to increased harmonic multiplication, (Weiss, 1992) while In response to anaesthetic, harmonic components in alpha (thalamus) have been found (Nunez and Srinivasan, 1981,p232-298). To summarize, not only can the limbic system be proposed to process like a linear harmonic system by review of its computational analysis, its EEG also reflects this proposal, and in regards to this is markedly different to the cortex, where researchers point out that the differences between cortical and thalamic EEG are linear and non linear respectively (Nunez and Srinivasan, 1981, Anokhin et al., 1999; Crick and Koch, 2003).

Key Points from section 3.

- Although distorted by evolution within a skull, and non mammalian brain regions the limbic system when mirrored has overall observable correlates with the harmonic components of complex electromagnetic multipoles, such as a linear quadrupole.
- Ventricle system is proposed to correlate with the ionic structure of a linear quadrupole, with the choroids plexus of these ventricles being a source of ion production. The brains primary coherent EEG (alpha, theta, delta) emerges from neurons that border these third and lateral ventricle areas.
- The thalamus processes information in a manner that parallels the spectral breakdown of a harmonic system. Phase locking a property of harmonic systems is found occurring between the EEG of the hippocampus and thalamus, but does not occur in the cortical EEG. Other studies find that the limbic system EEG can be proposed to have other harmonic properties such as superposition, synchrony, harmonic and linear processing.
- The limbic system then also has many correlates with the harmonic structures and processes of a linear quadrupole and is proposed to operate with the properties of a harmonic oscillator.

Section 4: Dipole /Quadrupole integration

4.1 Thalamocortical system breaks down to linear/nonlinear hybrid system

Having the right balance between these cortical/nonlinear and limbic/linear systems is an essential part of successful brain function (Anokhin, 1999; Crick and Koch, 2003; Cytowic, 1995). The Thalamocortical relationship is defined as "Oscillation-Assisted Processing", where sensory input modulates self generated cortical activity (Granger and Hearn, 2008). In other words the cortex appears to have processes which generate increased power from its inputs (Wu et al., Xu et al.,2007). As previously mentioned this power increase was partly due to noise assisted processing, (section 3.3) and in systems term the cortex is random and chaotic (Crick and Koch, 2003) with the thalamus in the control seat, linearly regulating any cortico-cortico processes. For example, thalamocortical inputs trigger a propagating envelope of gamma-band activity in auditory cortex (Metherate and Cruikshank,1999). This propogation in turn results in thalamic matrix nuclei being strongly driven by corticothalamic delta feedback (Granger and Hearn, 2008). One function of this thalamic matrix is to synchronize cortical oscillations via projections back to cortex (Granger and Hearn, 2008). This "Oscillation-Assisted Processing" is a continual thalamocortical integration, whereby alpha band constrained sensory input (thalamus) modulates self generated cortical activity (Granger and Hearn, 2008). As thalamus and cortex are linear and non-linear respectively, the thalamo-cortical system which provides a major contribution to conscious processing, can be said to be a continual interaction between a linear and non-linear system.

Is the limbic system an ordered, high latency linear system which tends towards long term encoding of information and so is less-dissipative in its processes ? Gamma oscillations - the sole means by which cortical coherence is measured, is considered a slave to the lower frequency and brain wide limbic system oscillations (Bonhoeffer and Grinvald et al., 1991). Is the coherence to cortical process given by gamma oscillations dominated by cortical decoherence ? Sex differences in brain structure and function can shed light on the primary processing features of cortex and limbic system. Female processing on similar tasks to male processing is marked out by connective (white matter) utilization as opposed to male utilization of grey matter (Haier et al., 2005). The ratio of grey to white matter in the cortex is 10:6 roughly equivalent to the ratio of subcortical white to grey matter 10:5 (Collins et al., 1998) (table 9). So female processing tends to be biased towards white matter connective processes more associated with the limbic system, and male processing more biased towards the noise based non linear amplification processes associated with the grey matter. Female processing is marked out by greater degrees of faster acting gamma oscillations than males (Hamilton et al., 2001). These fast acting gamma oscillations are the result of greater connective interneurons (Bartos et al., 2002) known to modulate brain wide structures such as corpus callosum (Hausmann and Güntürkün, 2000). and are bilateral, as opposed to lateral for higher latency gamma in males (Hamilton et al., 2001) implying that the corpus callosum is involved in the initial onset of gamma oscillations. As has been pointed out brain oscillations are dominated by the signals from the white matter based limbic system, so the point can be made that the oscillations arise from connective matter. The cortex is a more disconnected part of the brain system, (with connections tending towards local interactions) where noised based amplification dominate oscillatory processes.

Table 9: Volumetric analysis of human grey/white matter (Collins et al., 1998)

Matter volumes	Cm3	Root of Cm3
Total Cortical grey matter	814	9.38
Corpus Callosum	180	5.65
Total remaining white matter	565	8.27
Sub Cortical grey matter	54	3.80

These cortical amplification processes lead to increases in self generated activity resulting in "desynchronization", or the event related desynchronization, (ERD). ERD is a reduction of cortical non-phase-locked gamma oscillations (Polich, 2007). A "decrease in coherence across space", (Edwards, 2007) alpha event related synchronization (ERS) suppression, or deactivation (Polich, 2007). In other words in cortex, there is an increasingly chaotic non-linear divergence from a coherently linear thalamic entrained alpha signal as more cortical areas increase self activation by stimulus. The thalamus then serves to control this cortical tendency to self generated increases of activity. This thalamic / cortical interaction of controlled chaos is considered by some neuroscientists to offer increases in the speed of response or reduced latency in neural processing (Crick and Koch, 2003). That is the non linear processes such as in the cortex offer higher response speeds than linear ones from the thalamus. For example cortical activity which can reach a peak of Gamma 250hz is close to the limit of mental processing of 4ms (Weiss, 1992; Lago and Kon, 2004). By contrast the sensory sampling associated with an alpha wave (thalamus) actually decelerates with reverse convolution towards latencies of up to 250ms or greater (Weiss, 1992) and so tends towards increased latency of sensory processing.

Summary of findings in section 4.2

- Limbic system is white matter orientated, globally connective and tends towards producing synchronised oscillations, while the cortex is grey matter oriented, less connected (with connections being more localised) and tends towards amplifying inputs from noise.
- Cortical amplification processes tend towards decoherence and chaos which increases processing response times. The thalamus controls cortical chaos and tends towards higher latency processing.
- Non linear systems operate on local processing which is noise based amplification that shifts dynamically from one local region to another, based on response to external stimulation. Linear systems are more globally connected, synchronised and oscillatory tending to slow down sensitivity to the external environment.

4.2 P300 as a marker of Dipole/Quadrupole integration

The limit of sensory sampling occurs after about 250ms (Weiss, 1992). ERP (Event related potentials) are signals evoked by perceived and unperceived stimuli which start to diverge around 270 ms. Following this Increases in cortical processes at 300ms triggers a sequence of processes such as perception stabilization, maintenance in working memory, and generation of expectancies that are associated with conscious awareness, which are represented by an important ERP called P300 (Melloni et al., 2007). As it is proposed that P300 represents the integration of the outer dipole and inner quadrupole, then Dipole and Quadrupole processes should be well represented in study of the P300. Of course a logical limitation of this proposal is that it would be easy to state that smaller brain regions are encapsulated within these large electromagnetic structures. This limitation will be addressed by taking a more detailed look at the EEG expected from these structures.

One component of the ERP P300 is phase-locked delta (Polich, 2007) which is cortical to thalamic feedback (corticothalamic) (Bazhenov and Timofeev, 2006). As cortical activation increases, corticothalamic feedback may increase in response due to the looping interaction of the thalamocortical system. The P300 is also accompanied by thalamic alpha phase reset (Fell, 2004). Changes in cortical Gamma activity are partially due to this alpha phase resetting as the alpha wave phase reset contributes to the Generation of ERP such as p300 (Polich, 2007; Hanslmayr et al., 2007). These reset changes are common to non-linear oscillatory systems (which the cortex is proposed as, albeit more non-linear than oscillatory) in response to a perturbation such as viewing a flashing light (Brandt, 1997). ERD (desynchronization) onset which occurs due to cortical decoherence (Edwards, 2005) is positively associated with P300 latency – and negatively associated with P300 amplitude (Polich, 2007). In other words faster increases in cortical activation, lead to higher power in the lower latency ERP's such as P1-N1 (100ms). The higher latency ERP N400 (400ms) is initially associated with thalamic generated alpha wave phase reset, (Polich, 2007; Hanslmayr et al., 2007) without the power increases seen in lower latency ERP's (Fell et al., 2004). This is consistent with the previous concept that the cortex has lower latency than the limbic system, but tends towards faster self propagation on stimulus.

To summarize section 4.2, the lower ERP's below P300 represent more cortical process while the later acting ERP's above P300 represent more limbic controlled processes. The P300 which is the most functional and important of all the ERP's represents the critical integration point between cortical and limbic systems (Melloni, et al., 2007). The limbic / cortical integration cycle, here appears to be sensory input leads to cortical propagation (which can also be termed wave expansion (Xu et al., 2007)) and desynchronisation or decoherence. The resulting decoherence increases corticothalamic feedback, with a reciprocal thalamic alpha phase relocking and as mentioned previously (section 3.3) a possible spectral compression of the cortically feedback information occurring in thalamus. The integration point between inner quadrupole and outer dipole manifests itself as ordered thalamic control of cortically expanding chaotic wave activity, which is proposed in part to generate the P300. In systems terms this is perhaps conceptually similar to the chaos/regularity transitions which occur when harmonically oscillating linear systems are exposed to non linear amplifications (Bolotin, 1995).

4.3 Collapse/Expansion proposal for thalamocortical / septohippocampal system.

As well as thalamic alpha phase reset the P300 is composed of both phase-locked delta (which is a signal activated by corticothalamic feedback (Bazhenov and Timofeev, 2006)) as well as theta-range synchronized oscillations (Polich, 2007). A composition of theta / delta locked oscillations and alpha phase reset, implies that the septohippocampal system (memory) is working in conjunction with the thalamocortical (sensory) system (Aggleton and Brown, 1999). The septum which joins the lateral ventricles is key in generating the theta rhythms which play a key role in hippocampus function. Within the septum the medial septum-diagonal band (MSDB) complex area plays a key role in brain wave generation of the theta rythmns (Takeuchi et al., 1994, Sotty et al., 2003; Lawrence et al., 2006) implicated in spatial cognition, attention and memory processes (Vinogradova et al., 1998; Wang, 2002). In cats the septum has been found to be a modulator of P300 [152] Lesions in this septal area inhibit information gathering, (Ikonen, 2001) and the P300 disappears (Apartis et al., 1998). This then would include the septo-hippocampal region as contributing to the P300 generation.

These two major systems (thalamocortical / septohippocampal) which could be said to integrate the dipole (cortex) Quadrupole (limbic system), operate together marked by generation of the P300 (Aggleton and Brown, 1999). What occurs at P300 when the thalamocortical / and hippocampal cortical processes work in unison ? The Theta (hippocampus)/ Alpha (thalamus) synchronization between these two systems is responsible for the regularity of conscious perception and it's integration with memory (Gruber et al., 2005; Klimesch et al., 2004). The thalamocortical and septohippocampal systems sync together making a complete working inner brain system. (Striatum and reticular activation is also important but not a priority to completion of this model) At the point of P300 it is proposed the thalamus takes in cortical information, a process signalled by alpha wave reset, (Polich, 2007; Hanslmayr et al., 2007). and as mentioned previously (section 3.3) decomposes this mix of cortical signals to subcomponents before cortical decoherence. The P300 happens at the point just before cortical potentials propagate enough to become decoherent through event related desynchronisation (ERD) (Polich, 2007; Hanslmayr et al., 2007). To summarize the above, what is being attempted is to bring together a more integrated model of limbic/cortical system function by studying the thalamocortical system, generator of alpha/delta rhythm with the septohippocampal system which generates theta by integrating Structural models (Aggleton and Brown; 1999) with P300 research (Melloni et al.,2007). This integration will be used to make a proposal that simplifies extremely the cortical (dipole)/Limbic (linear multipole) brain structure.

Summary of proposals for section 4.3

The limbic system is proposed to operate as a harmonic oscillator entwined in a radiating cortex dipole with the two systems interacting. The limbic systems linear multipole fires from neural regions bordering the lateral ventricles (its poles) to those neural regions bordering the septum and third ventricle at the brain midline. (Section 3.1) It is the collapsing midline regions of these linear multipole structures (the multipole lens effect) which produce the alternating periodic oscillations (delta, theta, alpha) that mark out limbic processes.

Cortex (radiating dipole) and limbic system (linear multipole) have electromagnetic structures which tend towards expansion and collapse respectively when considering their overall electromagnetic properties. As the review and integration points out the cortex/limbic system processes are intertwined. The thalamocortical and septohippocampal systems sync together for about 300ms, making a complete working brain system, highlighted by the P300. A quick reacting and expansive cortex is continually pulled back from the brink of chaos (ERD) by a thalamus which compresses or collapses the result of cortical propagation into spectrally reduced chains of information, and a hippocampus which indexes this sensory reduced data for long term memory comparison and recall.

4.4 Dipole/Quadrupole integration point explanation for hippocampal appearance and epilepsy.

The limbic system linear quadrupole extends from thalamus (third ventricle) along the quadrupole axis to terminate at the hippocampus (lateral ventricles). The hippocampus which is situated along the lateral ventricles choroids plexus. If a limbic quadrupole with poles at the hippocampus, sits inside a cortical dipole which has its poles at the nearby temporal lobes, then one of these multipole structures requires the reverse polarity of the other, else the two systems would push each other apart due to similar charges. This reversal is consistent with contralateral (reversal of sensory functions) structure which is found in the cortex. This dipole/quadrupole approach also proposes that the hippocampus twists into the cortex as a result of opposing charges between the entorhinal cortex which has a neurochemical distribution that correlates with an overall negative or hyper-polarizing tendency (see section 1) and the hippocampus which has an overall positive or depolarizing tendency. The Limbic quadrupole is compromised of two dipoles, one at each hippocampus connected to the brain midline, (two dipoles along one axis = linear quadrupole) interacting with each pole of the cortical dipole.

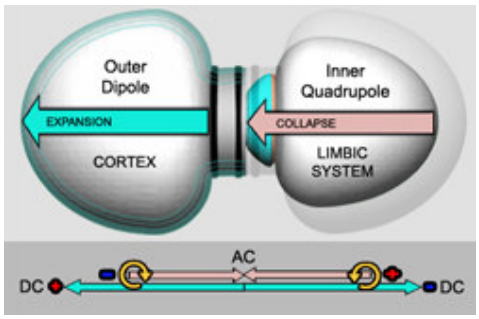

Figure 12: Putting the dipole and quadrupole together integrates two extremely different electromagnetic systems (author diagram) with a collapse/expansion trajectories. The quadrupoles files firing inwards collapse at the midline, while the dipole firing outwards to poles is in a state of expansion limited by the skulls surface. Bottom of figure. The Quadrupole and dipole integrate their poles at the hippocampus. The Quadrupole (AC in diagram) is compromised of two dipoles pointing to the brain midline. One of these dipoles poles interacts with the cortex dipoles poles producing an interaction of two separate dipole systems. This provides a theoretical explanation for the hippocampus wound S shape and its highly electrically active structure.

Each systems charges (the dipole and quadrupoles) are structurally moving in opposing directions from each other, but exist on the same axis. These opposite charges attract, while driving in opposite trajectories. The result is a spiral of intensely wound and dense neural activity of mixed charge. Hippocampus is then where limbic quadrupole meets cortical dipole. By analogy to other examples of two interacting dipole systems the S shaped twisting structure of the hippocampus is similar to the S shaped structure of binary stars, which are also two interacting dipoles in separate systems with their own trajectory. Simulations of two interacting dipoles moving on differing trajectories also produce the same S shape formations. (Schnetter, 2008) Binary star's can produce the most energetic oscillation's known as a result of this dipole/dipole interaction. Again by similar analogy, the hippocampus is marked with the brain's highest energies, marked out by the temporal lobe tendency towards epilepsy where spikes can rise to 600hz and in normal function produces high frequency ripples of between 100-300hz correlated to electrotonic activity. (Traub et al., 1999)

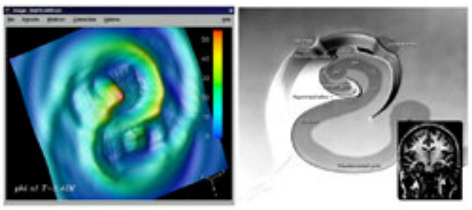

figure 13: Left, A binary star simulation (Schnetter, 2008) is two dipoles running along their own trajectories but still interacting. This produces S winding formations similar to the hippocampus (right) which is also proposed to be the interaction of two separate dipole systems.

4.5 Interaction of Dipole / Quadrupole as the engine of consciousness.

As sections 4.2, 4.3 attempts to show, key conscious processes are marked by recurring integration points between limbic and cortical system (hippocampus, thalamocortical) which is line with leading models of consciousness that highlight recurrent feedback between brain regions as the key marker of conscious processes (Lamme, 2006). Do the almost reverse differences between a quadrupole and dipole push and pull at each other in a manner which produce the highest amounts of recurrent feedback and so mark out the overarching engine of consciousness ? Anaesthetics operate along the limbic quadrupole midline - the thalamic reticular area and effect the connections to the cortex limbic-dorsolateral prefrontal cortex which spread from the midline (Mashour, 2006). These anaesthetics block gamma synchrony at gap junctions (Hameroff, 2007) which occurs in more highly connected cortical areas across the dipole midline from frontal to back (Imas et al., 2005). Areas found to synchronize sufficiently well to avoid connection lags (Roelfsema et al., 1997). In other words cortical binding and correlates of consciousness are highest where limbic quadrupole meets cortical dipole at the brain midline.

A quadrupole is centrolineal, it's products such as thalamus and hippocampus drive linear processes towards the centre where they synchronise into alternating current signals and phase locked harmonic combinations of their respective Theta, Delta and Alpha waves. The Quadrupole's phase locking enforces coherence and co-ordinates brain activity with repeated synchronization signals. The quadrapole fires inwardly to produce highly ordered dense structures, a result of constant inward pull. By contrast the cortex proposed as a conventional electric radiating dipole moves outwardly towards a fluid chaotic system in a state of expansion which decoheres (Rizi et al., 2000) after the P300. The cortical processes are fast acting temporary and outwardly radiating. The dipole/quadrupole are proposed to work together and produce consciousness, as an electromagnetic engine, with the inverse relationship between the radiating dipole and linear multipole pushing and pulling each other in integrated recurrent cycles.

Key Points from section 4

- The P300 which is the most functional and important of all the ERP's represents a critical integration point between cortical decoherence and limbic systems coherence. Thalamocortical / septohippocampal which could be said to integrate the dipole (cortex) Quadrupole (limbic system), operate together marked by generation of the P300.
- The P300 marks a point where cortically processed information can be encoded before it becomes decoherent. Lower ERP's below P300 represent more cortical process leading to decoherence while the later acting ERP's above P300 represent more limbic controlled processes which tend towards coherence.
- A quick reacting and expansive cortex is continually pulled back from the brink of chaos (ERD) by a thalamus which compresses or collapses the result of cortical propagation into spectrally reduced chains of information, and a hippocampus which indexes this sensory reduced data for long term memory comparison and recall.

- Hippocampus is formed due to the integration of the linear quadrupole and cortical dipole
- key conscious processes are marked by recurring integration points between limbic and cortical system (hippocampus, thalamocortical) which is line with leading models of consciousness that highlight recurrent feedback between brain regions as the key marker of conscious processes.
- The dipole/quadrupole are proposed to work together and produce consciousness, as an electromagnetic engine, with the inverse relationship between the radiating dipole and linear multipole pushing and pulling each other in integrated recurrent cycles.

DISCUSSION

The limbic quadrupole and cortical dipole differ markedly as systems, and appear to have properties which correlate with the difference between quantum and classical system properties respectively. (Table 10) Quantum approaches to conscious processing are not an area which is required to be approached by a brain EMS model. Trying to understand the emergent systems properties is the aim of this section. A systems basis for a quantum/classical proposal as a "style" of neural processing was an unexpected result (table 10) (Hameroff, S., 2007b). The dipole hemispheres are disconnected from each other, in comparison with the limbic system. Most cortical connections are within rather than cross hemisphere. This disconnection between hemispheres is more marked at the proposed temporal poles (see section 1). Chaotic and logical styles become separated processes isolated within the right and left hemispheres poles respectively. The cortical system tending towards decoherence. While in the limbic quadrupole system the deep interconnectedness correlates with its higher ratio of white to grey matter. The merging of opposite poles travelling towards the centre where they interact together possibly producing standing wave oscillations from alternate phase resembles the quantum properties of opposite paradox, merging into sameness. Constantly the limbic system moves towards coherence, while the cortex to decoherence.

This division of classical/quantum style systems is reflected in the neurons of each with the cortex having mainly ionic neurons which possess anisotropic vector properties that lend themselves well to conventional physical analysis. By contrast, the limbic system has messenger types where processes are environmentally isolated, a prerequisite for macroscopic quantum states. What is being suggested here is that rather than the brain operating at the interconnected quantum level, the paradoxical processes of quantum physics are themselves encoded in the limbic systems larger scale harmonic structure as an emergent property which serves complex systems themselves. This complex systems view is also in line with contemporary evolutionary proposals for electromagnetics and brain structure, which suggests that there are two concurrent neural systems; (a) Electromagnetic field sensitive (serial systems), and (b) electromagnetic field insensitive (parallel systems) (Tuszynski, 2006; Baars, 1993). The cortex is proposed as mainly field sensitive (so can fast react) is environmentally involved and tends towards serial processing, while the limbic system is mainly field insensitive (isolated) and tend towards parallel processing.

Table 10: Definitions for classical quantum systems relate to dipole/quadrupole (Hameroff, S., 2007b)

Cortex / Classical	**Limbic / Quantum**
Tends to decoherence	**Tends to coherence**
Concrete, chaotic	Sub Conscious, dream like
Definite, rigid	Multiple co-existing possibilities
Isolated, Disconnected	Deep interconnectedness
Logical separated	Paradox, opposites merge into sameness

The model's desribed here may help elucidate why many researchers look for quantum correlates to consciousness. These definitions for classical and quantum properties directly relate to the radiating dipole and linear quadrupole respectively.

The whole brain models presented here may appear to contradict standard evolutionary models that highlight how each part evolved bit by bit to fulfil adaptation. This view is not being proposed as incorrect, but at the same time a very clear electromagnetic structure is evident in the cortex and limbic system. This is proposed as the result of a natural evolution that occurs as biological electromagnetic systems gain greater efficiency. The reasoning being that as complex systems take on dense increases in electromagnetic processing capacity they develop self similar hierarchies of scale which display the form of the underlying physics involved in their production. In the brain these forms are more likely to represent the natural optimum structures for multi-level complex electromagnetic processing to emerge in an overarching manner. This is in line with current views on emergence in complex systems. For example complex multi level systems tend to encompass a balance of chaos and order (Kondor et al., 2008). The model proposed in this paper fulfils this requirement by proposing that the cortex is a radiating dipole and tends towards expansion - which gives rise to fast reacting (low latency) and chaotic processes. The limbic system is proposed as a linear quadrupole which tends towards collapse - producing synchronization, encoding and harmonic superposition (parallel processing) but is environmentally isolated with high latency. The mammalian brain functions due to the interaction of these two structures which give a mixture of sensitivity (limbic system) and robustness (cortex) (Kondor et al., 2008).

Objections and predictions.

Objections raised so far on the Cortical EMS model highlight that this model too is incomplete. It provides observable similarities to magnetic dipoles, extended information on lateralization and provides no actual mechanism for how a dipole structure can arise (Personal Correspondence W.Bains, 2009). It is not currently possible to complete this model to that degree, although the model is highly consistent and predictive in regards to the neurodevelopmental stages when brain structure forms. (Section 2.6). Further objections pointed to the fact as far as we know charges are individually isolated within neuronal membranes without net action (Personal Correspondence W.Bains, 2009). It is not currently proposed as part of Cortical EMS that there is a net charge based on ions. The net charge which would separate charges across hemispheres is proposed purely to arise in development. (Section 2.6) What we are left with is a fairly crystallized electromagnetic structure.

Having said that researchers in consciousness are still investigating whether there is more electromagnetic coherence between cortical regions than currently known (Hameroff, 2007,2009).

What is proposed is that the aggregation of these charges operating through neurons produces a net action that represents the systems property of these charges, and that this can be sustained in a polarized field. For example at the Left temporal regions, many negative chlorine ions (convergent (Lowrie, 1997)) firing by GABA receptors results in overall hyperpolarization or net inhibitory IPSP action which can also be defined as convergent. This net process is achieved by the magnetic fields in the NAAMF (Neuronal Activity Associated Magnetic Fields) astrocyte matrix proposed by M. Banachlocha (Banachlocha, 2001,2007). As neuronal density is highest at the temporal regions (Thompson et al., 2003) and most lateralized in terms of the required component for a separation of charges, (Section *1.5.4*) then such net action occurs not only at the regions predicted by the model but also at the furthermost cortex surface layer

It should be borne in mind that the Cortical EMS model at its inception was no more than an idea based on understanding of the psychology of lateralization and some brain images. At that time it made a list of predictions which were handed to the co-author as the prime reason for investigation. The list of predictions and subsequent evidence gathered so far has actually been quite surprising. Mainly was the prediction that *"If the entire brain has dipole or electromagnetic structure, then everything in it must also fall into such a structure." (Available online at WWW.Dipoleneurology.org.uk (predictions)*) The research since the inception has been borne out to a high degree of accuracy. Entire grey matter content does appear to fall in line with the distributions required for a separation of opposite charges, across the temporal regions. In other words Cortical EMS predicted that the furthermost temporal regions were where the separation of charges would be found. The subsequent, investigation at multiple levels bear this out : Neuronal ions, (Section 1.5) Neuronal assembly structure, (Section 1.2) Neuronal Densities, (Section *1.5.4*) Maximum asymmetry and a mechanism for polarization (NAAMF).

At the initial phase of investigation, the limbic system was also somewhat of a mystery, yet it too turned out that a multipole model (Limbic EMS) could be proposed, albeit with no current evidence for lateralization of its content. What is interesting is that both Cortical EMS and Limbic EMS turn out to be reversals of each other which are complementary in electromagnetic terms. (section 4) The Cortical EMS has its structure and function located at the furthermost regions (cortex surface) while the Limbic EMS has its structure and function located at the innermost regions (thalamus). The interaction of the two models (CorticoLimbic EMS) functionally complement each other (Section 4) while also integrating along the same axis and then subsequently predict the structure of the hippocampus (Section 4.4). There is currently no known model which attempts to address the electromagnetic observable features which mark out the brain, and has been successful in predicting a long list of functional properties. At this stage a reasonably confidently prediction would be that the burgeoning discoveries in neurodevelopment are going to prove to what extent the EMS models are valid. In this sense the EMS model still has high predictive properties. We propose that the previously mentioned predictions, subsequent discoveries and future predictions should hopefully providing a starting foundation and fuel some interest in this long overlooked area.

Limitations

The aim of this project has been to integrate seemingly fragmented data into a complete electromagnetic picture of mammalian brain function as possible. Such complex integration requires a picture building process that puts restraints on the deductive logical process which are successful in breaking biosystems into fragments (Capra, F., 1996). For example it is hard to find a single experiment which can falsify evolution. The analysis of hard evidence really only exists in sufficient quantity to start pushing Cortical EMS as a theory. The other branches such as Limbic EMS and CorticoLimbic EMS have to remain hypothetical. In this respect the evidence given is not a logical conclusion. Showing temporal polar lateralization at nearly every cortical level; ions, receptors, neuronal assemblies and processing does not logically provide a dipole model. However it does provide a complete structural picture that is consistent with the theory. In regards to the more hypothetical sections, 2,3 and 4 it is conceded that there could be other interpretation for the emerging evidence. For example there may be other reasons for cortical stripe domains than ferroelectric fluids or different multipole structures than those proposed here for the limbic system. In spite of these limitations sections 2,3 and 4 are retained as they facilitate one central theory of electromagnetic structures to simplify the majority of brain structure and function in terms of physics. In this respect it should also be borne in mind that a final question of limitation arising from the conclusions in this paper. This would be that if the electromagnetic analysis given here is correct, is such a reduction moving beyond the irreducibility of brain complexity ? Conversely this could be the start of a new approach to understanding the basic structure of conscious systems?

Negative Data

Negative Data occurred several times during the research, due to the initial hypothesis of the brain dipole model being a magnetically dominated dipole system, rather than a bioelectric dipole with magnetic interactions at the brain surface. It was also hypothesised that the neurotransmitters Glutamate and GABA themselves would have ionic charges which would fall in line with being positive and negative. Of course these neurotransmitters are the keys for an ionic system, so do not require such charge. It was also proposed on the basis of previous quantum consciousness theories (Hameroff, 2007) that superpositional processing could arise in the cortex, but it was not possible to find evidence. This then proved contrary to the emergent properties of the noise biased cortical model that arose later. However the quantum “style” of paradox, and harmonic interconnectedness appears to be an emergent property of the alternating currents proposed at the midline of the limbic system.

Falsifiability

The CorticoLimbic EMS solution to brain structure is a difficult theory to falsify, due to it being a descriptive simplification of the most complex system known. As it proposes many branches to the central dipole theory such as cortex surface magnetic structure, limbic system quadrupole. In that sense it suffers the same burden of proof problem with other simplified complex system theories. The most dramatic of these being evolution. Such theories try to formulate a single framework for the structure of trillions of interacting and varied components. A multifaceted theory needs a similarly multifaceted falsification process. It has been attempted to start things off by providing observable description’s and tabulated evidence that highlights that the distribution of the cortical content is lateralized in line with the model.

Further studies could concentrate along the lines of computer based electromagnetic simulations of the model. It would be predicted that an S type hippocampus formation would occur at the interaction point between the poles of the dipole and quadrupole system, and that cortical magnetic domains would appear, when a rough electric/magnetic dipole is simulated. EEG studies of the temporal lobes should find a marked difference between left/ hyperpolarized, and right/ depolarized signals, greater than the difference between any other cortical areas. This would concur with the concept that the highest dipole moment exists between left and right temporal lobes. As schizophrenia has been used as a means to decode temporal lobe laterality, existing EEG studies due to altered laterality (Edgar et a., 2008; Flynn et al., 2008) could be expanded upon to discern if normal EEG is asymmetrical in line with a dipole distribution of charge, and alterations from this distribution deviate in line with structural abnormalities. It would also be predicted that dissections of temporal lobes would also find higher densities of NMDA receptors at the RCH than the LCH.

Conclusion

This paper concludes that the human brain possesses too clear an electromagnetic structure throughout the cortex and limbic system to be ignored. We believe that using this basic structure will provide neuroscience with large scale integration and high explanative potential for many mysteries about brain structures and functions. Many positive implications in regards to dysfunction could be derived. For example neurological illness is quite unique in comparison to other disease. Many major brain disorders affect the laterality and structure of the entire brain: Williams's syndrome, autism, schizophrenia and Alzheimer's. Alzheimer's in particular is marked out by higher than normal amounts of magnetic compounds. If these compounds play a role in the brains electromagnetic structure and function then the magnetic ageing which occurs naturally to such compounds could contribute to a dysfunction of the brains electromagnetic system. If the brain is looked at from the view of a complete electromagnetic system where the entire brain has an electromagnetic structure that is determining the greater part of it systems hierarchy – then the subcomponents and smaller scale brain content can be placed within this hierarchy allowing the abnormality and functions of the brain to be reframed in a larger systems view. In this paper schizophrenia research was also used to highlight evidence which not only reveals dipole structure, but conversely the dipole structure is also able to make predictions about schizophrenia as primarily a breakdown of cortical dipole structure. The area where this theory should contribute its greatest insight is neurodevelopment. The connexins which increase electromagnetic coherence in neurodevelopment (see section 2.6) are proposed as the key to understand how the brains electromagnetic structure can build itself. The role of this theory then could have multiple applications. Mapping out complex brain wide neurological disorders in terms of their role in the brains electromagnetic structure. A guide to why neurodevelopmental stages occur in a particular order. Finally, if CorticoLimbic EMS is the brains physical structure, it is likely to play a role in any attempt to replicate its functions in machine consciousness.

Acknowledgements

Dr Ashi Sadiq and Prof. William bains for trial reviewing. Heather Dalgleish for grammar and editing. Neil Mackay for physics lessons. Dr Pamela Lanza for guidance on the publication process. Dr Marcos Banaclocha for personal correspondence regarding section 2.

REFERENCES (Online and figure references at end)

Abi-Saab, W.M., D'Souza, D.C., Moghaddam, B., Krystal, J.H., 1998. The NMDA antagonist model for schizophrenia: promise and pitfalls. Pharmacopsychiatry 31, 104-9.

Adamec, R.E., 1999. Evidence that limbic neural plasticity in the right hemisphere mediates partial kindling induced lasting increases in anxiety-like behaviour. Brain Res 839, 133-52.

Aggleton, J.P., Brown, M.W., 1999. Episodic memory, amnesia, and the hippocampal-anterior thalamic axis. Behav Brain Sci 22, 425-44.

Akbarian, S., Huang, H.S., 2006. Molecular and cellular mechanisms of altered GAD1/GAD67 expression in schizophrenia and related disorders. Brain Res Rev 52, 293-304.

Andrade, R., 1998. Regulation of membrane excitability in the central nervous system by serotonin receptor subtypes. Ann N Y Acad Sci 861, 190-203.

Anokhin, A.P., Lutzenberger, W., Birbaumer, N., 1999. Spatiotemporal organization of brain dynamics and intelligence: an EEG study in adolescents. Int J Psychophysiol 33, 259-73.

Apartis, E., Poindessous-Jazat, F.R., Lamour, Y.A., Bassant, M.H., 1998. Loss of rhythmically bursting neurons in rat medial septum following selective lesion of septohippocampal cholinergic system. J Neurophysiol 79, 1633-42.

Avidan, G., Behrmann, M., 2002. Correlations between the fMRI BOLD signal and visual perception. Neuron 34, 495-7.

Ayton, G., Gingras, M.J., Patey, G.N., 1995. Orientational ordering in spatially disordered dipolar systems. Phys Rev Lett 75, 2360-2363.

Baars, B.J., 1993. Experimental and Theoretical Studies of Consciousness. Ciba Found Symp 174, 1-316.

Babcock, T., Helton, W.S., Espat, N.J., 2000. Eicosapentaenoic acid (EPA): an antiinflammatory omega-3 fat with potential clinical applications. Nutrition 16, 1116-8.

Balcells, L., Tholence, J.L., Linderoth, S., Barbara, B., Tejada, J., 1992. Quantum tunneling of magnetization in metallic FeC ferrofluids. Zeitschrift für Physik B Condensed Matter 89, 209-212.

Banaclocha, M.A., 2002. Are neuronal activity-associated magnetic fields the physical base for memory? Med Hypotheses 59, 555-9.

Banaclocha, M.A., 2007. Neuromagnetic dialogue between neuronal minicolumns and astroglial network: a new approach for memory and cerebral computation. Brain Res Bull 73, 21-7.

Bartos, M., et al., 2002. Fast synaptic inhibition promotes synchronized gamma oscillations in hippocampal interneuron networks. Proc Natl Acad Sci U S A. 99,13222-7.

Bazan, N.G., 2004. Synaptic lipid signaling: significance of polyunsaturated fatty acids and platelet-activating factor. J Lipid Res 44, 2221-33.

Beckman ML, Bernstein EM, Quick WM. Protein Kinase C Regulates the Interaction between a GABA Transporter and Syntaxin 1A. J Neurosci 1998;18(16):6103-12.

Beene, D.L., Brandt, G.S., Zhong, W., Zacharias, N.M., Lester, H.A., Dougherty, D.A., 2002. Cation-pi interactions in ligand recognition by serotonergic (5-HT3A) and nicotinic acetylcholine receptors: the anomalous binding properties of nicotine. Biochemistry 41, 10262-9.

Bear, F.M., Connors, B.W., Paradiso, M.A., 2001. Neuroscience exploring the brain.

Bell, J.G., MacKinlay, E.E., Dick, J.R., MacDonald, D.J., Boyle, R.M., Glen, A.C., 2004. Essential fatty acids and phospholipase A2 in autistic spectrum disorders. Prostaglandins Leukot Essent Fatty Acids 71, 201-4.

Berger, G.E., Smesny, S., Amminger, G.P., 2006. Bioactive lipids in schizophrenia. Int Rev Psychiatry. 18, 85-98.

Birren, J.E., Schaie, K.W., (Eds.) 2006. Handbook of the psychology of aging. 6th ed., Academic Press. Satlhouse, T.A., (Part 1)

Bolotin, Y.L., Gonchar, V.Y., Granovsky, M.Y., 1995. The regularity-chaos-regularity transition in a periodically driven anharmonic oscillator. Physica D. Nonlinear Phenomena 86, 500-507.

Bonhoeffer, T., Grinvald, A., 1991. Iso-orientation domains in cat visual cortex are arranged in pinwheel-like patterns. Nature 353, 429-31.

Brandt, M.E., 1997. Visual and auditory evoked phase resetting of the alpha EEG. Int J Psychophysiol 26, 285-98.

Brazier, M., 1980. The Electrical Activity of the Nervous System .

Brem, F., Tiefenauer, L., Fink, A., Dobson, J., Hirt, A.M., 2006. A mixture of ferritin and magnetite nanoparticles mimics the magnetic properties of human brain tissue. Physical review B 73, 224-427.

Brovelli, A., Lachaux, J.P., Kahane, P., Boussaoud, D., 2005. High gamma frequency oscillatory activity dissociates attention from intention in the human premotor cortex. Neuroimage 28, 154-64.

Bruder, G.E., Stewart, J.W., Tenke, C.E. et al., 2001. Electroencephalographic and perceptual asymmetry differences between responders and nonresponders to an SSRI antidepressant. Biol Psychiatry 49, 416-25.

Callachan, H., Cottrell, G.A., Hather, N.Y., Lambert, J,J., Nooney, J.M., Peters, J.A., 1987. Modulation of the GABAA receptor by progesterone metabolites. Proc R Soc Lond B Biol Sci 231,359-69.

Capra, F., 1996. The Web of Life: A New Synthesis of Mind and Matter Hammersmith, London: HarperCollins.

Carter, R., 1998. Mapping the mind.

Cernácek, J., 1989. Biochemical and electrophysiological correlations of functional asymmetry of the brain. Bratisl Lek Listy 90, 458-61.

Chomsky N., 1969. Aspects of the Theory of Syntax. Mit Press.

Colin, A., Reggers, J., Castronovo, V., Ansseau, M., 2003. Lipids, depression and suicide. Encephale 29, 49-58.

Collingridge, G.L., Olsen, R.W,, Peters, J., Spedding, M., 2009. "A nomenclature for ligand-gated ion channels". Neuropharmacology 56, 2–5.

Crick, F., Koch, C., 2003. A framework for consciousness. Nat Neurosci 6, 119-26.

Cytowic, R.E., 1995. Synesthesia: Phenomenology And Neuropsychology. Psyche 2.

Demeter, E., Tekes, K., Majorossy, K., Arato, M., Somogyi, E., 1988. Does 3H-imipramine binding asymmetry indicate psychiatric illness? Acta Psychiatr Scand 77, 746

Dobson, J., 2001. On the structural form of iron in ferritin cores associated with progressive supranuclear palsy and Alzheimer's disease. Cell Mol Biol (Noisy-le-grand) 47.

Douady, S., Couder, Y., 1992. Phyllotaxis as a physical self-organized growth process. Phys Rev Lett 68, 2098-2101.

Dringen, R., Bishop, G.M., Koeppe, M., Dang, T.N., Robinson, S.R., 2007. The pivotal role of astrocytes in the metabolism of iron in the brain. Neurochem Res 32, 1884-90.

Edgar, J.C., Hanlon, F.M., Huang, M.X., 2008. Superior temporal gyrus spectral abnormalities in schizophrenia. Psychophysiology 45, 812-24.

Edwards, E., Soltani, M., Deouell, L.Y., Berger, M.S., Knight, R.T., 2005. High gamma activity in response to deviant auditory stimuli recorded directly from human cortex. J Neurophysiol 94, 4269-80.

Elias, L.A., Wang, D.D., Kriegstein, A.R., 2007. Gap junction adhesion is necessary for radial migration in the neocortex. Nature. 448,901-7.

Fell, J., Dietl, T., Grunwald, T., et al 2004. Neural bases of cognitive ERPs: more than phase reset. J Cogn Neurosci 16, 1595-604.

Ferronsky, V. I., Denisik, S. A., Ferronsky, S.V., 2005. Virial oscillations of celestial bodies III. The solution of the evolutionary problem in non-newtonian time scale. Celestial Mechanics and Dynamical Astronomy 32, 173-183.

Fink, G.R., Marshall, J.C., Halligan, P.W., Frith, C.D., Frackowiak, R.S., Dolan, R.J., 1997. Hemispheric specialization for global and local processing: the effect of stimulus category. Proc Biol Sci 264, 487-94.

Finke, R.A., Bettle, J.. 1996. Chaotic Cognition: Priniciples and Applications. Lawrence Erlbaum Associates

Fox, S.I., 1987. Human Physiology.

Flor-Henry, P., 1986. Observations, reflections and speculations on the cerebral determinants of mood and on the bilaterally asymmetrical distributions of the major neurotransmitter systems. Acta Neurol Scand Suppl 109, 75-89.

Flynn, G., Alexander, D., Harris, A., 2008. Increased absolute magnitude of gamma synchrony in first-episode psychosis. Schizophr Res 105, 262-71.

Gallivan, J.P., Dougherty, D.A., 1999. Cation-pi interactions in structural biology. Proc Natl Acad Sci U S A. 96, 9459-64.

Fuller, B.R., 1979. SYNERGETICS—Explorations in the Geometry of Thinking, Volumes I & II,
New York, Macmillan Publishing Co.

Gilbert, D., McClernon, J., Rabinovich, N. et al., 2004. Effects of quitting smoking on EEG activation and attention last for more than 31 days and are more severe with stress, dependence, DRD2 A1 allele, and depressive traits. Nicotine Tob Res 6, 249-67.

Gilbert, D.G., Robinson, J.H., Chamberlin, C.L., Spielberger, C.D., 1989. Effects of smoking/nicotine on anxiety, heart rate, and lateralization of EEG during a stressful movie. Psychophysiology 26, 311-20.

Ginobili de Martinez, M.S., Rodriguez de Turco, E.B,, Barrantes, F.J., 1985. Endogenous asymmetry of rat brain lipids and dominance of the right cerebral hemisphere in free fatty acid response to electroconvulsive shock. Brain Res 339, 315-21.

Ginobili de Martinez, M.S., Rodriguez de Turco, E.B., Barrantes, F.J., 1986. Asymmetry of diacylglycerol metabolism in rat cerebral hemispheres. J Neurochem 46, 1382-6.

Ginobili de Martinez, M.S., Barrantes, F.J., 1988. Ca2+ and phospholipid-dependent protein kinase activity in rat cerebral hemispheres. Brain Res 440. 386-90.

Glick, S.D., Ross, D.A., Hough, L.B., 1982. Lateral asymmetry of neurotransmitters in human brain. Brain Res 234, 53-63.

Gong, J.S., Morita, S.Y., Kobayashi, M., et al, 2007. Novel action of apolipoprotein E (ApoE): ApoE isoform specifically inhibits lipid-particle-mediated cholesterol release from neurons. Mol Neurodegener 15, 9.

Guarneri, P., Guarneri, R., La Bella, V., Scondotto, S., Scoppa, F., Piccoli, F., 1998. Lateral differences in GABA binding sites in rat brain. Neurochem Res 13, 209-11.

Guyton, Hall., 2000. Guytan's Textbook of Physiology.

Green, J.T., Orr, S.K., Bazinet, R.P., 2008. The emerging role of group VI calcium-independent phospholipase A2 in releasing docosahexaenoic acid from brain phospholipids. J Lipid Res 49, 39-44.

Grinvald, A., Slovin, H., Vanzetta, I., 2000. Non-invasive visualization of cortical columns by fMRI. Nat Neurosci 3, 105-7.

Gruber, W.R., Klimesch, W., Sauseng, P., Doppelmayr, M., 2005. Alpha Phase Synchronization Predicts P1 and N1 Latency and Amplitude Size. Cereb Cortex 15, 371-7.

Haag, M., 2003. Essential fatty acids and the brain. Can J Psychiatry 48, 195-203.

Haier, R.J., Jung, R.E., Yeo, R.A., Head, K., Alkire, M.T., 2005. The neuroanatomy of general intelligence: sex matters. Neuroimage 25, 320-7

Hameroff, S.R., 2007. The Brain Is Both Neurocomputer and Quantum Computer. Cognitive Science: A Multidisciplinary Journal 31, 1035-1045.

Han, L., Wong, D.L., Tsai, G., Jiang, Z., Coyle, J.T., 2007. Promoter analysis of human glutamate carboxypeptidase II. Brain Res 1170, 1-12.

Hanslmayr, S., Klimesch, W., Sauseng, P., 2007. Alpha phase Reset Contributes to the Generation of ERPs. Cereb Cortex 17,1-8.

Hashimoto, T., Bazmi, H.H., Mirnics, K., Wu, Q., Sampson, A.R., Lewis, D.A., 2008. Conserved regional patterns of GABA-related transcript expression in the neocortex of subjects with schizophrenia. Am J Psychiatry 165, 479-89.

Hashimoto, T., Volk, D.W., Eggan, S.M et al. 2003. Gene expression deficits in a subclass of GABA neurons in the prefrontal cortex of subjects with schizophrenia. J Neurosci 23, 6315-26.

Hattori, T., Nishimura, Y., Sakai, N., Yamada, H., Kameyama, Y., Nozawa, Y., 1986. Attenuation by pentobarbital of free fatty acid and diacylglycerol liberation during global ischaemia in rat brain. Neurol Res 8, 33-8.

Hausmann, M., Güntürkün, O., 2000. Steroid fluctuations modify functional cerebral asymmetries: the hypothesis of progesterone-mediated interhemispheric decoupling. Neuropsychologia 38, 1362-74.

Heeger, D.J., Ress, D., 2002. What does fMRI tell us about neuronal activity? Nat Rev Neurosci 3, 142-51.

Hong, K.H., Bonventre, J.C., O'Leary, E., Bonventre, J.V., Lander, E.S., 2001. Deletion of cytosolic phospholipase A2 suppresses ApcMin-induced tumorigenesis. Proc Natl Acad Sci U S A. 98, 39s35–3939.

Horn, R. A., Johnson, C.R., 1985. Matrix Analysis. Cambridge University Press. ISBN 0-521-38632-2.

Horn, R. A., Johnson, C.R., 1991. Topics in Matrix Analysis. Cambridge University Press. ISBN 0-521-46713-6.

Howard, K.J., Rogers, L.J., Boura, A.L., 1980. Functional lateralization of the chicken forebrain revealed by use of intracranial glutamate. Brain Res 188, 369-82.

Hutsler, J., Galuske, R.A., 2003. Hemispheric asymmetries in cerebral cortical networks. Trends Neurosci 26, 429-35.

Imas, O.A., Ropella, K.M., Ward, B.D., Wood, J.D., Hudetz, A.G., 2005. Volatile anesthetics disrupt frontal-posterior recurrent information transfer at gamma frequencies in rat. Neurosci Lett 387, 145-50.

Jeon, Y.W., Polich, J., 2003. Meta-analysis of P300 and schizophrenia: patients, paradigms, and practical implications. Psychophysiology 40,684-701.

Johansson, C., Lansner, A., 2007. Towards Cortex Sized Artificial Nervous Systems. Neural Netw 20, 48-61.

Kalpouzos, G., Eustache, F., de la Sayette, V., Viader, F., Chetelat, G., Desgranges, B., 2005. Working memory and FDG-PET dissociate early and late onset Alzheimer disease patients. J Neurol 252, 548-58.

Kamasawa, N., et al., 2006. Connexin-47 and connexin-32 in gap junctions of oligodendrocyte somata, myelin sheaths, paranodal loops and Schmidt-Lanterman incisures: implications for ionic homeostasis and potassium siphoning. Neuroscience 136, 65-86

Kane, J. W., Sternheim, M.M., 1988. Physics 3rd Edition.

Karson, C.N., Coppola, R., Daniel, D.G., 1988. alpha frequency in schizophrenia: an association with enlarged cerebral ventricles. Am J Psychiatry 145, 861-4.

Kauffman, S., 1993. The Origins of Order: Self-organization and Selection in Evolution. New York: Oxford University Press. Kimura, D. (1979).

Kim, S.G., Duong, T.Q., Kim, D.S., Nagaoka, T., Harel, N., 2002. Spatial specificity of CBF and BOLD responses induced by neural activity. International Congress Series 1235, 39-47.

Kim, D.S., Duong, T.Q., Kim, S.G., 2000. High-resolution mapping of iso-orientation columns by fMRI. Nat Neurosci 3, 164-9.

Kleinschmidt, A., Büchel, C., Hutton, C., Friston, K.J., Frackowiak, R.S., The neural structures expressing perceptual hysteresis in visual letter recognition. Neuron 34, 659-66.

Klimesch, W., 1996. Memory processes, brain oscillations and EEG synchronization. Int J Psychophysiol 24, 61-100.

Klimesch, W., Schack, B., Schabus, M., et al, 2004. Phase-locked alpha and theta oscillations generate the P1–N1 complex and are related to memory performance. Brain Res Cogn Brain Res 19, 302-16.

Kucharska-Pietura, K., Phillips, M.L., Gernand, W., David, A.S., 2003. Perception of emotions from faces and voices following unilateral brain damage. Neuropsychologia 41, 1082-90.

Kumar, S.S., Huguenard, J.R., 2001. Properties of Excitatory Synaptic Connections Mediated by the Corpus Callosum in the Developing Rat Neocortex. J Neurophysiol 86, 2973-85.

Kurup, R.K., Kurup, P.A., 2003a. The concept of cerebral chemical dominance. J Neurosci 113, 957-70.

Kurup, R.K., Kurup, P.A., 2003b. Hypothalamic digoxin, hemispheric chemical dominance, and creativity. Int J Neurosci 113, 565-77.

Kurup, R.K., Kurup, P.A., 2002. Hypothalamic digoxin, cerebral dominance, and membrane biochemistry. Int J Neurosci 112, 1439-47.

Kurup, R.K., Kurup, P.A., 2003c. Hypothalamic digoxin, cerebral chemical dominance, and calcium/magnesium metabolism. Int J Neurosci 113, 999-1004.

Kirichenko, E.V., Sobolewska, A., Stephanovich, V.A., 2003. The Domain Wall Structure in Disordered Ferroelectrics. Ferroelectrics 288, 211-219.

Kobayashi, A,K., Kirschvink, J.L., Nesson, M,H.m 1995. Ferromagnetism and EMFs. Nature 374, 123.

Kocsis, B., Li, S., 2004. In vivo contribution of h-channels in the septal pacemaker to theta rhythm generation. Eur J Neurosci 20, 2149-58.

Lahaye, T., Koch, T., Fröhlich, B., et al, 2007. Strong dipolar effects in a quantum ferrofluid. Nature 448, 672-5.

Lamme, V.A., 2006. Towards a true neural stance on consciousness. Trends Cogn Sci 10, 494-501.

Law, M.H., Cotton, R.G,, Berger, G.E., 2006. The role of phospholipases A2 in schizophrenia. Mol Psychiatry 11, 547-56.

Lawrence, J.J., Grinspan, Z.M., Statland, J.M., McBain, C.J., 2006. Muscarinic receptor activation tunes mouse stratum oriens interneurones to amplify spike reliability. J Physiol 571, 555-62.

Lester, H.A., Cao, Y., Mager, S., 1996. Listening to neurotransmitter transporters. Neuron 17,807-10.

Levy, J., Sperry, R.W., 1971. Lateral specialization and cerebral dominance in commissurotomy. Proc. 19th Int. Cong. Psychol., 244.

Levy, J., 2002. Oxide-Semiconductor Materials for Quantum Computation. physica status solidi (b) 233, 467-471.

Lewis, D.A., Glantz, L.A., Pierri, J.N., Sweet, R.A., 2003. Altered Cortical Glutamate Neurotransmission in Schizophrenia Evidence from Morphological Studies of Pyramidal Neurons. Ann N Y Acad Sci. 1003, 102-12.

Lindboe, C.F., 2003. Brain weight: what does it mean? Clin Neuropathol 22, 263-5.

Lowrie, W., 1997. Fundamentals of Geophysics. pp.204.

Macek, T.A., Schaffhauser, H., Conn, P.J., 1988. Protein kinase C and A3 adenosine receptor activation inhibit presynaptic metabotropic glutamate receptor (mGluR) function and uncouple mGluRs from GTP-binding proteins. J Neurosci 18, 6138-46.

Maguire, E.A., Frackowiak, R.S., Frith, C.D., 1997. Recalling routes around london: activation of the right hippocampus in taxi drivers. J Neurosci 17, 7103-10.

Malonek, D., Grinvald, A., 1996. Interactions between electrical activity and cortical microcirculation revealed by imaging spectroscopy: implications for functional brain mapping. Science 272, 551-4.

Manoucher., Messripour., 1992. The effects of pH on dopamine and serotonin synthesis by rat brain striatal synaptosomes. Indian journal of pharmacology 24, 32-35.

Mashour, G.A., 2006. Integrating the Science of Consciousness and Anesthesia. Anesth Analg 103, 975-82.

Matsuoka, F., Shinkai, M., Honda, H., Kubo, T., Sugita, T., Kobayashi, T., 2004. Hyperthermia using magnetite cationic liposomes for hamster osteosarcoma. Biomagn Res Technol 2, 3.

McFadden, J., 2002. The Conscious Electromagnetic Information (Cemi) Field Theory: The Hard Problem Made Easy? Journal of Consciousness Studies. 9,45-60.

McLaughlin, D., Shapley, R., Shelley, M., Wielaard, D.J., 2000. A neuronal network model of macaque primary visual cortex (V1): orientation selectivity and dynamics in the input layer 4Calpha. Proc Natl Acad Sci U S A 97, 8087-92.

Meinild, A.K., Sitte, H.H., Gether, U., 2004. Zinc potentiates an uncoupled anion conductance associated with the dopamine transporter. J Biol Chem. 279, 49671-9

Melloni, L., Molina, C., Pena, M., Torres, D., Singer, W., Rodriguez, E., 2007. Synchronization of neural activity across cortical areas correlates with conscious perception. J Neurosci 27, 2858-65.

Metherate, R., Cruikshank, S.J., 1999. Thalamocortical inputs trigger a propagating envelope of gamma-band activity in auditory cortex in vitro. Exp Brain Res 126, 160-74.

Miller, B., Sarantis, M., Traynelis, S.F., Attwell, D., 1992. Potentiation of NMDA receptor currents by arachidonic acid. Nature 355, 722-5.

Morand, N., Bouvard, S., Ryvlin, P., et al 2001. Asymmetrical localization of benzodiazepine receptors in the human auditory cortex. Acta Otolaryngol 121, 293-6.

Morris, D., 1978. Manwatching. Triad Granda. Page 284.(Laterality)

Moss, F., Ward, L.M., Sannita, W.G., 2004. Stochastic resonance and sensory information processing: a tutorial and review of application. Clin Neurophysiol 115, 267-81.

Nabekura, J., Noguchi, K., Witt, M,R., Nielsen, M., Akaike, N., 1998. Functional modulation of human recombinant gamma-aminobutyric acid type A receptor by docosahexaenoic acid. J Biol Chem 273, 11056-61.

Nadarajah, B., Jones, A.M., Evans, W.H., Parnavelas, J.G., 1997. Differential expression of connexins during neocortical development and neuronal circuit formation. J Neurosci. 17,3096-111.

Nunez, P., Srinivasan, R., 1981. Electric Fields of the Brain. First Edition 330-332, 232-298, 330.

Nyiri, G., Stephenson, F,A., Freund, T.F., Somogyi, P,. 2003. Large variability in synaptic N-methyl-D-aspartate receptor density on interneurons and a comparison with pyramidal-cell spines in the rat hippocampus. Neuroscience 119, 347-63.

Ogawa, T., 1983. Topological Disorder in Condensed Matter 60-77.

Oliva, A., Brady, T.F., 2008. Perceptual organization across spatial scales in natural images: Seeing more high spatial frequency than meet the eyes. Journal of Vision 8, 71a.

Parihar, H.S., Kirschbaum, K.S., 2002. Electrostatic potential surfaces of 5-HT(3)R agonists suggest accessory cation-pi site adjacent to agonist binding domain. Bioorg Med Chem Lett 12, 2743-7.

Palestini, P., Toppi, N., Ferraretto, A., Pitto, M., Masserini, M., 1997. Ganglioside lateralization in the brain of female rats. J Neurosci Res 1997;50(4):643-8.

Park, J.H., Chin, B.D., Park, O.O., 2001. Rheological Properties and Stabilization of Magnetorheological Fluids in a Water-in-Oil Emulsion. J Colloid Interface Sci 240, 349-354.

Pediconi, M.F., Barrantes, F.J., 1990. Brain asymmetry in phospholipid polar head group metabolism: parallel in vivo and in vitro studies. Neurochem Res 15, 25-32.

Pediconi, M.F., Rodriguez de Turco, E.B., 1984. Free fatty acid content and release kinetics as manifestations of cerebral lateralization in mouse brain. J Neurochem 43, 1-7.

Pediconi, M.F., Barrantes, F.J., 1993. Phospholipid metabolism under muscarinic cholinergic stimulation exhibits brain asymmetry. Neurochem Res 18, 559-64.

Pelaez, J.R., 2003. Schizophrenia Positive Symptoms Interpreted as Cognitive Hallucinations. Lecture Notes in Computer Science 2774, 1156-1163.

Pellerin, L., Wolfe, L.S., 1991. Donner. Release of arachidonic acid by NMDA-receptor activation in the rat hippocampus. Neurochem Res 16, 983-9.

Peters, A., Payne, BR., Josephson, K., 1990. Transcallosal non-pyramidal cell projections from visual cortex in the cat. J Comp Neurol 302, 124-42.

Piccolino, M., Neyton, J., Gerschenfeld, H.M., 1984. Decrease of gap junction permeability induced by dopamine and cyclic adenosine 3':5'-monophosphate in horizontal cells of turtle retina. J Neurosci 4,2477-88.

Polich, J., 2007. Updating P300: an integrative theory of P3a and P3b. Clin Neurophysiol 118, 2128-48.

Prechtl, J.C., Cohen, L.B., Pesaran, B., Mitra, P.P., Kleinfeld, D., 1997. Visual stimuli induce waves of electrical activity in turtle cortex. Proc Natl Acad Sci U S A 94, 7621-6.

Prasad, M.R., Lovell, M.A., Yatin, M., Dhillon, H., Markesbery, W.R., 1998. Regional membrane phospholipid alterations in Alzheimer's disease. Neurochem Res 23, 81-8.

Putnam, N.H. et al., 2007. Sea Anemone Genome Reveals Ancestral Eumetazoan Gene Repertoire and Genomic Organization. Science 317,86-94.

Quintana, C., Cowley, J.M., Marhic, C., 2004. Electron nanodiffraction and high-resolution electron microscopy studies of the structure and composition of physiological and pathological ferritin. J Struct Biol 147, 166-78.

Quintana, C., Bellefqih, S., Laval, J.Y. et al., 2006. Study of the localization of iron, ferritin, and hemosiderin in Alzheimer's disease hippocampus by analytical microscopy at the subcellular level. J Struct Biol 153, 42-54.

Rabary, O., Boussofara, M., Grimaud, D., 1994. Acid-base equilibrium and the brain Ann Fr Anesth Reanim 13, 111-22.

Ravikumar, A., Deepadevi, K.V., Arun, P., Manojkumar, V., Kurup, P.A., 2000.Tryptophan and tyrosine catabolic pattern in neuropsychiatric disorders. Neurol India 48, 231-8.

Richardson, A.J., Easton, T., Gruzelier, J.H., Puri, B.K., 1999. Laterality changes accompanying symptom remission in schizophrenia following treatment with eicosapentaenoic acid. Int J Psychophysiol. 34, 333-9.

Rizi, R.R., Ahn, S., Alsop, D.C., 2000. Intermolecular zero-quantum coherence imaging of the human brain. Magn Reson Med 43, 627-32.

Roberts, E., Sherman, M., 1993. GABA—The quintessential neurotransmitter: Electroneutrality, fidelity, specificity, and a model for the ligand binding site of GABAA receptors. Neurochem Res 18, 365-376.

Rodriguez, A., Whitson, J., Granger, R., 2004. Derivation and analysis of basic computational operations of thalamocortical circuits. J Cogn Neurosci 16, 856-77.

Roelfsema, P.R., Engel, A.K., König, P., Singer, W., 1997. Visuomotor integration is associated with zero time-lag synchronization among cortical areas. Nature 385, 157-61.

Rörig, B., Klausa, G., Sutor, B., 1995. Dye coupling between pyramidal neurons in developing rat prefrontal and frontal cortex is reduced by protein kinase A activation and dopamine. J Neurosci 15,7386-400.

Rorig, B., Klausa, G., Sutor, B., 1996. Intracellular acidification reduced gap junction coupling between immature rat neocortical pyramidal neurones. J Physiol 490, 31-49.

Ross, B.M., Turenne, S., Moszczynska, A., Warsh, J.J., Kish, S.J., 1999. Differential alteration of phospholipase A2 activities in brain of patients with schizophrenia. Brain Res 821, 407-13.

Roth, B.J., Wikswo, J.P.Jr., 1985. The magnetic field of a single axon. A comparison of theory and experiment. Biophys J. 48,93-109.

Ringach, D.L., Malone, B.J., 2007. The operating point of the cortex: neurons as large deviation detectors. J Neurosci 27, 7673-83.

Ryan, T.J., Grant, S.N., 2009. The origin and evolution of synapses. Nature Reviews Neuroscience 10, 701-712

Sadowska, G.B., Stopa, E.G., Stonestreet, B.S., 2009. Ontogeny of connexin 32 and 43 expression in the cerebral cortices of ovine fetuses, newborns, and adults. Brain Res 1255,51-6.

Satinover, J., 2001. The Quantum Brain: The Search for Freedom and the Next Generation of Man. Wiley.

Scemes, E., Dermietzel, R., Spray, D.C., 1998. Calcium waves between astrocytes from Cx43 knockout mice. Glia. 24, 65-73

Sewell, K.K., 1999. The Cosmic Sphere. Barnes&Noble.

Siegel, G.J., Agranoff, B.W., Fisher, S.K., Albers, R.W., Uhler, M.D., 1999. Basic Neurochemistry Sixth Edition.

Shiu, G.K., Nemoto, E.M., 1981. Barbiturate attenuation of brain free fatty acid liberation during global ischemia. J Neurochem 37, 1448-56.

Smith, J.M., article for Carter, R., 1998. Mapping the mind. page 157.

Smith, S.S., Waterhouse, B.D., Chapin, J,K., Woodward, D.J., 1987 Progesterone alters GABA and glutamate responsiveness: a possible mechanism for its anxiolytic action. Brain Res 400, 353-9.

Springer, S.P., Deutsch, G., 1993. Left brain, right brain fourth edition, W. H. Freeman and Company.

Sotty, F., Danik, M., Manseau, F., Laplante, F., Quirion, R., Williams, S., 2003. Distinct electrophysiological properties of glutamatergic, cholinergic and GABAergic rat septohippocampal neurons: novel implications for hippocampal rhythmicity. J Physiol 551, 927-43.

Stevens, P.,S. 1974. The similarity between the pattern of the magnetic domains and that in the brain coral is pointed out as the meanders. Patterns in Nature, Little, Brown & Co., 1974.

Strachan, T., Read, A.P., Leptospira, In., 1999. Human Molecular Genetics, 2nd ed., Wiley-Liss.

Takeuchi, K., Takigawa, M., Fukuzako, H., et al, 1994. Correlation of third ventricular enlargement and EEG slow wave activity in schizophrenic patients. Psychiatry Res 55, 1-11.

Tarazi, F.I., 2001. Neuropharmacology of dopamine receptors: Implications in neuropsychiatric diseases. QU Journal For Scientific Research: Medical Sciences 3, 87–104.

Thompson, P.M., Vidal, C., Giedd, J.N. et al 2001. Mapping adolescent brain change reveals dynamic wave of accelerated gray matter loss in very early-onset schizophrenia. Proc Natl Acad Sci U S A 98, 11650-5.

Thompson, P.M., Hayashi, K.M., de Zubicaray, G., et al, 2003. Dynamics of gray matter loss in Alzheimer's disease. J Neurosci 23, 994-1005.

Thompson, A.J., Lummis, S.C., 2003. A single ring of charged amino acids at one end of the pore can control ion selectivity in the 5-HT3 receptor. Br J Pharmacol 140, 359-65.

Tiesinga, P.H., José, J,V., 2000. Robust gamma oscillations in networks of inhibitory hippocampal interneurons. Network 11, 1-23.

Tiihonen, J., Kuikka, J., Rasanen, P., 1997. Cerebral benzodiazepine receptor binding and distribution in generalized anxiety disorder: a fractal analysis. Mol Psychiatry 2, 463-71.

Toga, A.W., Thompson, P.M., 2003. Mapping brain asymmetry. Nat Rev Neurosci 4, 37-48.

Traub, R.D., Schmitz, D., Jefferys, J.G., Draguhn, A., 1999. High-frequency population oscillations are predicted to occur in hippocampal pyramidal neuronal networks interconnected by axoaxonal gap junctions. Neuroscience 92, 407-26.

Tuszynski, J.A., 2006. The Emerging Physics of Consciousness Series: The Frontiers Collection. 385-404.

Vallortigara, G., Pagni, P., Sovrano, V,A., 2004. Separate geometric and non-geometric modules for spatial reorientation: evidence from a lopsided animal brain. J Cogn Neurosci 16, 390-400.

VanRullen, R., Koch, C., 2003. Is perception discrete or continuous? Trends Cogn Sci 7, 207-213.

Vianin, P., Posada, A., Hugues, E., et al, 2002. Reduced P300 amplitude in a visual recognition task in patients with schizophrenia. Neuroimage 17, 911-21.

Vinogradova, O.S., Kitchigina, V.F., Zenchenko, C.I., 1998. Pacemaker neurons of the forebrain medical septal area and theta rhythm of the hippocampus. Membr Cell Biol 11, 715-25.

Volterra, A., Trotti, D., Racagni, G., 1994. Glutamate uptake is inhibited by arachidonic acid and oxygen radicals via two distinct and additive mechanisms. Mol Pharmacol 46, 986-92.

Walther, J.S., 2000. The Story of Unified Cordic. The Journal of VLSI Signal Processing 25, 107-112.

Wang, X.J., 2002. Pacemaker neurons for the theta rhythm and their synchronization in the septohippocampal reciprocal loop. J Neurophysiol 87, 889-900.

Ward, L.M., Doesburg, S.M., Kitajo, K., MacLean, S.E., Roggeveen, A.B., 2006. Neural synchrony in stochastic resonance, attention, and consciousness. Can J Exp Psychol 60, 319-26.

Weiss, V., 1992. The relationship between short-term memory capacity and EEG power spectral density. Biol Cybern 68, 165-72.

Weiss, V., 1989. From short-term memory capacity toward the EEG resonance code. Person individ Diff 10, 501-508.

Weissman, T.A., Riquelme, P.A., Ivic, L., Flint, A.C., Kriegstein, A.R., 2004. Calcium waves propagate through radial glial cells and modulate proliferation in the developing neocortex. Neuron. 43, 647-61.

Williard, D.E., Harmon, S.D., Preuss, M.A., Kaduce, T.L., Moore, S.A., Spector, A.A., 2001. Production and release of docosahexaenoic acid by differentiated rat brain astrocytes. World Rev Nutr Diet 88, 168-72.

Wolpert, L., Beddington, R., Jessell, T., Lawrence, P., Meyerowitz, E., Smith, J., 2002. Principles of Development, Second Edition. Oxford University Press

Womelsdorf, T., Schoffelen, J.M., Oostenveld, R., 2007. Modulation of neuronal interactions through neuronal synchronization. Science 316, 1609-12.

Woo, T.U., Walsh, J.P., Benes, F.M., 2004. Density of glutamic acid decarboxylase 67 messenger RNA-containing neurons that express the N-methyl-D-aspartate receptor subunit NR2A in the anterior cingulate cortex in schizophrenia and bipolar disorder. Arch Gen Psychiatry 61, 649-57.

Wu, J.Y., Huang, X., Zhang, C.. 2008. Propagating waves of activity in the neocortex: what they are, what they do. Neuroscientist 14, 487-502.

Xu, J., Yu, S., Sun, A.Y., Sun, G.Y., 2003. Oxidant-mediated AA release from astrocytes involves cPLA(2) and iPLA(2). Free Radic Biol Med 34, 1531-43.

Xu, W., Huang, X., Takagaki, K., Wu, J.Y., 2007. Compression and reflection of visually evoked cortical waves. Neuron 55, 119-29.

Zhong, W., Gallivan, J.P., Zhang, Y., Li, L., Lester, H.A., Dougherty, D.A., 1998. From ab initio quantum mechanics to molecular neurobiology: a cation-pi binding site in the nicotinic receptor. Proc Natl Acad Sci U S A. 95, 12088-93.

Web references

Allan, D.W., 2004. Quadrupole magnets were used in previous atomic clocks. http://www.pureenergysystems.com/news/2004/06/28/6900027AtomicClockQuadrupoles/index.html 24/06/2009

Ashton, C.H., 2002. Benzodiazepines : How they work and how to withdraw. http://www.benzo.org.uk/manual/bzcha01.htm Aug 30/11/2008

Bazhenov, M., Timofeev, I., 2006. Thalamocortical oscillations. http://www.scholarpedia.org/article/Thalamocortical_oscillations 30/11/2008.

Begany, T., 2004. Emerging Schizophrenia Treatments Aim to Enhance NMDA Receptor Function. http://www.neuropsychiatryreviews.com/aug04/npr_aug04_schizo.html. Aug 30/11/2008

Collins, D.L., Kabani, N.J, Evans A.C., 1998. Automatic volume estimation of gross cerebral structures. http://www.bic.mni.mcgill.ca/~louis/papers/hbm98b/index.html#Collins96a 12/04/2005

Dhar, A.S., Banerjee, S., 1995. Doppler ultrasonograph with CORDIC based spectrum analyzer forblood flow velocity estimation. http://ieeexplore.ieee.org/xpl/freeabs_all.jsp?arnumber=532181 30/11/2008.

Doherty, A., 2001. Centre for Synaptic Plasticity. Glutamate Receptors - Structures and Functions http://www.bris.ac.uk/synaptic/info/tools.html 30/03/2009

Granger, R.H., Hearn, R.A., 2007. Models of thalamocortical system. http://www.scholarpedia.org/article/Models_of_thalamocortical_system#Oscillation-Assisted_Processing 30/11/2008.

Hameroff, S., 2007b. Life, health and consciousness - A quantum perspective, Netherlands lecture slides. Page 99. http://www.quantumconsciousness.org/netherlands1.htm 30/04/2009

Hameroff, S., 2009. The "conscious pilot"—dendritic synchrony moves through the brain to mediate consciousness. http://www.springerlink.com/content/a415kw68517m2v87/?p=53a9d7edb3c04cf5bfc1bf563491e1ca&pi=0 02/05/2009.

Hamilton, R.J., et al., 2001. How the brain puts the pieces together: Sex differences in integrative function. http://www.braindynamics.med.usyd.edu.au/braindynamics/publications/pub_files/Hamilton_Gamma.pdf 24/04/2009

Harrison, S.A., Tong, F. Decoding reveals the contents of visual working memory in early visual areas. http://www.nature.com/nature/journal/vaop/ncurrent/abs/nature07832.html 30/02/2009

Ikonen S., 2001, The role of the septohippocampal cholinergic system in cognitive functions. http://www.uku.fi/neuro/54the.htm 30/11/2008.

Kondor, I., Barabási, A.L., Billoire, A., Saclay, N., Gulyás, N., Lukic, J., Marinari, E. Strong Random Correlations in Complex Systems http://carbon.videolectures.net/2008/pascal2/eccs08_jerusalem/kondor_srcics/eccs08_kondor_srcics_01.pdf 25/03/2009

Korn, M.L., 2000. Glutamatergic and GABAergic based Issues in Schizophrenia. http://www.medscape.com/viewarticle/420834 30/11/2008.

Lago, N.P., Kon, F., 2004. The quest for low latency. http://www.ime.usp.br/~kon/papers/icmc04-latency.pdf 30/11/2008.

Moss, S.J., 2006. The synaptic targeting and regulation of inhibitory amino acid neurotransmitter receptors. http://www.ucl.ac.uk/Pharmacology/Research/sm.html 30/03/2009

Palm, R., Korenivski, V., 2007. A ferrofluid based neural network: design of an analogue associative memory. http://arxiv.org/abs/0705.3402 30/11/2008.

Rosack, J., 2002. Research Reveals More About Alcoholism's Complex Action on Brain. http://www.pn.psychiatryonline.org/cgi/content/full/37/18/21-a 30/11/2008.

Schnetter, E. Carpet — Adaptive Mesh Refinement for the Cactus Framework. http://www.carpetcode.org/pictures/quadrupole.jpeg 30/03/2009.

Steffek, A.E., The Role of Astrocytes in the Pathophysiology of Schizophrenia. http://hdl.handle.net/2027.42/55679 30/11/2008.
Ujfalussy, B., Kiss, T., Hajos, M., Erdi, P., 2007. Theta synchronization in the medial septum and the role of the recurrent connections. http://geza.kzoo.edu/~ubi/publications/CNSpos.pdf 30/11/2008.

FIGURE REFERENCES

Belcher, J., 2005. MIT Physics 8.02. Electricity and Magnetism. http://web.mit.edu/8.02t/www/802TEAL3D/visualizations/magnetostatics/MagneticForceAttract/MagneticForceAttract.htm 30/03/2008

Hubel, D., 1998. Eye, brain and vision. New York: American Scientific Library. 138-139.

Williams, T.H., Gluhbegovic, N., 1980. The human brain : a photographic guide. Harper and Row Publishers, Inc., Hagerstown

Basser, P.J., Pajevic, S., Pierpaoli, C., Duda, J., Aldroubi, A., 2000. In vivo fiber tractography using DT-MRI data.
Magn Reson Med 44, 625-32.

Thomspon et al, 2001,2003 (in main references)

Hardenbergh. J., 2004. fMRI Visualization of Multiple Functional Areas. http://www.jch.com/volumes/fmriviz.htm 30/03/2009

Ellis, H., Logan, B., Dixon, A., 1991. Human cross-sectional anatomy. Butterworth Heinemann

URMC 2009. http://www.urmc.rochester.edu/smd/Rad/diagneuro.htm 24/04/2009

Zani, G., 2000. Electricity and Magnetism 5G20.20 Magnetic Domains. http://www.cns.brown.edu/physics/demopages/Demo/em/demo/5g2020.htm 24/4/2009.

Cord, J., Westwood, J., 2001. Domain optimization of sputtered permalloy shields for recording heads. IEEE Transactions on Magnetics 37,1755-1757.

Cooper, K.B., 2003. New phases of two-dimensional electrons in excited Landau levels. http://resolver.caltech.edu/CaltechETD:etd-05272003-170504 30/11/2008.

Rubin, M., Safdieh, J.R., 2007. Netter's Concise Neuroanatomy. Elsevier.

Douady, S., Couder, Y., 1992. (in online references)

Bonhoeffer, T., Grinvald, A., 1991. Iso-orientation domains in cat visual cortex are arranged in pinwheel-like patterns. Nature 353,429-31

McLaughlin, D., Shapley, R., Shelley, M., Wielaard, D.J., 2000. A neuronal network model of macaque primary visual cortex (V1): orientation selectivity and dynamics in the input layer 4Calpha. Proc Natl Acad Sci USA 97, 8087-92.

Harrison, F.J., 2004. Density Difference and Reduced Quadrupole Density Plots for Homonuclear Diatomics. http://www.cem.msu.edu/~harrison/quadrupole/quad.html 24/04/2009.

Review of the Universe, 2009. Multicellular Organisms, 10-80 in website: www.universe-review.ca

Weisstein, E., 2009. Spherical Harmonic. http://mathworld.wolfram.com/SphericalHarmonic.html 30/03/2009

Best, B., 2009. http://www.benbest.com/science/anatmind/anatmd7.html 24/03/2009.

Sundsten, J.W., 2009. Digital Anatomist Project. http://www9.biostr.washington.edu/da.html 24/03/2009.

Nieuwenhuys, R., Voogd, J., van Huijzen, C., 1988. The Human Central Nervous System. Berlin: Springer-Verlag :300

Rienstra, D., 2009. http://www.rienstraclinic.com/newsletter/2007/2007Jan.html 24/03/2009.

Schnetter, E., 2009: (in online references)