TENSEGRITY SYSTEMS WITHIN NEURON CHEMISTRY

(2nd paper of supporting evidence for a Brain Dipole model)

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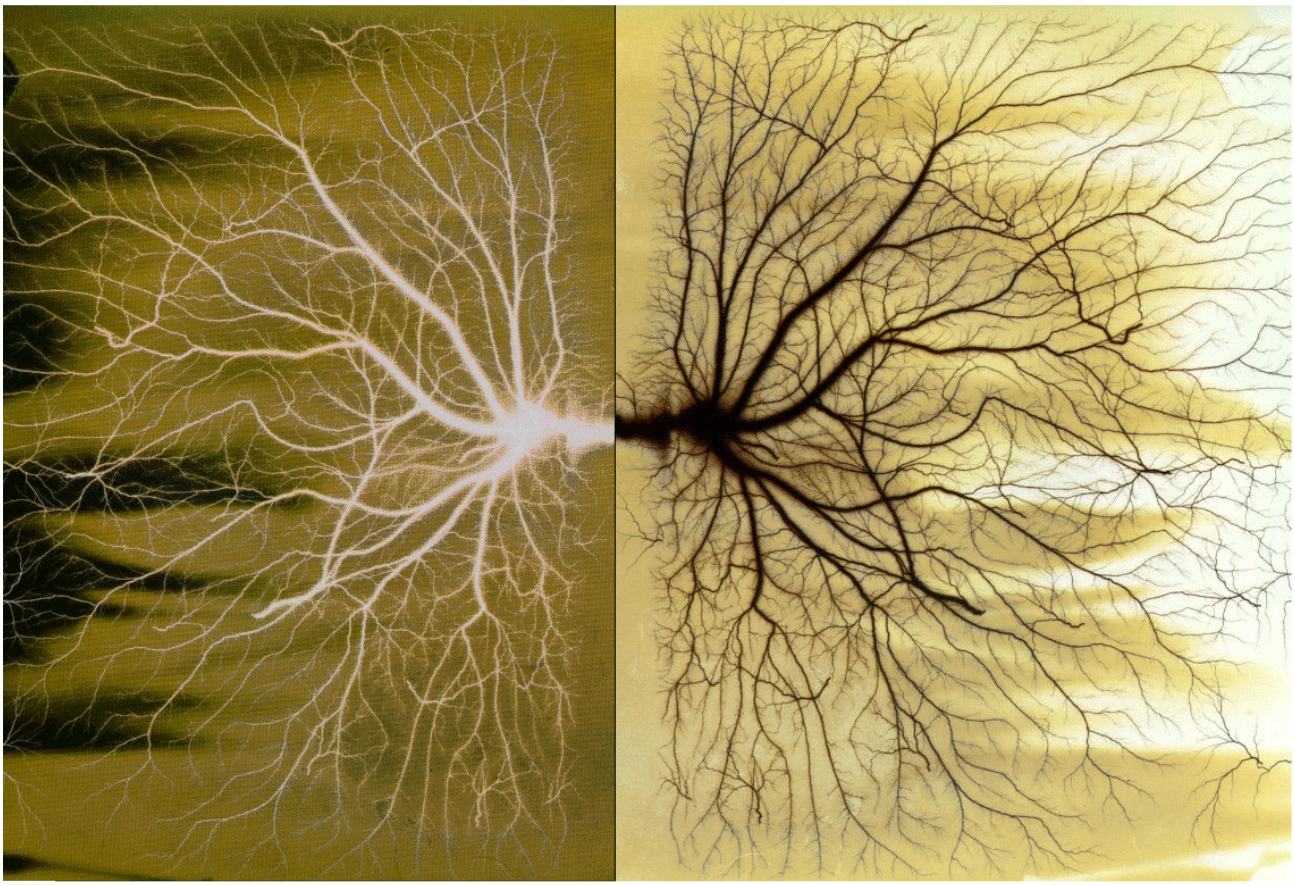

ABSTRACT

Searching for convergent / divergent aspects of brain chemistry reveals another view that the brains prevailent Glutamate / Gaba system is lateralized in the Right and Left hemispheres respectively. It is considered that this system self organized from an extracellular tensegrity which exists in oppositely charged ions such as sodium and chlorine. These ions are proposed to be respectively lateralized in the right and left hemispheres, re-enforcing that the left hemisphere acts upon a greater percentage of negative charges and the right side positive, which fits with current models of lateralization in schizophrenia.

Laying this out graphically a surprising lateralized symmetry has been found in the Glutamate / Gaba receptors which operate the extracellular pumps which pull ions into neurons. The extracellular ions are the focus of this paper, primarily because it is these which are responsible for the electromagnetic EEG reading. Analysis of the receptors themselves also highlight that Gaba is a convergent system, and glutamate a divergent system, which reflect the chemical findings here also.

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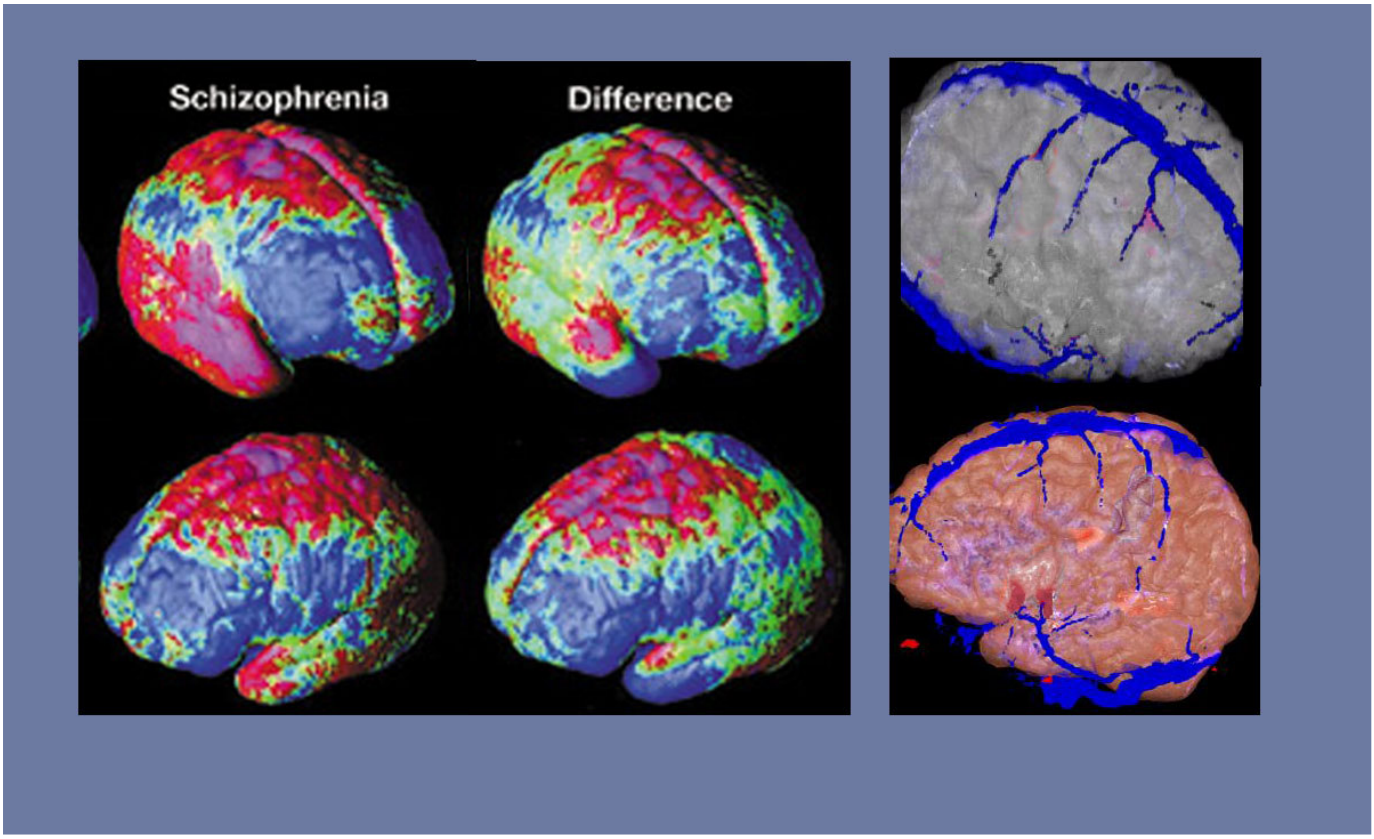
INTRODUCTION

This is the first of two technical reviews, which set out to use a tensegrity framework as a means of unravelling neuron chemistry. This paper deals with the majority of brain neurons which are Glutamate excitatory neurons with Gaba inhibition. "Neurochemical modulators" the next in this series (3) described how modulation neurons such as dopamine and serotonin mainly triggered cellular messengers rather than ions, and that the neurotransmitters themselves had chemical properties with direct ion effects for their receptors. The two systems *(glutamate / gaba and dopamine / serotonin)* operate together because they are so different, they do not need each others resources.

This section will describe how the reverse is true for the glutamate / Gaba system, *(that these neurotransmitters are keys to pull sodium and chlorine ions* (**respectively Sodium for glutamate and chlorine for Gaba**) *within the neuron for fast and reverse opposite results)* Since this section is heavily based upon the ionic basis of the vast majority of brain neurons, It begins with a refresher on the subject. The table on the next page is a guide to the basic ionic charges and concentrations in Invertebrates and Vertebrates cells. This is to serve as an introduction to the kinds of elements and molecules which balance charge throughout cells, and cellular components of the nervous system.



Reminder of diagram from previous section. The premise of this section is to provide a case for lateralization of sodium and chlorine in the right and left hemisphere respectively. This would explain these obvious visual lateral differences shown here by damage to glutamate neurons in schizophrenia.

1.1 BASIC IONS OF THE NERVOUS SYSTEM

Ion	Cell (mM)	Blood (mM)
SQUID AXON*		
Na+	50	440
Cl–	40 – 150	560
K+	400	20
X–†	300 – 400	5 – 10
Ca2+	0.0003	10
MAMMALIAN CELL		
Na+	12	145
Cl–	4	116
HCO3–	12	29
K+	139	4
X–	138	9
Ca2+	<0.0002	1.8
Mg2+	0.8	1.5

Table adapted from Molecular Cell Biology, W. H. Freeman

* The large nerve axon of the squid, an invertebrate cell, has been widely used in studies of the mechanism of conduction of electric impulses.
† X–represents proteins, which have a net negative charge at the neutral pH of blood and cells.

For the purposes of this review, ions in blood plasma are similar to fluid outside neurons. In the table above the largest negative values equate to X- (intracellular proteins and phosphate), Cl- (Chlorine) and positive charges of K+ (potassium) and Na + (sodium). The positive and negative charges balance each other out on either side of the cell.

The table has been adapted to group the larger quantities of ions into what is considered their natural positive / negative pairings. Sodium and chlorine as a grouping first, for their obvious prevalence in all living systems. In the mammalian cell example, the author has the presence of (HCO3-) bicarbonate. This is a natural biochemical acidity regulator, and would also be presumed to be present in squid axons also. Calcium and magnesium are grouped together. These were found to work with each other in a homeostatic manner throughout the entire nervous system. (04)**.** Magnesium is not listed present in squid axon by the author.

Adapting the table, by grouping in this manner balances the quantities of charge, while leaving potassium and cellular proteins together. The reason for pointing out ionic grouping in this way, is primarily to draw attention to the grouping of the elements sodium and chlorine. These ions will be investigated here in a new role as key balance elements within the brain system itself.

1.2 IONS OF THE CEREBROSPINAL FLUID

In electrolytic terms CSF (Cerebrospinal Fluid) is at physiological pH due to it's largest electrolytic components sodium and chlorine. (65) Neurochemistry itself is a system which maintains an optimum pH of 7.4 (66) through means of a sodium chloride Bicarbonate buffered homeostasis regulated within the CSF and ventricles. (8, 9, 11, 19, 20, 21)

"In physiological conditions, the regulation of acid-base balance in brain maintains a noteworthy stability of cerebral pH." 66

In comparison to the other ions in the table above, it can now be seen that the degree to which sodium and chlorine operate to innervate extra cellular and blood brain chemistry is pronounced. A great deal of chemistry in physiology and neurobiology defines chemical process according to their dependence or independence to sodium and chlorine. (12, 14, 15, 16, 17, 18, 19)

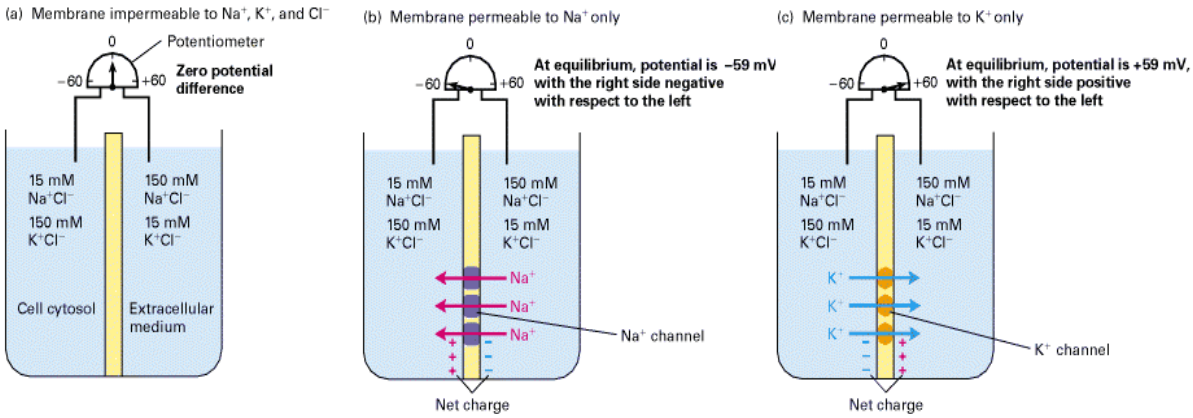
1.3 SODIUM AS A PLAYER IN NEUROTRANSMISSION

Sodium plays a key role in neurotransmission, by remaining mainly outside the nerve cell as part of the extra cellular fluids. In the brain, extra cellular fluid is derived from blood products and Cerebrospinal fluid by passive diffusion. (21)

"Diffusion is the net movement of material from an area of high concentration of that material to an area with lower concentration. The difference of concentration between the two areas is often termed as the concentration gradient, and diffusion will continue until this gradient has been eliminated." (67)

In neurotransmission sodium is a key player in active transport with potassium in the well known Na+/K+-ATPase. Active transport is the mediated transport of biochemical's, and other atomic/molecular substances, across membranes. (68)

ATPase is an enzyme reaction which controls diffusion of sodium and potassium across their concentration gradients in precisely controlled quantities. (69)



This figure above (from Molecular Cell Biology, W. H. Freeman) gives a simplified demonstration of the essential ions for simulation of the sodium / potassium pump. The positive ions are Sodium / Potassium and negative ions Chlorine. An extra amount of chlorine is present to compensate for the missing negatively charged intracellular proteins X listed in the ion table above. As can be seen, **in ion terms the negatively charged components look as if they do very little.** While the positive ions do all the cycling and shuffling. Setting up isolated experiments at a lower PH of 5.5 demonstrates more clearly how chlorine is capable of activating these currents from its own presence. (13)

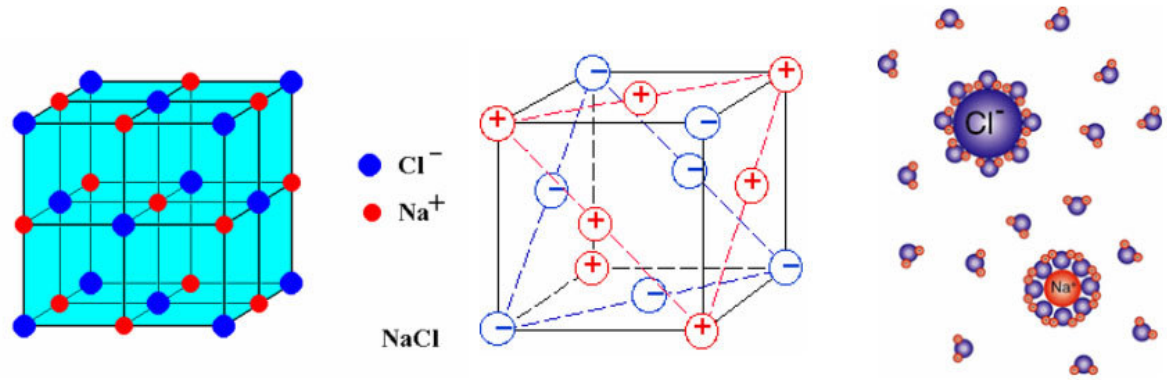
1.4 TENSEGRITY AND ION SIZE

Properties of cellular transport systems are greatly defined in terms of their dependence on sodium and chlorine. (12, 15) These kind of reciprocal anion / cation scenarios which facilitates cellular transport mechanisms are also firmly dependant on the size of these small ions. Negatively charged Anions can only be efficiently replaced by smaller anions. (10) Is the reverse is true of cations**. ?**

Predicting an opposite of the reducing anion situation within sodium / chlorine based systems is sought after here in the hunt for Tensegrity system symmetry. Using this method yet again, predicts that a reverse scenario to the reducing anion size might apply whereby cations can be effectively replaced by larger cations. This does not need any investigation. It is how the cellular system operates. The larger size of potassium ions push out at the sodium in the cycling cation gradients of the sodium potassium pump. For simplicity the relationship on ions as a system will concentrate on extra cellular ions. The next section looks at the tensegrity bonds which exist between sodium and chlorine.

2 TENSEGRITY OF SODIUM CHLORIDE (NaCl)

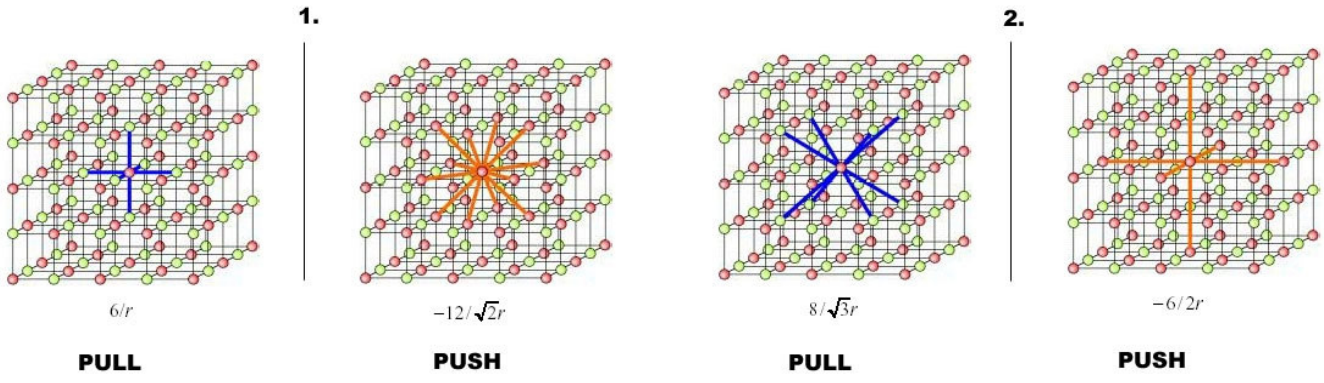
The section following this redescribes brain structure as an organ which has lateralized sodium / chlorine ion gates. Giving it some degree of opposite charge. *(although still layered and entwined throughout the system)* To understand the behaviour of these ions requires looking at sodium and chlorine properties isolated and in solution.



Sodium chloride (NaCl) crystal structures, have already been proposed as a tensegrity system itself. (70) Do these properties still retain enough tensegrity within solution to provide the necessary electrical ingredients from which electromagnetic tensegrity would develop ?

Traditional tensegrity structures such as geodesics, defined themselves by surface structures. These were said to have increased integrity, as the number of components increased, the push / pull relationships would still remain if the structure was compromised. NaCl has a deep intrinsic cubic structure, deriving from every atom, in every direction. *(see second image in figure above)* Salt water as a solution still has the taste of salt. Not of sodium or chlorine. The elements still retain their push pull relationship through electronegative bonding. The properties are described by the cubic lattice interactions given above. Even while in solution a "loose" cubic push pull interaction would take place, because a cubic structure is the only means by which sodium and chlorine electrons interact.

This aspect will be looked at next. Following this, the rest of the section is more technically demanding, because it is constructed from re-interpreting the current research in neurotransmission within the context of this sodium chloride basis. The fact that is has been pretty easy to do this, with the surprising bulk of evidence falling into the proposed structure, is testament both to scientific research itself and a proposed validation of the dipole neurology theory (1).



Tensegrity patterns within sodium chloride crystal, taken from visualizations of Madelung constant (174) (***The energy of a particular crystal structure relative to the same number of isolated molecules)*** A symmetry of constant and converging quantities appear to recurse for both interactions with attraction and repulsion

The above examples looks into the qualities of dry NaCl crystal, which resolves not unexpectedly into a structural tensegrity of the most classic kind. From left to right. Sodium is red, chlorine is yellow. In (1) each sodium pulls 6 chlorine neighbours within it's own radius whole pushing 12 sodium atoms at less than twice the original radius

(sqrt 2r). (2) Moving outwards in the structure again : sodium pulls 8 chlorine neighbours within less than 3 times the original radius and pushes 6 sodium atoms at exactly twice its original radius.

(As a mathematical reference putting the values from the above gives the Madelung Constant (A) = [6 - 12/sqrt 2 + 8/sqrt 3 - 6/2 + 24/ sqrt 5] = 1.74756.)

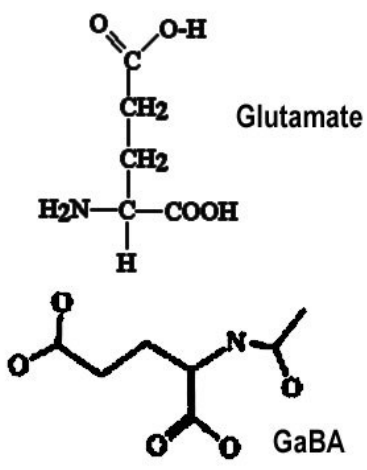
Looking at the patterns from the NaCl lattice. For equal values of R, the number of constant connections remains 6 throughout any scale.

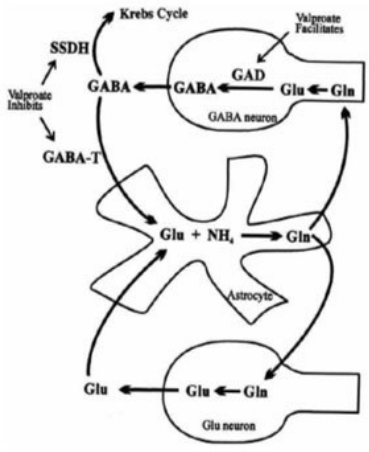
What's of interest in tensegrity terms is that the constant and changing quantities within NaCL appear to recurse for both interactions with attraction and repulsion. That is they appear if beginning outwards from Sodium or chlorine in the centre, while working outward through the increasing connective radius. Although the madelung constant is not specific to the atomic qualities of Sodium and chlorine, (70) this cubic kind of charge opposite symmetrical tension and integrity exists for NaCl as a crystal and only a handful of other organic element combinations.

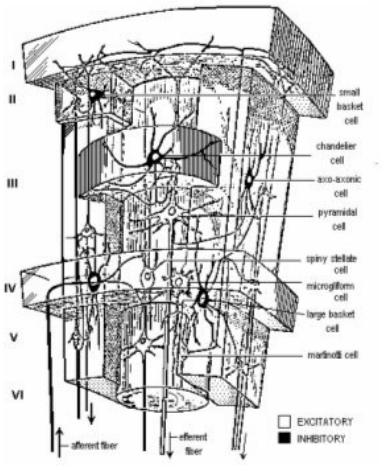
2.1 AMINO ACIDS COMPONENTS OF NEUROTRANSMITTERS

Most of the components of biology posses some type of propensity for holding charge in acid / base terms. (200) Every amino acid has acid and base properties, (6) the "R" groups can determine whether the molecule tends to be polar (hydrophilic), nonpolar (hydrophobic), acidic, basic or neutral. (6, 7)

Glutamate is the primary neurotransmitter in the cortex. It's R-group is a carboxyl group, giving it a "Dibasic" quality of double negative charges (23). This makes it the most charged of all the amino acids. The brake on glutamate transmission is Gaba which is derived from glutamate within a related chemical cycle between Glutamate/Gaba neurons and astrocytes. (22) Out of all the neurotransmitter transporters glutamate has the greatest requirements for sequencing Na+ ions (3), followed by Gaba (2) with the remaining co-modulators transmitters such as serotonin or dopamine having (1). (63) The greater requirements and prevalence of glutamate as the brains master chemical could be due to this dibasic structure among the amino group, facilitating it extra ionic leverage.

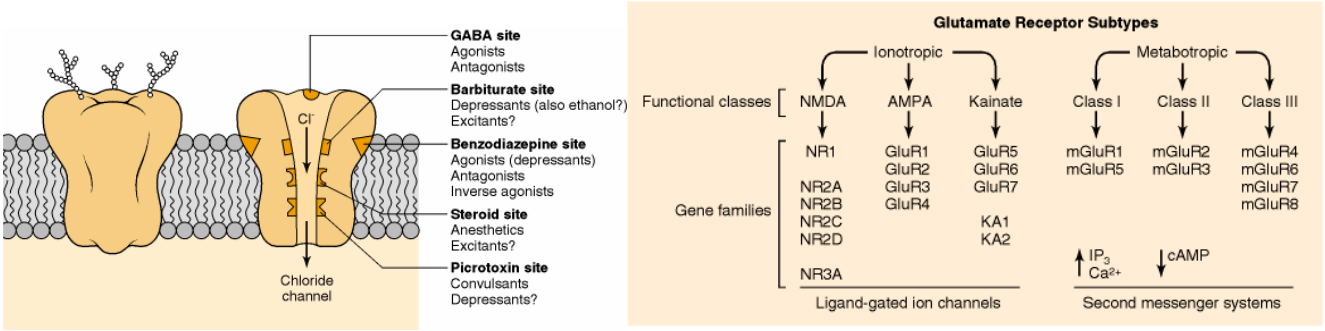






MIDDLE : Chemical relationships between glutamate and gaba. Gaba is derived from glutamate. RIGHT: A slice of cortical grey matter illustrating visually the interdependence of glutamate excitatory (white) and gaba inhibitory neurons (black).

3 CONVERGENT / DIVERGENT ASPECTS OF GLUTAMATE / GABA SYSTEM



http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=bnchm.figgrp.1185

The ratio of cortical glutamate to gaba neurons is 80/20. (74) This ratio would tend towards, cortical ionic chaos if not synchronised by sensory thalamic input, (75) the thalamic / cortical pathway contains mainly longer lasting G-protein GabaB inhibitory receptors. (76)

The amounts of cortical sodium and chlorine released may not reflect the 80/20 neuron ratio. Cortical Glutamate will be firing a divergent range of responses, mixing metabotropic cascades with the ionotropic release, while Ionotropic GabaA and C receptors are entirely cortical with many triggers firing these same targets. The ratio of cortical gaba synapses is estimated at 40%. (78)

Convergent networks terminate as a many onto one focus. Gaba has a diverse number of transmitters directed at a single chloride channel. *(see diagram above)* Divergent networks activate many types of response from a single source. Glutamate is a divergent network. The same transmitter can activate many kinds of receptors and responses. *(see diagram above)*

The next section will begin the primary investigation as to whether the sought after convergent / divergent system symmetry is immediately obvious by looking at existing research into neurotransmission and after that lateralization.

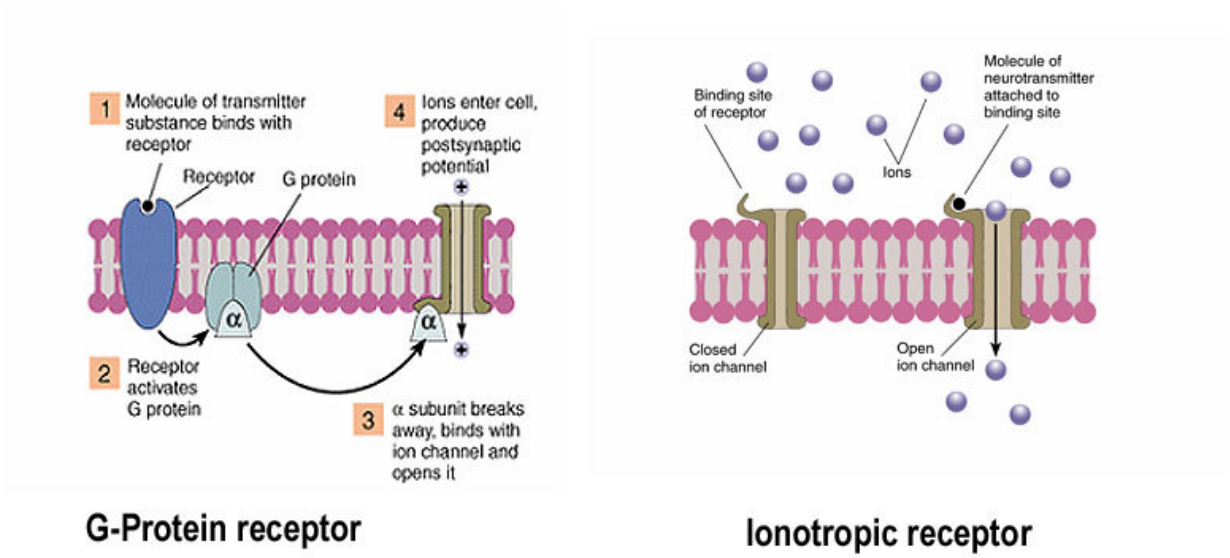
3.1 BACKGROUND TO SYSTEMS INVESTIGATION

One aim of this research is to go through as many brain systems as possible to find chemical and biological levels which fit the convergent / divergent profile initially proposed for left / right hemisphere in Dipole Neurology draft 1. Because the aim is to fit the theory to the data, and there is so much data, any evidence which arises that does not fit, requires stopping to re-evaluate and diverge to research this. Doing this research is a personal journey of discovery, motivated by the apparent cortical symmetry, and well known reverse behaviours from each side of the brain. *(The convergent / divergent aspects were proposed to exist in Behavioural psychology, many levels of Neuropsychology, and Neuroscience (1).)*

From that agenda, when answers do not come easily, the adventure is to re-evaluate, because somewhere along the line, it will fall back together. Answers will exist which reflect these basic facts of reverse symmetry. Much of this research has come together so easily, that the re-evaluation just for the sake of deeper insight and not to make everything fit the dipole model, is really what matters. Initially the basic idea was that neurotransmitters would at least reflect the negative / positive charge requirements for the left / right hemisphere respectively. Glutamate with a double negative charge, and Gaba with a positive / negative charge (23) does not fit. However the fact that double charges exist on these molecules adds the same level of complexity when faced with wondering how two positive ions, sodium and potassium operate a firing gradient. The one positive charge of Gaba can still act as a brake on the two negatives of glutamate. That is all that matters here. Glutamate is required, to predominately activate release of fast acting ionotropic receptors. A switch like key is all that is needed here, unlike the G-protein receptors of serotonin and dopamine, (3) which may require a stable representation of the ionic requirement from the neurotransmitter within the receptor to resonate for a longer period.

3.2 GABA / GLUTAMATE AS LATERALIZED KEYS FOR NaCl

Glutamate / Gaba molecules are prevalent throughout brain systems. These are the primary keys which operate the underlying ionotropic systems mentioned previously. The Gaba / glutamate receptors, combine metabotropic (chemical trigger) and ionotropic (electrical trigger). The Metabotropic receptors appear to mediate activation of the neighbouring ionotropic receptors. Glutamate ionotropic receptors pull sodium from the extracellular fluid into the receptor. Gaba ionotropic receptors pull Chlorine from the extracellular fluid into the receptor. *(evidence of this later)* Each of these are lateralized. Glutamate in the right hemisphere. Gaba in the left hemisphere. *(Evidence of this later)* The charge requirements for the dipole theory are present.

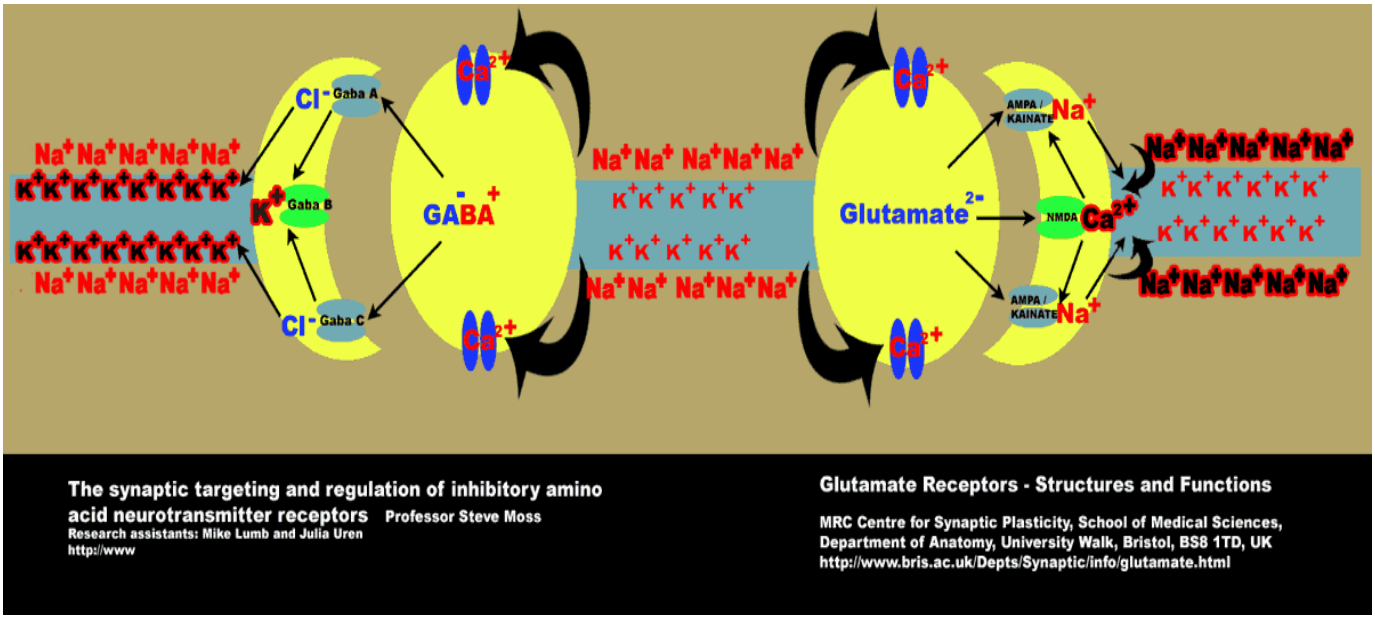


LEFT: G-protein receptors trigger complex intracellular cascades. RIGHT : ionotropic receptors gate intracellular ions.

Re-uptake by sodium transporters is lowest within the co-modulators reviewed in another section in this series (3) all of that chemistry also fits a convergent / divergent tensegrity model. Ph of the molecules was also hemisphere dependent. (3) The reason for this is that co-modulators are derived from the powerful tryptophan / tyrosine amino acids.

The following page shows a diagram which illustrates a lateralized, symmetrical and LCH / RCH opposite role for chlorine and sodium ions respectively in Gaba and Glutamate post synaptic action. Both Gaba and glutamate have been found to be LCH / RCH respectively. Case for this will be made following explanation of the diagram.

4 SODIUM / CHLORIDE SYMMETRY WITHIN GABA GLUTAMATE SYSTEM



Simplified Convergent / Divergent model of Glutamate / Gaba based on left / right brain lateralization.
(Felix Lanzalaco © 2004)

To the **left** of the diagram the presynaptic trigger to Gaba is broadly similar to glutamate. Calcium triggered by nerve impulses pushes Gaba across the synaptic cleft to Gaba A and C receptors which trigger a **hyperpolarizing chlorine channel** and activation of Gaba B receptors which also produces hyperpolarizing potassium.
The hyperpolarization inhibits the following nerve impulse in favour of potassium.

To the **right** of the diagram the Presynaptic trigger to Glutamate. Calcium triggered by nerve impulses pushes Glutamate across the synaptic cleft to NMDA receptors which trigger a depolarizing calcium channel and re-activation of Ampa / Kainate receptors **which produces depolarizing sodium.** The depolarization excites the following nerve impulses in favour of sodium.

The nature of complex tensegrity systems of which the brain is unsurpassed, is that layers of lateralized convergent / divergent chemical systems evolve and intertwine, giving not a 50/50 split ratio of chemical divisions, but a clear percentage bias for each hemisphere. The symmetry with which the basic sodium chloride charge constituents of fluids appear within this system drives the need to make a case here for some degree of left hemisphere lateralization of Gaba / chlorine and right hemisphere lateralization of glutamate / sodium as another level of convergence / divergence within brain hemispheres at neurochemistry level.

4.1 RECAP OF SODIUM POTASSIUM PUMP IONS.

1. It is the chlorine bathed Na K pump, that sends signals down axons.
2. Sodium / potassium pumps facilitate ionic movement
3. Sodium / potassium pumps operate a gradient of revolving double layered charges

These ions are attracted to the negative charge which occurs due to proteins within the intracellular environment and chlorine within the extracellular environment. Gaba acts as a brake on these glutamate activated signals by using negatively charged chlorine and potassium to hyperpolarize an axon (23,29) GABA is believed to act by increasing membrane conductance of chloride ions. (47)

5 THE CASE FOR GABA IN LEFT CEREBRAL HEMISPHERE (LCH)

Gaba receptors are lateralized in the left hemisphere. (24,25,26) GabaA and C receptors are chlorine based. (28) With the proposal that Chlorine is lateralized in the left hemisphere due to this prevailance of GaBA inhibition, or that the chlorine is lateralized simply due to systems self organization around convergent / divergent electromagnetic principles (1). Since Gaba has been well documented as a left hemisphere distribution, the space for this will be used to see if this has any relation to the finding that Phospholipid protein kinase (PKC) is left hemisphere asymmetrical

*56 "**Ca2+/ phospholipid-dependent protein kinase** (PKC) activity was found to be asymmetrically distributed between the two cerebral hemispheres of rat brain, whereas basal protein phosphorylation was not lateralized. **The left cerebral hemisphere (LCH) displayed about 50% more PKC activity in synaptosomal fractions than the right cerebral hemisphere** (RCH)."*

45 "Activation of PKC inhibits the ability of group II and group III mGluRs to regulate transmission at three major synapses in the hippocampal formation. Thus, this effect may be a widespread phenomenon that occurs at glutamatergic synapses throughout the CNS ……Protein kinase C (PKC), which modulates GABA transporter function, exerts its modulatory effects by regulating the availability of syntaxin 1A to interact with the transporter "

34 "Syntaxin 1A and PKC functionally regulate GABA transport in cells that endogenously express these proteins. Syntaxin 1A is a plasma membrane protein involved both in trafficking and (neurotransmitter) vesicle docking and/or fusion and in the direct regulation of Ca2+ channels and cystic fibrosis transmembrane regulator (CFTR) Cl channels"

PKC by peroxidation affects tyrosine. (46) The tyrosine derived hyperpolarizing chemicals (49) are left lateralized. (48,50)

5.1 A CASE FOR GLUTAMATE IN THE RIGHT CEREBRAL HEMISPHERE (RCH)

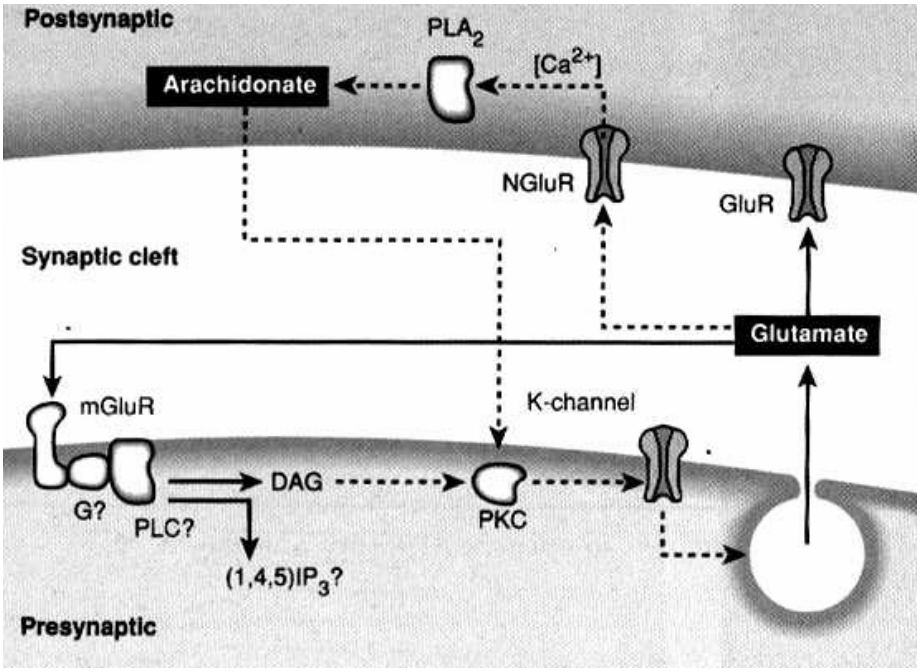
A laterality is expected for glutamate and sodium. Studies of lateralization of neurochemistry was a trend in 70's and 80's when the prevailance of glutamate as a primary neurotransmitter was not studied to the degree of the modulatory neurotransmitters such as Dopamine / Serotonin. A functional right hemisphere lateralization of glutamate will be proposed by :

A. RCH (Right cerebral hemisphere) lateralization of it's modulator Arachidonic acid
B. Controversial studies indicating progesterone decouples the right hemisphere by inhibition of Glutamate.
C. Experiments involving a right lateralized isoprenoid pathway indicate increased modulation of glutamate.
D. Left hemisphere lateralization in schizophrenia, a disorder of NMDA glutamate neurons.

5.1 1. ARACHIDONIC ACID

Arachidonic acid is derived from long-chain polyunsaturated fatty acids (LC PUFA) and is a precursor of pro-inflammatory (PGE2) prostaglandin E2 (60)

Arachidonic acid (AA) and other free fatty acids influence the vesicular uptake of glutamate and (GABA) gamma-aminobutyric acid. (36) GaBA neurons are barbiturate activated. (37) Barbituates attenuate arachidonic acid during global ischemia (72) and DG (diacylglycerol) enriched in arachidonate and stearate (54) The attenuation of AA by LCH GaBA receptor precursors could explain why the lateralization of AA is significantly higher in the right hemisphere (57,58) Mechanisms within the glutamate system have been found which facilitate amplification of glutamate and Calcium release (43) by arachidonic acid. Furthermore these mechanisms are thought
\ to be implicated in NMDA (LTP) Long term potentiation. (43, 42) Behavioral change depends on LTP in the right amygdalo–hippocampus. (79) The next section looks at the AA LTP pathway in more detail.



5.1.2 AA MODULATION OF GLUTAMATE

The AA derived cyclooxygenase-2-generated Prostaglandin E2, (58) arachidonoyldiacylcylglycerol, and arachidonic acid-containing endocannabinoids modulate postsynaptic membrane excitability and long-term synaptic plasticity (53) AA originates at postsynaptic glutamate site in response to Phospholipase platelet A2 (PLA2) activation. PLA2 activating factor is a retrograde messenger of long-term potentiation, a modulator of glutamate release, and an upregulator of memory formation. (53) PLA2 hydrolyzes fatty acids from membrane liberated omega-6 PUFAs (73)

43 "Arachidonic acid may act by binding to a site on the NMDA receptor, or by modifying the receptor's lipid environment. Our results suggest that ***arachidonic acid released by activation of NMDA (or other) receptors will potentiate NMDA receptor currents, and thus amplify increases in intracellular calcium concentration caused by glutamate****. This may explain why inhibition of phospholipase A2 blocks the induction of long-term potentiation"*

5.2.3 PROGESTERONE AND TRANSCOLLOSAL GABA / GLUTAMATE

Neuropsychology studies of the effect of progesterone on brain hemispheres found that left hemisphere performance increased under progesterone due to a decoupling of the right hemisphere. (38) Those results from 2000 are refuted by the reviewers in 2004 (39) while the study has since been duplicated in 2002 without (40) attracting criticsm. In any case progesterone enhances the Gaba chloride ion channel receptor (31, 32) regardless of whether steroid receptors exists (30, 33) implying the action is non-genomic**.** Progesterone attenuates the effect of glutamate (31) This could diminish cortico-cortical transmission which is mostly dependent on a glutamate-induced initial excitatory postsynaptic potential (EPSP) exciting pyramidal neurons which receive transcallosal input. (38) (EPSP) are followed by a dual component inhibitory postsynaptic potential (IPSP) which probably involve the activation of GABA-ergic (GABAA and GABAB) interneurons. (44) This increase in left hemisphere performance can be better explained in terms of a RCH Gaba / LCH Glutamate model, with the reduction of more right hemisphere glutamate neurons than left.

41 "Glutamate treatment of the left hemisphere retards visual discrimination learning and auditory habituation, as does glutamate treatment of both hemispheres, but ***treatment of the right hemisphere is without effect on these behaviours.*** *An imbalance generated by administering glutamate to the left hemisphere* ***causes a marked increase in aggressive and sexual behaviour****, which does not occur either after treatment of both hemispheres or treatment of the right hemisphere. "*

This finding of no effect by glutamate treatment of the RCH is consistent with a left hemisphere working towards control of it's transcallosal counterpart. All the citied studied of lateralized chemistry used for these reviews, use various methods to determine a greater ratio for the hemispheres. If not agreed on the the dipole theory of reversal for each hemisphere, it should at least be apparent that a greater ratio of inhibitory systems Gaba and tyrosine derived G-protein modulators, such as Dopamine occupy the left hemisphere, (3) conversely a greater ratio of excitatory systems glutamate and Tryptophan derived g-protein modulators such as serotonin occupy the right hemisphere. (3)

Treating only the left hemisphere with glutamate results in responses which suggest a lack of inhibition, because there is no transcallosal inhibition or pull pre-existing within the RCH.

5.2.4 ISOPRENOID PATHWAY AND GLUTAMATE

recent research in Indian neurology from Kerala (49,50,51,52) uses a chemical pattern approach to decipher brain systems. This approach has received some criticism for being unscientific. Is there a way in which using a whole systems approach can be scientific ? The idea of looking for lateral chemical reversals, should have began in the 1970's. Reading these papers from India finds the beginning of that process. It is an adjunct to motivate a fresh approach to otherwise quite everyday research which has been proliferated through many kinds of disorder to determine the role of hemisphere dominance in dozens of studies. Their proposal that entire brain systems are lateralized in LCH/RCH such as Tryptophan / Tyrosine derived neurotransmitters respectively, has been added to by research into cellular metabolism. In particular the Isoprenoid Pathways. The reasons for this have not been clarified. For laterality the results are impressive.

*49 "**quinolinic acid and serotonin being NMDA (N-Methyl D-Aspartate) agonist can contribute to NMDA excitotoxicity** reported in epilepsy.[19] In the presence of hypomagnesemia, the magnesium block on the NMDA receptor is removed leading to NMDA excitotoxicity.[20] The plasma membrane glutamate transporter (on the surface of the glial cell and presynaptic neuron) is coupled to a Na+ gradient which is disrupted by the inhibition of **membrane Na+-K+ ATPase**, resulting in decreased clearance of glutamate by presynaptic and glial uptake at the end of synaptic transmission.[20] By these mechanisms**, inhibition of neuronal membrane Na+-K+ ATPase can promote glutamatergic transmission and excitotoxicity** contributing to epileptogenesis"*

Here two of the tryptophan derived modulating co-transmitters quinolininc acid and serotonin are found to add greater excitement to glutamate transmission as well as elevated HMG CoA reductase activity which correlates with elevated digoxin levels inhibiting membrane Na+-K+ ATPase. (49) All three of these of these glutamate exciting biochemical factors have been found by these researchers to be lateralized in RCH. (48,50,51,52)

5.2.5 LEFTWARD LATERALIZATION IN SCHIZOPHRENIA

Schizophrenia is now considered to be a blockade of NMDA (81). The enzyme that degrades the NMDA receptor antagonist N-acetyl-alpha L-aspartyl-L-glutamate (NAAG), is in the hippocampus, prefrontal cortex, and temporal cortex of patients with schizophrenia. Less (GCP) II Results in more of NAAG the NMDA receptor antagonist. (82) In schizophrenia, there is leftward asymmetry in the temporal hippocampus regions (80).

Left brain asymmetry in Patients with schizophrenia was shifted to balanced left/right processing following treatment with an omega 3 eicosapentaenoic acid. (759c) Does this highlight lateralization of lipids as well ? The left cerebral hemisphere (LCH) contains more (FFA) free fatty acids than the right cerebral hemisphere (RCH), the difference being mainly accounted for by increased saturated and monoenoic fats. (755) reviewing papers on lipids could point to a LCH/RCH axis based round Omega 3/Omega 6. As the following table highlights.

	Left Cerebral hemisphere	Right Cerebral hemisphere
Omega 3 (DHA) modulates (35)	**Gaba (24,25,26)**	
Omega 3 reduces		**Prostaglandin E2 (Omega 6 metabolite) (84)**
GaBA / barbiturate reduces		**AA (85)(86)**
Glutamate LTP modulated by		**Omega 6 (AA) (87,83,88)**

SUMMARY

Lateral reverse symmetry appears to exist within glutamate / Gaba system, with Gaba being clearly defined by left brain receptors and glutamate indirectly from multiple RCH modulations. This reflects very similarily what happened when looking at the Dopamine / Serotonin system as LCH / RCH. (3) There was a greater amount of clearer research regarding where dopamine receptors were within the left hemisphere, and a greater amount of indirect effects of Serotonin within the right. I propose that this itself reflects the very nature of digital / analogue systems.

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