

## **The Center For Modeling Optimal Outcomes® LLC**

*“The Think Tank for Creativity & Innovation”®*

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### **Model for Homeostasis “Brief” on Alzheimer’s disease**

While working on research into the application of neuroscience in business, The Center for Modeling Optimal Outcomes®, LLC discovered a scientifically verifiable model for assessing homeostasis (balance) between substances in the body.

This Model for Homeostasis is grounded in the laws of physics and chemistry that dictate all matter is comprised of positive, negative and neutral electrical charges (*in physics these “neutral” substances are neutrons and in chemistry they are transitional elements*). Given the fact that the body is comprised of matter and “powered” by a minute amount of electricity, all substances in the body must be positive, negative or neutral.

In conjunction with today's medical research, applying the Model for Homeostasis allows for the identification of corollary relationships of substances in the body as well as the consequences (i.e. illnesses and disease states) when they are disrupted (imbalanced).

Given the fact that the scientific community has not utilized homeostatic modeling to assess causal paths for illnesses and diseases, scientific literature does not make direct reference to the dynamics of disruptions of homeostasis in the body as contributing to illnesses and disease entities. Accordingly, in order to provide verifiable information based on valid scientific studies, the Life Sciences Group of The Center has researched and correlated studies from numerous disciplines that support the information contained in this “brief.”

The purpose of this “brief” is to enable the scientific community to link the highly probable variables that contribute to the “perfect storm” that results in Alzheimer’s disease (AD). Specific details of the many disruptions (imbalances) that occur simultaneously to form a “perfect storm” scenario that leads to a disease state are not conducive to adequate explanation in a document of this nature. We have, however, provided several links to information in order for members of the scientific community to validate the universal applicability of the Model for Homeostasis.

The specific dynamics associated with homeostasis of the substances in the body are in a tutorial provided by The Center on its web site [www.TheCenterNJ.com](http://www.TheCenterNJ.com) --- [Model for Homeostasis](#). *This tutorial is designed to help explain the scientific underpinnings of the model and how they were applied to identify highly probable variables that play a key role in the formation of Alzheimer’s disease (AD). Further explanation is suggested and available from a member of the Life Sciences Group.*

Based on the application of the models created by the Life Sciences Group of The Center for Modeling Optimal Outcomes®, LLC, as outlined herein, it now obvious that past scientific studies were based on information that has changed as new technologies have

been developed and utilized. Several examples of the new information that necessitates a complete review of AD, its symptoms and possible causes are included herein.

The number of references provided in this document has been limited solely for the purpose of brevity. In nearly all cases, Internet search will provide substantial additional documentation.

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When the Life Sciences Group of The Center decided to investigate possible causes of AD, they identified that scientific studies already indicated various minerals are involved in neurodegenerative diseases.<sup>1</sup> Using this information and the processes identified in the Model for Homeostasis, they researched scientific literature to identify the existence of a modulated pair (refer to the tutorial for an explanation) of minerals that, if disrupted, could lead to AD. This process led the group to the possibility that low levels of iron could trigger a variety of downstream consequences similar to those of AD. Further preliminary research identified a corollary relationship between iron-aluminum as a possible base pair of minerals already linked to AD as well as the probability that (in terms of homeostasis) phosphorus is the “modulator” to maintain homeostasis.

Realizing that the scientific community had previously identified excessive aluminum as a possible cause of AD in the 1990s, the Life Sciences Group of The Center decided to proceed cautiously since substantial research had been conducted to dispel that hypothesis.

Knowing that the dynamics associated with the consequences of disruption between homeostatic relationships within the body were not considered during earlier research, the group decided to re-visit previous studies and assumptions from 15+ years ago and then compare that information to current scientific literature. This exercise was necessary to determine if adequate scientific support based on new technologies and recent studies existed that could warrant new paths in neuroscientific research for a highly probable cause of AD.

As an outcome of that exercise, the group was initially able to identify how aluminum could enter the brain as well as how increased aluminum and reduced iron might result in neurological disruptions.<sup>2 3</sup>

As one example of their findings, current scientific literature provided the answer for how aluminum could enter the brain; i.e. aluminum binds to transferrin.<sup>4</sup> Transferrin (a protein that contains two iron atoms) enables the transport of iron through the blood brain barrier.<sup>5</sup>

Realizing that numerous other variables existed beyond those attributable solely to disruption of homeostatic relationships in the brain as a result of excessive minerals,

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<sup>1</sup> <http://www.ebmonline.org/cgi/content/full/231/9/1481>

<sup>2</sup> <http://jn.nutrition.org/cgi/content/full/133/5/1468S>

<sup>3</sup> <http://www.ncbi.nlm.nih.gov/pubmed/18723004>

<sup>4</sup> <http://www.emedicine.com/med/byname/Toxicity--Aluminum.htm>

<sup>5</sup> <http://www.sciencedaily.com/releases/2005/02/050213135505.htm>

efforts were expanded to encompass all possible disruptions that could contribute to the causal path of the disease and its many physiological outcomes.

During its investigative process, the group found links between aluminum and iron in combination with phosphorus and their impact in the brain and the body. This information includes but is not limited to the following:

- Since increased aluminum would decrease iron, based on this scenario, AD patients would have to have increased incidences of anemia. When they investigated possible links between AD and incidences of anemia, their suspicions were confirmed; excessive aluminum is linked to anemia.<sup>6 7 8 9</sup>
- Iron and aluminum has been linked to the neurofibrillary tangles of Alzheimer's disease.<sup>10</sup>
- Aluminum overload in the body's extra-cellular matter (ECM) has been linked to the possibility of chronic fatigue syndrome.<sup>11</sup> Information pertaining to the ECM is provided later in this document.
- A recent study confirmed the probable involvement of phosphorus in AD.<sup>12</sup>

Based on findings relative to other neurodegenerative diseases, disruption of enzymatic signaling related to minerals is only one of the factors that constitute the "perfect storm" scenario that results in the disease state. Accordingly, to provide the research community with a solid foundation of variables that must be analyzed in order to rectify prior misconceptions and to address dementia as a dimension of disruptions in the brain, each with different causal paths, the group pursued numerous variables that can result in symptoms typically attributed to AD. This task was undertaken in order to segregate possible disease states in order for research to address separate treatments and possible cures for each one. Based on these efforts, the following are a sampling of the variables identified that must be addressed through integrative scientific research relative to AD and the various dementias.

-Minerals in the body are part of a cascade of corollary relationships.<sup>13</sup> The consequences of downstream disruptions that involve aluminum, iron, and phosphorus must be researched; especially those that may disrupt the homeostatic relationships that regulate the various iterations of superoxide dismutase.

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<sup>6</sup> <http://www.emedicine.com/med/byname/Toxicity--Aluminum.htm>

<sup>7</sup> <http://www.ncbi.nlm.nih.gov/pubmed/9141646>

<sup>8</sup> [http://www.anemia.org/pdf/slr\\_Geriatrics.pdf](http://www.anemia.org/pdf/slr_Geriatrics.pdf)

<sup>9</sup> <http://www.cababstractsplus.org/abstracts/Abstract.aspx?AcNo=19971406964>

<sup>10</sup> <http://www3.interscience.wiley.com/journal/109679546/abstract>

<sup>11</sup> <http://www.sciencedaily.com/releases/2008/11/081118141856.htm>

<sup>12</sup> Contact the Life Sciences Group of The Center to obtain a copy of the supporting documentation

<sup>13</sup> The Life Science group of The Center has a comprehensive model for the correlation of metalloenzymes and metalloproteins it will share with members of the neuroscientific community as part of partnership agreements. Reference to these models is not included in this document due to their complexity and problems associated with terminology between the numerous scientific disciplines associated with the interpretation of the information.

-The half-life of aluminum in body's ECM (estimated at 7 years) must be considered because of its probable cumulative effect.

-Introduction of aluminum into the body's ECM can occur as an adjuvant in vaccines, absorbed through the application of certain deodorants and antiperspirants, in medications such as antacids and enteric-coated aspirins, etc.

-Other edible products (foods or drinks) contain aluminum and phosphorus. They must be identified and considered as contributing to the accumulation of these minerals in the body's ECM.

-The impact of higher aluminum and lower iron on the synthesis of the neurohormones dopamine, norepinephrine and serotonin must be studied. Lower levels of these neurohormones have been identified in AD patients.

-Tau proteins include six isoforms that must be segregated into pairings of 3s to determine causes of disruption that may lead to causal paths for diseases within the dimension of dementias.

-Studies indicate a correlation between transferrin and apolipoproteins. Research should be initiated to determine the role, if any, of this glycoprotein in the disruption of homeostasis between the three apoEs.

### **Segregating dementia from Alzheimer's**

This "brief" is complicated somewhat by the fact that considerable confusion exists between AD and dementia; i.e. many individuals who are not well versed in neurodegenerative diseases often confuse these two entities as being the same. Adding to this confusion is the fact that, clinically speaking, it is currently difficult if not impossible to differentiate between them. Furthermore, the reality that dementia is a dimension of disorders as opposed to one entity is often overlooked.

Considerable research has focused on AD and dementia as being the same disease rather than the possibility that they may be two separate disruptions; each with separate causal paths that may impact a patient separately or concurrently. Based on current scientific literature, it now appears that AD is merely one of the many neurodegenerative disorders of the brain that results in the loss of proper cognitive functioning; i.e. one of several forms of dementia.

While this document is not intended to address all of the forms of dementia and their potential causes, The Center's web site provides information relative to two paths; i.e. corticosteroid induced as well as Chronic Traumatic Encephalopathy (CTE); i.e. the form linked to head trauma. [http://www.thecenternj.com/images/Dementia\\_Brief\\_1-11-10\\_Rev.pdf](http://www.thecenternj.com/images/Dementia_Brief_1-11-10_Rev.pdf)

In addition to the two aforementioned potential causes of dementia, considerable research has been devoted to the probability that amyloid beta plaque is a contributing factor in AD. Only recently, however, has research indicated that these proteins are actually

essential for brain function.<sup>14</sup> Accordingly, the link between these proteins and AD must be revisited. Most likely, the real issue may be one of the levels of these proteins reaching a tipping point whereby “the dose determines the poison” and the outcome is another disorder within the dimension of dementia.

### **Lipids linked to dementia**

Correlating the various studies between disciplines within medical research also enabled the Life Sciences group to identify the likely components of the metalloenzyme that signals the activity of amyloids. While, in The Center’s opinion, amyloid disruption is not directly attributable to aluminum – iron – phosphate driven AD, such disruptions appear to be linked to some dementias as well as to the prions in amyotrophic lateral sclerosis (ALS). A recent study supports the probability that the minerals identified as a “modulated pair” by the Life Sciences group are, in reality, Interleukin 6<sup>15</sup>. Based on that model, when the signaling molecule is in homeostasis, the brain is capable of clearing amyloid plaques.

Another critical point to be revisited is the link that has been established between apolipoprotein E and AD.<sup>16</sup> Careful analysis of apolipoproteins indicates there are three primary types; i.e. apoE2, 3 and 4.<sup>17</sup> Our modeling of homeostasis provides scientifically verifiable evidence that substances in the body are configured in modulated pairings of three.<sup>18</sup> Accordingly, there is a strong probability that apoE is not a factor in the cause of AD but that the disruption (imbalance) between the three forms of apolipoprotein is a likely cause of another one of the dementias; possibly the one associated with excessive amyloid beta as the outcome.

### **Applying another novel model to research**

During its years of interdisciplinary review of the many facets of medical research, The Center’s Life Sciences group identified several maxims; one of which is “If a mistake is made in the foundation of anything, all that is built upon it can be flawed. Then, the longer the deformity continues, the process of correcting the error becomes increasingly more difficult.”

Applying that maxim to its work, the group identified another model associated with the storage and transfer of nutrients in the body. This model, one for the body as a Mobius Loop, tracks the processing of foods, fluids and substances through the digestive process from chyme to chyle to interstitial fluid, lymph, marrow and blood.

While this model is not directly related to Alzheimer’s disease or any of the dementias, it is relevant because medical science has overlooked the fact that chyme (also known as chymus) consists of partially digested food that passes through the pyloric sphincter and into the duodenum where nutrients are extracted. The noteworthy aspect of this process is the fact that nutrients (e.g. lipids, minerals, amino acids, etc.) are stored in the body’s

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<sup>14</sup> <http://www.sciencedaily.com/releases/2009/11/091123114813.htm>

<sup>15</sup> <http://www.sciencedaily.com/releases/2009/10/091022114315.htm>

<sup>16</sup> <http://www.sciencedaily.com/releases/2009/12/091209121148.htm>

<sup>17</sup> [http://www.usatoday.com/news/health/2009-07-15-genetic-tests\\_N.htm](http://www.usatoday.com/news/health/2009-07-15-genetic-tests_N.htm)

<sup>18</sup> Refer to the Model for Homeostasis on The Center’s web site -

[http://www.thecenternj.com/images/Open\\_Source\\_Model\\_for\\_Homeostasis\\_Web\\_Site\\_Version\\_.ppt](http://www.thecenternj.com/images/Open_Source_Model_for_Homeostasis_Web_Site_Version_.ppt)

ECM (extra-cellular matter or interstitial fluid) awaiting use to synthesize proteins, peptides, hormones, enzymes and cells.

### **Nutrient transfer into the brain**

It is now obvious that past scientific studies were based on information that has changed as new technologies have been developed and utilized. An example of such new information addresses the dynamics of the blood brain barrier. Today, mainstream medical research has generally resigned itself to the belief that the blood brain barrier (BBB) is truly “a barrier” as opposed to a “filter.” Apparently, when the initial controversy regarding aluminum as a potential causal factor for AD was raised, the concept was dismissed because it was deemed impossible for aluminum to pass through the BBB. Only when current studies are carefully examined does it become obvious that lipids, minerals and other substances can pass through the BBB from the ECM where they are stored. Suddenly, the possibility and probability that aluminum is involved in AD becomes an issue that cannot be overlooked by medical science. At the same time, the transfer of lipids from the ECM becomes an issue that must be considered as a variable that can contribute to some of the forms of dementia.

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It should be noted that the Life Sciences group of The Center is committed to the preparation of highly probable causal path processes for review and consideration by the community of medical research scientists. The Center does not seek grants or funding for its modeling. While the numerous causal path models identified by The Center may appear to be nearly certain links to the causes of illnesses or diseases, verification of the outcomes based on the correlation of existing studies (several of which are contained in this document) must still reviewed by the scientific community.

While the information contained in this “brief” is provided in an open source format, in the interests of full disclosure, it should also be noted that pending patents have been filed for predictive tests associated with AD, disruptions attributable to enzymatic signaling for amyloid, variables that contribute to some forms of dementia as well as processes for the remediation of AD.

Parties interested in discussing relationships to the use of the IP relative to AD and dementias for development and commercialization should contact Linda Oliver Perrier at [loliverperrier@TheCenterNJ.com](mailto:loliverperrier@TheCenterNJ.com)