Release Notes xxxx-xx-xx : : not ready at all
The AD literature seems to go from miracle to failed miracle almost daily with little attempt to integrate new results with old observations. There are now a lot of data poitns to put together with cause and effect like any TV detective would do or as described in fables like the blind men and the elephant even though apparently Natalie Merchant set it to music. This work tries to fix that and finds that all these things are logically explained by well known issues such as age related digestive defects. Suggestions are further supported by in-house work on optimization of dog diets demonstrating apparent safety and utility of out of favor vitamins that could be impacted by changes in stomach chemistry. Another problem may be politics. One issue may be related to tyrosine metabolism and increased relative abundance of analogs capable of becoming toxic. This immediately jumps out as a contributor to race based AD differences not just social aspects that are touted as they are politically correct. To be sure, constituitive tyrosine metabolizers may have adaptive responses which support this flux with no net "bad" effects elsewhere and theere may turn out not to be any anwyay. In any case, its an important issue and like any other law of nature will not yield to human desire and the only way for people to benefit is follow the data. If you want to fool society, appease angry people, instead of solving problems and eliminating run on sentences for everyone turn back now to your safe space...
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I am not a veterinarian or a doctor or health care professional and this is not particular advice for any given situation. Read the disclaimers in the appendicies or text, take them seriously and take prudent steps to evaluate this information.
This work addresses a controversial topic and likely advances one or more viewspoints that are not well accepted in an attempt to resolve confusion. The reader is assumed familiar with the related literature and controversial issues and in any case should seek additional input from sources the reader trusts likely with differing opinions. For information and thought only not intended for any particular purpose. Caveat Emptor
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Alzheimer's Disease : 10k maniacs agree its an elephant

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(Dated: January 30, 2026)

Effective treatments for Alzheimer's Disease(AD) have been difficult to create. Much effort has been directed at amyloid beta removal with minimal clinical benefits demonstrated. Several alternative approaches have become popular including the infection hypothesis. Since so many pathogens appear to causes AD an age related vulnerability may be a better focus. Over various time spans, a series of headlines has emerged claiming benefits for various nutrients or supplements such as thiamine, choline, lithium, or NAD+. However, none appear to generate robust recovery. Taking these results and other observations together, similar to the fable of the blind men grasping different part of an elephant, a plausible unifying theme is nutrient deficiency due to age related digestive defects. Isolated profound nutrient deficiencies will not likely exist so obvious attribution of disease to single molecular entity is not a good starting point. Note that this goes beyond failure to absorb nutrients but also the creation of age related toxins, possbly methanol for example, that may need to be mitigated. Adaptive and malapative responses may make such an initial cause difficult to discern. This work motivates "meal engineering" as an intervention strategy that may be able to correct many of these problems. If this analysis accurately captures cause and effect in the real disease, correction or prevention may be fairly easy with well known small nutrient molecules or addition of acid or chloride to meals. Individual responses to nutrient details will appear to make each case different but still possibly treatable with similar simple remedies. Indeed, there has been

some limited success with dietary interventions but these fail to be designed around likely age related causes although they may coincidentally mitigate some of these problems. It may not be possible get all required nutrients from food once damage to digestive system has occured and supplements or other entities may be required. Cations such as metals, protein bound nutrients, amino acids, and lipophilic nutrients would likely be the first suspects. In particular, its likely that vitamin K and copper, along with more accepted components such as amino acids and SMVT substrates all need to be included to replace the younger absorption profile. However, brute force supplementation without regard to formulation may not work due to chemical and metabolic interactions. Two important amino acids with solubility issues in particular, tyrosine and tryptophan, and critical but reactive metals such as copper may require special attention. There are also possible political concerns if there is a hesitancy to link tyrosine metabolism to dementia. However, as always, social influences will not change the part to a best solution.

CONTENTS

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I. INTRODUCTION

Alzheimer's disease remains an unresolved problem despite all the money and effort directed at it for the last several decades. Despite decades of development, anti-amyloid products continue to show questionable net clinical benefiti [38] and do not always get FDA approval even with creative trial design [16] . This is in spite of the fact that as early as 2002 some had noted too many inconsistent observations of amyloid or tau to be serious targets [37] .

Others are using AI for precision medicine thinking that patient heterogeneity is a problem [48].

Thinking outloud
generally with such a narrow window there is either an important constraint or it is just chasing noise.

In response, some are proposing the use of emerging AI [18] which may yield results but often is limited by traning not on raw data but human commentary and a statistical rather than "model of reality" based view.

Some are searching for connections between apparently unrelated observations [21].

Headlines sometimes appear about new scientific or anecdotal evidence pointing to a new treatment but rarely do these translate into useful therapies. The thesis of this work is that many of these results are useful but tend to be interpretted in isolation and without regard for cause and effect in real disease. "Animal models" may mimic some features of real disease but are predicated on a cause unlikely to be relevant to the pathology being treated. By coincidence, sometimes however results may be transferable but don't seem to add to understanding.

In the search for alternatives to the amyloid hypotheses, a number of other ideas have been generated. A variety of metabolic ideas have been considered.

Genetics have been investigated too with APOE continuing to get most of the attention. This gene was identified as important for vitamin K transport to bones as early as 1996 with apoe4 noted for association with lower serum K and increased fracture risk [29] and it was noted response to supplementation occurs over months. However, a later work found "superior vitamin K status is associated with the apoE4 genotype in healthy older individuals from China and the UK" [49] . APOE is associated with many vitamins including vitamin K.

A 2025 article found ADAMTS2 to be one of the genes robustly increased in black and European Alzheimer's brains [44]. Essentially this translates into pervasive "weak structural proteins" as described below.

As no particularly good suspect has emerged, it may be worth lookin at all the data and trying to guess at age related vulnerabilities that cause more infections to lead to dementia and classic symptoms such as amyloid build ups. Nutritional defects make some sense as frailty, more than age per se, may be a correlate of age related diseases.

Works cataloging the utility of vitamins or derivatives for AD exist [45] but generally treat these things as "one at a time" approaches. Some mixes such as a mitrochondria nutrient mix [31] have been considered.

In a prior work [33] I interpreted a brain microbiome result to describe some host factors that would explain differences in the most and least abundant organisms in AD and healthy brains post mortem. While the existence of a brain microbiome is controversial due to the expected low absolute abudance and opportunity for contamination, the analysis is somewhat robust in that contamination from other parts of the patient should still reflect differential body nutrient levels. One argument against contamination was similarity to an unrelated work on uterine microbiome and possible synergistic role of these organisms but still a lot of coincidences can occur. This work generally pointed to deficiencies in lipophilic amino acids but also suggested additional methanol in the AD patients. One likely source for this is the GI tract due to changes in microbiome or initially changes in chemistry. Other toxins could associate with methanol production too and a good strategy may be to restore GI chemistry back to youthful state.

The GLP-1 data may also motivate an interest. GLP-1's apparently are causing non-specific nutrient deficiencies maybe similar to problems common in old age. Clinical trials showed either no benefit in AD or a positive trial [12] appeared to be cherry picked. Meanwhile anecdotes about an aged appearance (" Ozempic face") and brain fog persist. There are a lot of lessons here but the immediate thing of relevance is that nutrient limitation that is can be "plausibly denied" appears to generate some symptoms of aging and more to the point frailty.

Evidence pointing towards a cognitive benefit for GLP-1's may be a bit misleading. In one case, they were run against sulfonylureas which have known relevant side effects. In other cases, words suggesting a cognitive benefit in actuality mean that some risk factor has been reduced without clear evidence of real clinical benefit against dementia.

Women and blacks appear to be at higher risk of AD than European males. Factors unrelated to the disease per se may confound results but that is the case with even politically correct association studies.

One obvious issue with skin pigment is tryosine metabolism. Both the depltion of tyrosine and generation of toxic analogs could be considered. It is possible that constituitive skin pigmentation (CSP) evolved with adaptive mechanisms that may be instructive to investigate. While CSP may predispose to various maladies, it may also be easily treated if existing adpatations are not already active. Indeed, speculation on beneficial tyrosine analogs can not be dismissed.

Tyrosine anecdotes around menopause may be one indicator. While not considered essential as it can be derived from phenyalanine, it is possible that overall supply of the precursor and regulatory systems reduce tyrosine production

pathologically. This is most likely during times of "stress" and shortages. Interestingly in connection with GLP-1 there is a tyrosine dipeptide apparently acting as a signalling molecule Amino acids such as tyrosine may have limited availability in cell cultures and dipeptides have been explored [25] for many years now. Also of interest is the need to supplement tyrosine as a dipeptide to Chinese hamseter ovary cells in bioreactors [47] with work showing possible pickiness of cells for uptake although translation to human GI tract remains to be done. Similar problems have been noted for tyrosine usage in humans from either IV injection or parenteral feeding [32]. Tyrosine-tyrosine peptide is considered a hormone and may be responsible for anorexia in urea cycle disorder patients [36]. Indeed GLP-1 is often considered with PYY for understanding obesity signalling [10]. While its unclear if PYY can replace the GLP-1 drugs, if supplementation is attempted it will benefit a lot from recognizing the issues explored here. Dipeptides or reproduction of "best' GI chemistry may be more important than simply adding tyrosine to diet.

Copper has a lot of motivation behind it but its rich chemistry may make it one of the more difficult to study.

For whatever reason, vitamin K is often ignored even though interactions with APOE and appearance in "healthy" foods motivates at least an association with health.

Choline has also made recent headlines

adding to a long history for it [46] [8] and related pathways [17] including cholinesterase inhibitors [5].

Biotin and the SMVT substrates [42] have also come up in AD work and dementia more generally [43] [9] . Biotin is critical for many processes and can be obtained from digestion or intestinal microbe production [26].

Thiamine is also being considered [14] and that too is through to have acid sensitive absorption [30]. A 2012 study did show some benefits from supplementation in the elderly but due to poor absorption parental dosing was mentioned [40]. It seems that many studies on isolated nurients have come to similar conclusions but not attempted to think through cause and effect more generally. A 1988 study claimed to use a niacinamide placebo [6] which motivates another problem of an unintentionally active placebo. And in fact niacinamide has been discussed as a treatment too [28].

Lithium has recently been rediscovered [3] although positive review articles date back to at least 2012 [15] or so [35] and its not clear what happened over the intervening decade.

In 1971, it was observed that lithium could impact carbohydrate generally and inositol status in the brain [2]. In another manuscript considering inositol as a dog supplement (unpublished result)

but as of 2005 it could not be determined if lithium or inositol was more directly causal to brain changes that matter in the clinic [20]. This is likely a recurring issue in Alzheimer's and medicine overall. Associations are difficult to map to an actionable cause and effect sequence that can be beneficially modified. It is the main theme of this work that the primary cause is well removed from these associations and the elephant is not apparent from the sum of its parts. It is likely that both inositl and lithium are impacted by some common cause and simply supplementing one or the other or both can make some improvements but is a deadend to a cure.

An early proposal for vitamin K deficiency was described in 2000 citing "regulation of sulfotransferase activity and the activity of a growth factor/tyrosine kinase receptor (Gas 6/Axl) " [1].

In the last few years many works have explored vitamin K in old age conditions [13] [24] including association between vitamin K intake and cognition [7]. As bleeding or microbleeding is an issue in AD, its worth noting that 82 percent of "chronic stroke subjects" were vitamin K intake deficient and excessive vitamin E may also be a common problem [19]. The above work also describes more direct effects of vitamin K in AD including interactions with amyloid beta. (there are works that concentrate on emerging problems with vitamin E supplementation too, for example [27])

Thinking outloud

II. COMMON AGE-RELATED DIGESTION DEFECTS

To begin to make sense of the above and look towards the design of a better intervention, some knowledge of likely defects and resulting deficiencies needs to be established.

By 1992, common themes in impairment reports were emerging. One review suggested decreased taste and hypochlorhydria with decreased uptake of B-12 and minerals but increased absorption of lipids [41]. A small 1997 study of independently living people over 65 found about 90 percent had basal pH under 3.5 [23] although there is no indication of how this compares to the young or very old.

The pancreas and intestines may also be effected. One review mentions decreases in calcium, zinc, and magnesium but not copper in old rats [22]. However, the pancreas and intestines must work with whatever the stomach produces with the meal. Earlier, I considered that lack of an early low pH step may produce more idiosyncratic results as some mineral can irreversibly precipitate [34].

Around 2004, it was thought that reserve capacity was sufficient although some reduced absorption of vitamins such as A,D,K, and B6 but tended to minimize significance for treating aging [11]. One problem however is that even today sufficient intake and uptake may not be clear and the impact of multiple "low levels" could invoke adpative responses.

Today however protein deficiency appears to be reasonably well accepted as an issue with aging and its manifestation as sarcopenia [39]. What mey be less apparent is any changes in quality such as due to translational infidelity or lack of "other stuff." Sarcopenia or frailty appear to be highly correlated with vulnerability to several "age related" conditions [50].

Further, the impact of drugs common in the elderly on nutrient status is becoming recognized [4].

III. MEAL ENGINEERING CONSIDERATIONS

Based on the above, the goal then is to replace nutrients and remove toxics that are more common in old age. Return to a youthful GI tract may be a reasonable goal although it may be possible to do even better. Conceivably if nutrient uptake can overcome GI limitations some healing may be observed making possible a return to normal food for a while.

Nutrients need only be supplied over "relevant" time scales. This can vary a lot from nutrient to nutrient and allows better segregation and rotation than trying to get everything everyday. Incompatibilities include chemistry and competition for receptors or enzymes or transporters etc.

Not every meals needs to be the same and in fact currently with the dogs one meal contains most things while a second is largely reserved for copper.

IV. CANDIDATE INGREDIENTS

Candidates are drawn from prior thoughts on food and vitamin supplements along with the presumed GI disturbances common in old age.

Any foods or supplements reputed to improve some age related condition could be examined. In many cases the folklore appears to be a useful starting point as it may rationalize well with the above theory or be supported by in-house work here with dog feeding.

Fermented foods meet many of the criteria for obtaining nutrients with age impaired digestions. They tend to have low pH, and can even include pH as part of the definition, and essential molecules such as free amino acids and vitamin k.

Olive oil has been an important part of in-house dog diets (manuscript in preparation) as well as being a highlighted component of the Mediterranean Diet. A variety of possible causal pathways could be imagined for it but here it is considered somewhat uniquely, along with lecithin and phosphorous sources, as an absorption enhancer. Its interesting to note, although of unknown significance, that yeast with a higher amount of membrane oleic acid have higher ethanol tolerance [51]. As part of a meal formulation it may help with uptake of lipophilic nutrients.

2 related objectives are to improve chloride levels and reduce pH. In the results cited previously, acid came from citric acid and HCl supplement formulations. Formulations that use HCl as a raw component would require great care and may be avoided initially. Some soft drinks such as diet cola beverages contain phosphoric acid that may have some benefits. Chloride was largely from potassium chloride and again added HCl.

Title			
Ingredient	Amount	InCompatible	Remarks

TABLE I.

V. DEVELOPMENT PATHS

Since many candidate components are GRAS, its possible to outline informal or "crowd sourced" data from motivated parties such as caregivers. I have been using MUQED daily to record dog diets and relate to outcomes. This helped to identify olive oil as an improtant component although it may have a long response time if other sources of oleic acid such as chicken are included in the diet. The added workflow just requires a few minutes after each meal or dosing to enter whatever was given and have it checked for basic syntax. I use "vi" although more sophisticated or

voice entry may be easy to do. The current implementation of MUQED creates a small vocabulary of foods, supplements and outcomes as well as a simply syntax for entering amounts right after or even skeletons before consumption. As the current implementation is designed for just "a few" people or dogs, everything is done with text files locally although collation with a standard vocabulary could be done with better scaling implementations.

My in-house results with dogs does suggest some folk lore or anecdotes may have useful signals but will require more data to sort out and a MUQED-like approach may fill the void.

Even if this fails to mitigate disease, it may not be a frivolous effort especially if any trends are observed with prescription medicines.

VI. CONCLUSIONS

VII. SUPPLEMENTAL INFORMATION

VII.1. Computer Code

VIII. BIBLIOGRAPHY

[1] A.C. Allison. The possible role of vitamin k deficiency in the pathogenesis of alzheimer's disease and in augmenting brain damage associated with cardiovascular disease. *Medical Hypotheses*, 57(2):151–155, 2001. URL: https://www.sciencedirect.com/science/article/pii/S0306987701913076, doi:10.1054/mehy.2001.1307.

[2] J. H. ALLISON and M. A. STEWART. Reduced brain inositol in lithium-treated rats. *Nature New Biology*, 233, 10 1971. URL: https://www.nature.com/articles/newbio233267a0, doi:10.1038/newbio233267a0.

[3] Liviu Aron, Zhen Kai Ngian, Chenxi Qiu, Jaejoon Choi, Marianna Liang, Derek M. Drake, Sara E. Hamplova, Ella K. Lacey, Perle Roche, Monlan Yuan, Saba S. Hazaveh, Eunjung A. Lee, David A. Bennett, and Bruce A. Yankner. Lithium deficiency and the onset of alzheimer's disease. *Nature*, 645, 09 2025. URL: https://www.nature.com/articles/s41586-025-09335-x, doi:10.1038/s41586-025-09335-x.

[4] Victoria Bell, Ana Rodrigues, Maria Antoniadou, Marios Peponis, Theodoros Varzakas, and Tito Fernandes. An update on drug-nutrient interactions and dental decay in older adults. *Nutrients*, 15, 12 2023. doi:10.3390/nu15234900.

[5] Jacqueline S Birks. Cholinesterase inhibitors for alzheimer's disease. *Cochrane Database of Systematic Reviews*, 2016, 03 2016. doi:10.1002/14651858.cd005593.

[6] John P. Blass. Thiamine and alzheimer's disease. *Archives of Neurology*, 45, 08 1988. doi:10.1001/archneur.1988.00520320019008.

[7] Sarah L. Booth, M. Kyla Shea, Kathryn Barger, Sue E. Leurgans, Bryan D. James, Thomas M. Holland, Puja Agarwal, Xueyan Fu, Jifan Wang, Gregory Matuszek, and Julie A. Schneider. Association of vitamin k with cognitive decline and neuropathology in communitydwelling older persons. *Alzheimer's & Dementia: Translational Research & Clinical Interventions*, 8, 01 2022. doi:10.1002/trc2.12255.

[8] Elissavet Chartampila, Karim S Elayouby, Paige Leary, John J LaFrancois, David Alcantara-Gonzalez, Swati Jain, Kasey Gerencer, Justin J Botterill, Stephen D Ginsberg, and Helen E Scharfman. Choline supplementation in early life improves and low levels of choline can impair outcomes in a mouse model of alzheimer's disease. *eLife*, 12:RP89889, jun 2024. doi:10.7554/eLife.89889.

[9] Janelle L. Cooper. P3400: Biotin deficiency and abnormal pantothenic acid levels in dementia. *Alzheimer's & Dementia*, 4, 07 2008. doi:10.1016/j.jalz.2008.05.1970.

[10] Akila De Silva and Stephen R Bloom. Gut hormones and appetite control: A focus on pyy and glp-1 as therapeutic targets in obesity. *Gut and liver*, pages 10–20, 01 2012. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC3286726/, doi:10.5009/gnl.2012.6.1.10.

[11] Gerald W. Dryden and Stephen A. McClave. *Gastrointestinal Senescence and Digestive Diseases of the Elderly*, pages 569–581. Humana Press, Totowa, NJ, 2004. doi:10.1007/978-1-59259-391-0_25.

[12] Paul Edison, Grazia Daniela Femminella, Craig Ritchie, Joseph Nowell, Clive Holmes, Zuzana Walker, Basil Ridha, Sanara Raza, Nicholas R. Livingston, Eleni Frangou, Sharon Love, Gareth Williams, Robert Lawrence, Brady Mcfarlane, Hilary Archer, Elizabeth Coulthard, Benjamin R. Underwood, Paul Koranteng, Salman Karim, Carol Bannister, Robert Perneczky, Aparna Prasanna, Kehinde Junaid, Bernadette McGuinness, Ramin Nilforooshan, Ajay Macharouthu, Andrew Donaldson, Simon Thacker, Gregor Russell, Naghma Malik, Vandana Mate, Lucy Knight, Sajeev Kshemendran, Christian Holscher, Anita Mansouri, Mae Chester-Jones, Jane Holmes, Trisha Tan, Steve Williams, Azhaar Ashraf, David J. Brooks,

John Harrison, Rainer Hinz, George Tadros, Anthony Peter Passmore, and Clive Ballard. Liraglutide in mild to moderate alzheimer's disease: a phase 2b clinical trial. *Nature Medicine*, 12 2025. URL: https://www.nature.com/articles/s41591-025-04106-7, doi:10.1038/s41591-025-04106-7.

[13] Ebru Emekli-Alturfan and A. Ata Alturfan. The emerging relationship between vitamin k and neurodegenerative diseases: a review of current evidence. *Molecular Biology Reports*, 50(1):815–828, Jan 2023. doi:10.1007/s11033-022-07925-w.

[14] Jeffrey Fessel. Supplemental thiamine as a practical, potential way to prevent alzheimer's disease from commencing. *Alzheimer's & Dementia: Translational Research & Clinical Interventions*, 7, 01 2021. doi:10.1002/trc2.12199.

[15] Orestes V. Forlenza, Vanessa J. de Paula, Rodrigo Machado-Vieira, Breno S. Diniz, and Wagner F. Gattaz. Does lithium prevent alzheimer's disease? *Drugs & Aging*, 29(5):335–342, May 2012. doi:10.2165/11599180-000000000-00000.

[16] Orestes Vicente Forlenza and Breno José Alencar Pires Barbosa. What are the reasons for the repeated failures of clinical trials with anti-amyloid drugs for ad treatment? *Dementia & Neuropsychologia*, 19, 2025. doi:10.1590/1980-5764-dn-2025-e001.

[17] Ai Ling Fu, Qian Li, Zhao Hui Dong, Shi Jie Huang, Yu Xia Wang, and Man Ji Sun. Alternative therapy of alzheimer's disease via supplementation with choline acetyltransferase. *Neuroscience Letters*, 368(3):258–262, 2004. URL: https://www.sciencedirect.com/science/article/pii/S0304394004007153, doi:10.1016/j.neulet.2004.05.116.

[18] Cory C. Funk, Tom Paterson, Alex Bangs, David M. Cannon, George Savage, Eric Ringger, and Lee Hood. Mining the gaps: Deciphering alzheimer's biology through ai-driven reconciliation. *The Journal of Prevention of Alzheimer's Disease*, 13(1):100402, 2026. URL: https://www.sciencedirect.com/science/article/pii/S2274580725003449, doi:10.1016/j.tjpad.2025.100402.

[19] Lorenzo Grimaldi, Rosaria A. Cavallaro, Domenico De Angelis, Andrea Fuso, and Giulia Sancesario. Vitamin k properties in stroke and alzheimer's disease: A janus bifrons in protection and prevention. *Molecules*, 30, 03 2025. doi:10.3390/molecules30051027.

[20] A J Harwood. Lithium and bipolar mood disorder: the inositol-depletion hypothesis revisited. *Molecular Psychiatry*, 10, 01 2005. URL: https://www.nature.com/articles/4001618, doi:10.1038/sj.mp.4001618.

[21] Zaw Myo Hein, Barani Karikalan, Prarthana Kalerammana Gopalakrishna, Krina Dhevi, Aisyah Alkatiri, Farida Hussan, Mohamad Aris Mohd Moklas, Saravanan Jagadeesan, Muhammad Danial Che Ramli, Che Nasril Che Mohd Nassir, and Thirupathirao Vishnumukkala. Toward a unified framework in molecular neurobiology of alzheimer's disease: Revisiting the pathophysiological hypotheses. *Molecular Neurobiology*, 63(1):282, Dec 2025. doi:10.1007/s12035-025-05602-0.

[22] Peter R. Holt. Intestinal malabsorption in the elderly. *Digestive Diseases*, 25, 04 2007. doi:10.1159/000099479.

[23] Aryeh Hurwitz. Gastric acidity in older adults. *JAMA: The Journal of the American Medical Association*, 278, 08 1997. doi:10.1001/jama.1997.03550080069041.

[24] Julia Kamierczak-Baraska and Bolesaw T Karwowski. The protective role of vitamin k in aging and age-related diseases. *Nutrients*, 12 2024. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC11676630/, doi:10.3390/nu16244341.

[25] Sohye Kang, Johanna Mullen, Les P. Miranda, and Rohini Deshpande. Utilization of tyrosine and histidinecontaining dipeptides to enhance productivity and culture viability. *Biotechnology and Bioengineering*, 109, 09 2012. doi:10.1002/bit.24507.

[26] Chrysoula-Evangelia Karachaliou and Evangelia Livaniou. Biotin homeostasis and human disorders: Recent findings and perspectives. *International Journal of Molecular Sciences*, 25, 06 2024. doi:10.3390/ijms25126578.

[27] Alan D Kaye, Austin S Thomassen, Sydney A Mashaw, Ellie M MacDonald, Aubert Waguespack, Lily Hickey, Anushka Singh, Deniz Gungor, Anusha Kallurkar, Adam M Kaye, Sahar Shekoohi, and Giustino Varrassi. Vitamin e (upalpha-tocopherol): Emerging clinical role and adverse risks of supplementation in adults. *Cureus*, 02 2025. doi:10.7759/cureus.78679.

[28] Jule Klotter. Niacinamide (nicotinamide) and alzheimer's disease. *Townsend Letter*, 12 2010. URL: https://go.gale.com/ps/i.do?id=GALE%7CA243290423&sid=googleScholar&v=2.1&it=r&linkaccess=abs&issn=19405464&p=HRCA&sw=w&userGroupName=anon%7E7071297&aty=open-web-entry.

[29] Martin Kohlmeier, Andreas Salomon, Jörg Saupe, and Martin J. Shearer. Transport of vitamin k to bone in humans. *The Journal of Nutrition*, 126:1192S–1196S, 1996. URL: https://www.sciencedirect.com/science/article/pii/S0022316622017527, doi:10.1093/jn/126.suppl_4.1192S.

[30] Satoshi Kurisu and Hitoshi Fujiwara. Overestimation of aortic stenosis in thiamine deficiency: Two cases with distinct underlying conditions. *Cureus*, page e99487, 12 2025. URL: https://pubmed.ncbi.nlm.nih.gov/41552235/, doi:10.7759/cureus.99487.

[31] Jiankang Liu and Bruce N. Ames. Reducing mitochondrial decay with mitochondrial nutrients to delay and treat cognitive dysfunction, alzheimer's disease, and parkinson's disease. *Nutritional Neuroscience*, 8. doi:10.1080/10284150500047161.

[32] Timoth J. Maher, Paul J. Kiritsy, Fernando A. Moya-Huff, Franco Casacci, Franco De Marchi, and Richard J. Wurtman. Use of parenteral dipeptides to increase serum tyrosine levels and to enhance catecholamine-mediated neurotransmission. *Journal of Pharmaceutical Sciences*, 79(8):685–687, 1990. URL: https://www.sciencedirect.com/science/article/pii/S0022354915483078, doi:10.1002/jps.2600790807.

[33] Mike J Marchywka. Brain microbiome : Make your garden grow? feed your head. techreport MJM-2023-008, not institutionalized, independent, 44 Crosscreek Trail, Jasper GA 30143, 10 2023. URL: https://www.researchgate.net/publication/374946157_potuse-2023-10-24-v3, doi:10.5281/zenodo.10038912.

[34] Mike J Marchywka. Copper supplementation in dogs: Listen to her heart everybody's got a hungry heart. techreport MJM-2024-010, not institutionalized, independent, 157 Zachary Drive Talking Rock GA 30175 GA, 7 2025. URL: https://www.researchgate.net/publication/393240388_Copper_Supplementation_in_Dogs, doi:10.5281/zenodo.15785347.

[35] Shinji Matsunaga, Taro Kishi, Peter Annas, Hans Basun, Harald Hampel, and Nakao Iwata. Lithium as a treatment

for alzheimer's disease: A systematic review and meta-analysis. *Journal of Alzheimer's Disease*, 48, 09 2015. doi:10.3233/jad-150437.

[36] Sabrina Mitchell, Teresa Welch-Burke, Logan Dumitrescu, Jefferson P Lomenick, Deborah G Murdock, Dana C Crawford, and Marshall Summar. Peptide tyrosine tyrosine levels are increased in patients with urea cycle disorders. *Molecular genetics and metabolism*, pages 39–42, 02 2012. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC3336020/, doi:10.1016/j.ymgme.2012.02.011.

[37] Amritpal Mudher and Simon Lovestone. Alzheimer's disease - do tauists and baptists finally shake hands? *Trends in Neurosciences*, 25, 01 2002. doi:10.1016/s0166-2236(00)02031-2.

[38] Francesco Panza, Vittorio Dibello, Rodolfo Sardone, Roberta Zupo, Fabio Castellana, Ivana Leccisotti, Maria Claudia Moretti, Mario Altamura, Antonello Bellomo, Antonio Daniele, Vincenzo Solfrizzi, Emanuela Resta, and Madia Lozupone. Successes and failures: the latest advances in the clinical development of amyloid-upbeta-targeting monoclonal antibodies for treating alzheimer's disease. *Expert Opinion on Biological Therapy*, 25, 03 2025. doi:10.1080/14712598.2025.2463963.

[39] Lili Qiu, Qianqian Huang, Wenhao Li, Qian Zhang, Jun Zhou, Juan Chen, Yixuan Li, Ran Wang, Pengjie Wang, Siyuan Liu, Bing Fang, and Xiaoyu Wang. Aging influences protein digestion, absorption and amino acid metabolism. *Biogerontology*, 26(4):146, Jul 2025. doi:10.1007/s10522-025-10289-w.

[40] Khanh vinh quUx1ed1[bad char vv=7889]c Lu'o'ng and Lan Thi Hoàng NguyUx1ec5[bad char vv=7877]n. Role of thiamine in alzheimer's disease. *American Journal of Alzheimer's Disease & Other Dementias®*, 26, 12 2011. doi:10.1177/1533317511432736.

[41] RM Russell. Changes in gastrointestinal function attributed to aging. *The American Journal of Clinical Nutrition*, 55(6):1203S–1207S, 1992. URL: https://www.sciencedirect.com/science/article/pii/S0002916523317222, doi:10.1093/ajcn/55.6.1203S.

[42] Crystal Sang, Sasha A. Philbert, Danielle Hartland, Richard. D Unwin, Andrew W. Dowsey, Jingshu Xu, and Garth J. S. Cooper. Coenzyme a-dependent tricarboxylic acid cycle enzymes are decreased in alzheimer's disease consistent with cerebral pantothenate deficiency. *Frontiers in Aging Neuroscience*, 14, 06 2022. doi:10.3389/fnagi.2022.893159.

[43] Melissa Scholefield, Stephanie J. Church, Jingshu Xu, Stefano Patassini, and Garth J.S. Cooper. Localized pantothenic acid (vitamin b5) reductions present throughout the dementia with lewy bodies brain. *Journal of Parkinson's Disease*, 14, 07 2024. doi:10.3233/jpd-240075.

[44] Andrew F. Teich, Katarnut Tobunluepop, Juan C. Troncoso, ShihHsiu Wang, Zihan Wang, Benjamin Wolozin, Jesse Mez, Lindsay A. Farrer, Mark W. Logue, Adam Labadorf, Nicholas K. O'Neill, Dennis W. Dickson, Brittany N. Dugger, Margaret E. Flanagan, Matthew P. Frosch, Marla Gearing, LeeWay Jin, Julia Kofler, Richard Mayeux, Ann McKee, Carol A. Miller, Melissa E. Murray, Peter T. Nelson, Richard J. Perrin, Julie A. Schneider, and Thor D. Stein. Novel differentially expressed genes and multiple biological pathways for alzheimer's disease identified in brain tissue from african american donors. *Alzheimer's & Dementia*, 21, 10 2025. doi:10.1002/alz.70629.

[45] Tanmaykumar Varma, Pradnya Kamble, Madhavi Kumari, Vineet Diwakar, and Prabha Garg. *Vitamin-Based Derivatives for the Management of Alzheimer's Disease*, pages 317–344. Springer Nature Singapore, Singapore, 2023. doi:10.1007/978-981-99-6038-5_12.

[46] Ramon Velazquez, Eric Ferreira, Sara Knowles, Chaya Fux, Alexis Rodin, Wendy Winslow, and Salvatore Oddo. Lifelong choline supplementation ameliorates alzheimer's disease pathology and associated cognitive deficits by attenuating microglia activation. *Aging Cell*, 18, 12 2019. doi:10.1111/acel.13037.

[47] Natascha Verhagen, Andy Wiranata Wijaya, Attila Teleki, Muhammad Fadhlullah, Andreas Unsld, Martin Schilling, Christoph Heinrich, and Ralf Takors. Comparison of l-tyrosine containing dipeptides reveals maximum atp availability for l-prolyl-l-tyrosine in cho cells. *Engineering in life sciences*, pages 384–394, 06 2020. URL: https://pmc.ncbi.nlm.nih.gov/articles/PMC7481768/, doi:10.1002/elsc.202000017.

[48] Andrew E. Welchman and Zoe Kourtzi. Solving the 'goldilocks problem' in dementia clinical trials with multimodal ai. *The Journal of Prevention of Alzheimer's Disease*, 13(1):100397, 2026. URL: https://www.sciencedirect.com/science/article/pii/S2274580725003395, doi:10.1016/j.tjpad.2025.100397.

[49] Liya Yan, Bo. Zhou, Shailja Nigdikar, Xiaohong Wang, Janet Bennett, and Ann Prentice. Effect of apolipoprotein e genotype on vitamin k status in healthy older adults from china and the uk. *British Journal of Nutrition*, 94, 12 2005. doi:10.1079/bjn20051578.

[50] Lian Ye, Rui Liang, Xiaolei Liu, Jun Li, Jirong Yue, and Xinjun Zhang. Frailty and sarcopenia: A bibliometric analysis of their association and potential targets for intervention. *Ageing Research Reviews*, 92:102111, 2023. URL: https://www.sciencedirect.com/science/article/pii/S1568163723002702, doi:10.1016/j.arr.2023.102111.

[51] Kyung Man You, Claire-Lise Rosenfield, and Douglas C. Knipple. Ethanol tolerance in the yeast*saccharomyces cerevisiae*is dependent on cellular oleic acid content. *Applied and Environmental Microbiology*, 69, 03 2003. doi:10.1128/aem.69.3.1499-1503.2003.

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2. Free software including Linux, R, LaTex etc.

3. Thanks everyone who contributed incidental support.

Appendix A: Statement of Conflicts

No specific funding was used in this effort and there are no relationships with others that could create a conflict of interest. I would like to develop these ideas further and have obvious bias towards making them appear successful. Barbara Cade, the dog owner, has worked in the pet food industry but this does not likely create a conflict. We have no interest in the makers of any of the products named in this work.

Appendix B: About the Authors and Facility

This work was performed at a dog rescue run by Barbara Cade and housed in rural Georgia. The author of this report ,Mike Marchywka, has a background in electrical engineering and has done extensive research using free online literature sources. I hope to find additional people interested in critically examining the results and verify that they can be reproduced effectively to treat other dogs.

Appendix C: Symbols, Abbreviations and Colloquialisms

TERM definition and meaning

Appendix D: General caveats and disclaimer

This document was created in the hope it will be interesting to someone including me by providing information about some topic that may include personal experience or a literature review or description of a speculative theory or idea. There is no assurance that the content of this work will be useful for any paricular purpose.

All statements in this document were true to the best of my knowledge at the time they were made and every attempt is made to assure they are not misleading or confusing. However, information provided by others and observations that can be manipulated by unknown causes ("gaslighting") may be misleading. Any use of this information should be preceded by validation including replication where feasible. Errors may enter into the final work at every step from conception and research to final editing.

Documents labelled "NOTES" or "not public" contain substantial informal or speculative content that may be terse and poorly edited or even sarcastic or profane. Documents labelled as "public" have generally been edited to be more coherent but probably have not been reviewed or proof read.

Generally non-public documents are labelled as such to avoid confusion and embarassment and should be read with that understanding.

Appendix E: Citing this as a tech report or white paper

Note: This is mostly manually entered and not assured to be error free.

This is tech report MJM-2026-001.

Version	Date	Comments
0.01	2026-01-17	Create from empty.tex template
-	January 30, 2026	version 0.00 MJM-2026-001
1.0	20xx-xx-xx	First revision for distribution

Released versions,

build script needs to include empty releases.tex

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Supporting files. Note that some dates,sizes, and md5's will change as this is rebuilt.

This really needs to include the data analysis code but right now it is auto generated picking up things from prior build in many cases

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