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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 points 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, constitutive tyrosine metabolizers may have adaptive responses which support this flux with no net "bad" effects elsewhere and there may turn out not to be any anyway. 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... **This is a draft and has not been peer reviewed or completely proof read but released in some state where it seems worthwhile given time or other constraints. Typographical errors are quite likely particularly in manually entered numbers. This work may include output from software which has not been fully debugged. For information only, not for use for any particular purpose see fuller disclaimers in the text. Caveat Emptor.**

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Alzheimer's Disease : 10k maniacs agree its an elephant

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(Dated: January 28, 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 cause 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 parts 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, possibly methanol for example, that may need to be mitigated. Adaptive and maladaptive 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 occurred 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

I. Introduction	4
II. Meal Engineering Considerations	5
III. Candidate Ingredients	5
IV. Conclusions	6
V. Supplemental Information	6
V.1. Computer Code	6
VI. Bibliography	6
References	6
Acknowledgments	8
A. Statement of Conflicts	9
B. About the Authors and Facility	9
C. Symbols, Abbreviations and Colloquialisms	9
D. General caveats and disclaimer	9
E. Citing this as a tech report or white paper	9

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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 benefit [26] and do not always get FDA approval even with creative trial design [11]. 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 [25].

Others are using AI for precision medicine thinking that patient heterogeneity is a problem [34].

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 [13] which may yield results but often is limited by training 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 [15].

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 interpreted 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.

As no particularly good suspect has emerged, it may be worth looking 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 [31] but generally treat these things as "one at a time" approaches. Some mixes such as a mitochondria nutrient mix [20] have been considered.

In a prior work [22] 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 abundance 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 [8] 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 tyrosine metabolism. Both the depletion of tyrosine and generation of toxic analogs could be considered. It is possible that constitutive 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 adaptations 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 phenylalanine, 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 [16] for many years now. Also of interest is the need to supplement tyrosine as a dipeptide to Chinese hamster ovary cells in bioreactors [33] 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 [21]. Tyrosine-tyrosine peptide

is considered a hormone and may be responsible for anorexia in urea cycle disorder patients [24]. Indeed GLP-1 is often considered with PYY for understanding obesity signalling [7]. 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 [32] [5] and related pathways [12] including cholinesterase inhibitors [3].

Biotin and the SMVT substrates [28] have also come up in AD work and dementia more generally [29] [6] . Biotin is critical for many processes and can be obtained from digestion or intestinal microbe production [17].

Thiamine is also being considered [9] and that too is through to have acid sensitive absorption [19]. A 2012 study did show some benefits from supplementation in the elderly but due to poor absorption parental dosing was mentioned [27]. It seems that many studies on isolated nutrients 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 [4] which motivates another problem of an unintentionally active placebo. And in fact niacinamide has been discussed as a treatment too [18].

Lithium has recently been rediscovered [2] although positive review articles date back to at least 2012 [10] or so [23] 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 [1]. 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 [14]. 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 inositol 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.

Thinking outloud

A 2025 article found ADAMTS2 to be one of the genes robustly increased in black and European Alzheimer's brains [30]. Essentially this translates into pervasive "weak structural proteins" as described below.

APOE is associated with many vitamins including vitamin K.

II. 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.

III. CANDIDATE INGREDIENTS

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 [35].

IV. CONCLUSIONS

V. SUPPLEMENTAL INFORMATION

V.1. Computer Code

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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 particular 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.

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

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

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date={January 28, 2026} ,
startdate={2026-01-17} ,
day={28} ,
month={1} ,
year={2026} ,
author1email={marchywka@hotmail.com} ,
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author1id={orcid.org/0000-0001-9237-455X} ,
pages={ 11}
}

```

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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49514 Jan 27 14:25 elephant.log 5c3c4450472ceba4a0cc339f4c38ca35
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