

**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 the observations and try to put them together with cause and effect like any TV detective or old wisdom fables like the blind men and the elephant even as done by Natalie Merchant lol. This work tries to fix that and finds that all these things are logically explained by well known issues such as age related digestive decay. 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 become toxic. This immediately jumps out as a contributor to race based AD differences not just social aspects that are touted to be 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 instead of solve problems for everyone turn back now to your safe space...

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

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(Dated: January 19, 2026)

Effective treatments for Alzheimer's Disease have been difficult to create. Much effort has been directed at amyloid beta removal with minimal clinical benefits. Several alternative approaches have become popular including the infection hypothesis although many pathogens appear to be possible causes. An age related vulnerability rather than a small group of pathogens may be a better focus for attention. Recently, a series of headlines has emerged claiming benefits for various nutrients or supplements such as choline, lithium, or NAD<sup>+</sup>. Taking these and other observations together it is likely that, similar to the fable of the blind men grasping different part of an elephant, a plausible unifying theme is nutrient deficiency with adaptive and maladaptive responses making this initial cause difficult to discern. This work explains all of these disparate observations as the result of age related GI decay. 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. Isolated profound nutrient deficiencies will not likely exist so obvious attribution of disease to single molecular entity is not appropriate. 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. Cations such as metals, protein components, 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.

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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 and do not always get FDA approval even with creative trial design. 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.

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 [2] 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.

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.

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 [3] for many years now. Also of interest is the need to supplement tyrosine as a dipeptide to Chinese hamster ovary cells in bioreactors [7] 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 [4]. Tyrosine-tyrosine peptide is considered a hormone and may be responsible for anorexia in urea cycle disorder patients [5]. Indeed GLP-1 is often considered with PYY for understanding obesity signalling [1]. 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.

### Thinking aloud

A 2025 article found ADAMTS2 to be one of the genes robustly increased in black and European Alzheimer's brains [6]. Essentially this translates into pervasive "weak structural proteins" as described below.

APOE is associated with many vitamins including vitamin K.

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.

## II. CONCLUSIONS

## III. SUPPLEMENTAL INFORMATION

### III.1. Computer Code

#### IV. BIBLIOGRAPHY

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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

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

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