

Seeking the Cause of Dementia: An Analysis of Correlation Leading to Hypotheses for Causation and Treatment

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Abstract

The US Health and Human Services Secretary, RFK Junior, said in testimony to the House Labor-HHS Appropriations Subcommittee on 2026 March 14-15 in response to the heavily funded and fruitless amyloidal plaque focussed dementia research that:

"We should have the cure for Alzheimer's today. We don't have it purely because of corruption at NIH. And we are going to have it quickly."

So lets have a look at some of the evidence for the potential causes and potential cures for dementia and see if we can offer some alternative hypotheses for the NIH funders.

Keywords: Dementia, Alzheimer's disease, PFAS, microplastics, nanoplastics, microbial dementia hypothesis, vitamin D receptor suppression, type 2 diabetes, gut-brain axis, ischaemic stroke, correlation, causation, post mortem decomposition, RFK jr., NIH.

Method

We perform a meta analysis on dementia correlation. In other words we throw the kitchen sink at dementia.

Evidence

1. Nanoplastics: Campen et al [2] found from autopsies on brain tissue that the typical non demented brain in 2016 had 3.345µg/g of nanoplastics and in 2024 had 4.917µg/g of nanoplastics. Whereas dementia victims had 26.076 µg/g in 2024. He also showed that brain PFAS deposition occurs mainly in cerebrovascular cell walls and microglia. That is where the immunity battle would be occurring.

Fleshing his finding out a little: The average adult human brain weighs approximately 1,300 -1,400 grams (about 3 pounds), with a commonly cited mean of ~1,350 g for mixed-sex adults. This is based on large-scale autopsy data [21,22] bearing in mind that brains shrink with old age.

Median micro-nano plastic concentration [2] in non demented brains (2024 samples): $\sim 4.917 \mu\text{g/g}$ (micrograms per gram of brain tissue).

Brain mass: 1,350 g.

Plastic concentration: $4.917 \mu\text{g/g} = 5$ micrograms plastic per gram brain tissue.

Total plastic mass in brain:

$1,350 \text{ g} \times 4.917 \mu\text{g/g} = 6,750 \mu\text{g} = 6.638 \text{ mg} = 0.006638$ grams.

This does not seem to be too significant at first sight. But Campen found that the median length of the nanoplastic shards in the brain was 150 nm...

"We examined resuspended brain pellets by TEM, which revealed largely 100–200nm long shards or flakes" [2]

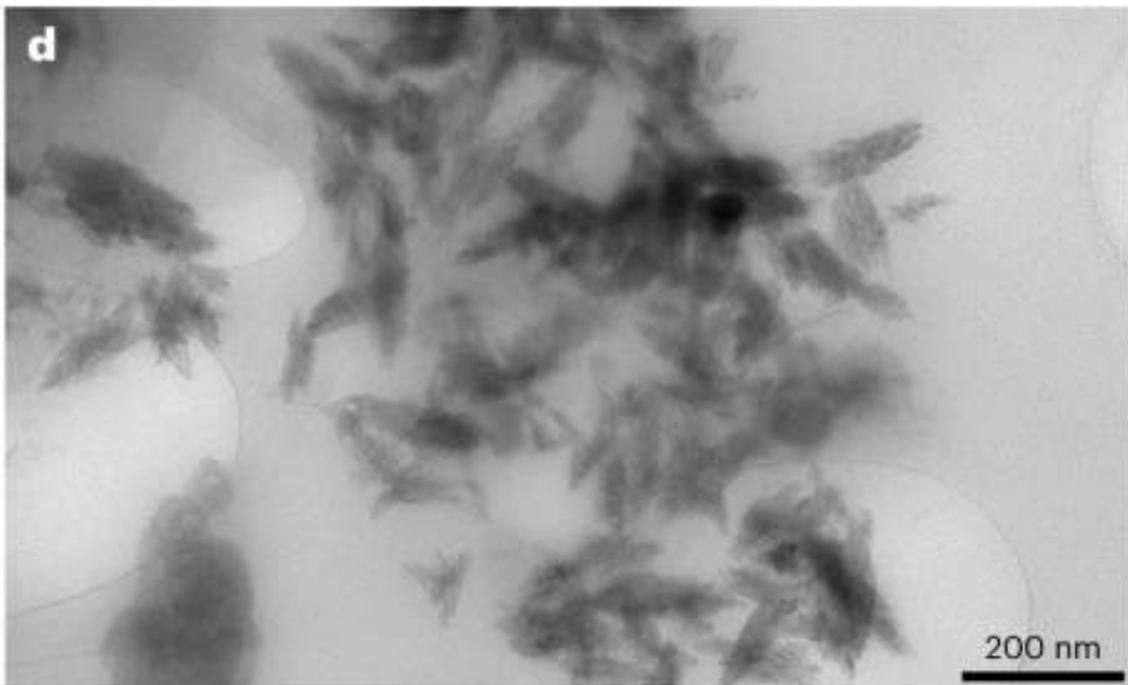


Fig1: TEM image of Nanoplastic shards from human brains.

If we look at his TEM image of those shards, we can see that they typically have an aspect ratio of between 2 and 4. So we take 3 as very approximate mean aspect ratio. The transparency of the shard shows them to be thin compared to their length and their width. They look very much like flat wings. So we take them to be $150\text{nm} \times 50 \text{ nm} \times 10 \text{ nm}$ on average, since that is their appearance as presented in the TEM image.

The plastics found were Polythene (75%) Polypropylene (15%) and the rest of the list below (10%).

Polyethylene (PE) — by far the dominant polymer (~majority of the load)

- Polypropylene (PP)

- Polyvinyl chloride (PVC)

- Polystyrene (PS)
- Polyethylene terephthalate (PET)
- Polymethyl methacrylate (PMMA - Perspex - Plexiglass)
- Polycarbonate (PC)
- Polyurethane (PU)
- Acrylonitrile butadiene styrene (ABS)
- Nylon-6 (N6)
- Nylon-66 (N66)
- Styrene-butadiene rubber (SBR)

These are all carbon based polymers. Silicon rubber (such as medical grade polysiloxane used in breast implants) was not tested for.

Typical densities of these plastics (solid, non-foamed forms) [2]:

Polyethylene (PE): 0.91–0.96 g/cm³ (low-density PE ~0.91–0.94; high-density PE ~0.94–0.96)

Polypropylene (PP): 0.90–0.91 g/cm³

Polystyrene (PS): 1.04–1.06 g/cm³

PVC: 1.3–1.45 g/cm³

The weighted average density (assuming ~75% PE at 0.93 g/cm³, 15% PP at 0.91, 10% others at ~1.05): ~0.93–0.95 g/cm³. We take it as 0.94 gm/cm³

So the total volume of nanoplastics in the human brain in 2024 was

$$0.94 \times 0.006638 \text{ grams} \times 0.94 \text{ gm/cc} = 0.00624 \text{ cc.}$$

Again this appears to be insignificant. But how many 150x50x10 nm shards would we need to make 0.00624 cc?

$$150 \text{ nm} \times 50 \text{ nm} \times 10 \text{ nm} = 75,000 \text{ nm}^3$$

There are 10 million nanometres in a centimetre

$$\text{So } 0.00624 \text{ cc} = 0.00624 \times 10^{21} \text{ nm}^3 = 6.24 \times 10^{18} \text{ nm}^3$$

$$6.24 \times 10^{18} \text{ nm}^3 / 75,000 \text{ nm}^3 = 6.24 / 7.5 \times 10^{14} = 83.2 \times 10^{12} \text{ nanoshards}$$

That is 83.2 trillion nanoshards in a *non demented* human brain in 2024. The approximate number in a demented brain would be 26.076/4.917 = 5.303x greater at 441.2 trillion. These are average values achieved through an accurate median value for length but visual approximations for width and thickness. It would be more conservative to say 20-100 trillion shards in non demented brains and 100-500 trillion shards in demented brains. What we do know accurately from Campen et al. is that terminally demented people have 5.3x more shards.

Regrettably, the average human brain only has around 86 billion neurons and around 85 billion glial cells making a total of 171 billion brain cells [24,25].

So the invasion rate found by Campen et al we visually approximate to be 487 shards per brain cell in the non demented and 2580 shards per brain cell in the demented. The accuracy of this approximation being perhaps $\pm 75\%$

The surface area of the 83.2 trillion nanoshards we approximate as:

$$\begin{aligned}
 & 83.2 \times 10^{12} \times (2 \times 150 \times 50 + 2 \times 150 \times 10 + 2 \times 50 \times 10) \text{ nm}^2 \\
 &= 83.2 \times 10^{12} \times (2 \times 7500 + 2 \times 1500 + 2 \times 500) \text{ nm}^2 \\
 &= 83.2 \times 10^{12} \times (15,000 + 3,000 + 1,000) \text{ nm}^2 \\
 &= 83.2 \times 10^{12} \times 19,000 \text{ nm}^2 \\
 &= 15.808 \times 10^{17} \text{ nm}^2 \\
 &= 1.5808 \times \text{m}^2
 \end{aligned}$$

Now we model the surface roughness of the shard. If we were to plaster equilateral tetrahedral pyramids all over the surface, then since these have 4 sides of equal area, the base is one quarter of their total surface area. So the non base part of the pyramid is 3x as large in area as the base. So these would increase the surface area of the plastic nanoshard by a further 3x. If the surface of the shard was a uniform sine wave of small wavelength compared to the shard, in one dimension only, just like a corrugated PVC roofing sheet, then the surface area would go up by a factor of $\pi/2$ or 1.57x. If it had uniform sine waves of small wavelength compared to its overall length in both surface dimensions then the surface area would go up by a factor of $\pi^2/4 = 2.47x$. But shards have all sorts of irregularities all over them as can be seen above. So the roughness factor will be nearer to the 3x pyramid factor than the 2.47x 2D sine wave factor.

Yet modern plastics do have a preferred direction. The shards demonstrate this by their high aspect ratio (by not being square). So we take the factor as half way between the 2D sine wave model and the Tetrahedron model, at 2.74x.

Then we must add the 3D effects. These include shards being bent around on themselves and having non straight line boundaries (on the shard scale) and holes and partial holes etc. All of these increase the surface area to volume ratio, by variations of the Swiss cheese effect and by twisting (the twisted helical meat skewer effect). The less planar the shards are and the less uniform in shape, the greater their surface area to volume ratio will be. The twisting and perforation and partial perforation would conservatively add a further multiplication factor of 1.4x we assert. The TEM image does not show much twisting but does show ravines and gaps. So this Swiss cheese effect will be dominant here. So the combined shape factor we put at

$$2.74 \times 1.4 = 3.84x \text{ compared to a smooth rectangular box.}$$

So that gives a total surface area of nanoplastics in a non demented human brain in 2024 as $1.58 \times 3.84 = 6.07 \text{ m}^2 = 65.4$ square feet, the size of a small bedroom and in demented brains it was $32.2 \text{ m}^2 = 346.6$ square feet, the size of a studio flat. These figures are a crude visual and mathematical approximation intended to provoke further serious consideration.

So we have gone from what appeared at first sight to be an insignificantly small amount of plastic debris in the brain, to the realisation that there is a huge surface area of indestructible (5-7 year half life) nanoplastic foreign material in a quantity of hundreds or thousands of shards per brain cell.

2. Type 2 diabetes is known [10] to increase dementia risk by 60%–80%. We propose that the increased blood sugar fuels pathogenic microbial overgrowth both in the gut and in the brain.

3. Vitamin D is known to be effective in preventing dementia. It is also known to be neuroprotective and antimicrobial. It upregulates cathelicidin and enhances phagocytosis. The large cohort analysis of UK Biobank data [11] by Ghahremani et al. (2023) showed that vitamin D supplementation was associated with 40% lower dementia incidence vs. no exposure. Yet, vitamin D is ineffective as a treatment for dementia. This points to a pathogenic cause for dementia which has the ability to suppress Vitamin D reception. A passive condition would not have this vitamin D sensitivity profile. But an active condition can have this profile, being more effective once the infection is entrenched.

Several pathogens downregulate vitamin D receptor (VDR) expression as an immune-evasion strategy, thereby limiting the antimicrobial and anti-inflammatory effects of vitamin D. This has been demonstrated for *Mycobacterium tuberculosis* [12,13], *Borrelia burgdorferi* (Lyme disease) [14], Epstein-Barr virus [15], and *Aspergillus* species [16], all of which suppress VDR in infected cells or tissues. A 2024 study in *Frontiers in Immunology* showed that gut fungi, particularly *Candida albicans*, induce VDR downregulation in intestinal epithelial cells, providing a mechanistic link between gut dysbiosis and impaired vitamin D signalling [17]. In Alzheimer's disease brains, VDR expression is significantly reduced in affected regions, including the hippocampus and prefrontal cortex, compared to age-matched controls [18]. These findings support the hypothesis that chronic microbial persistence in the brain may actively counteract vitamin D-mediated neuroprotection, rendering supplementation less effective once dementia is established.

4. Ischaemic strokes are known to increase dementia risk [1, 19]. Basically they double the risk.

5. The Bredesen ReCODE Protocol (2025)

Vitamin D: 5,000–10,000 IU/day (aim 50–80 ng/mL); used throughout (supports immune, anti-inflammatory)

Omega-3: 2,000–4,000 mg/day (high DHA preferred); key for membrane repair & anti-inflammation

Curcumin: 1,000–2,000 mg/day; anti-amyloid, anti-tau, anti-inflammatory

Lion's Mane Mushroom extract: Often included (500–3,000 mg/day); NGF stimulation, neurogenesis

MCT oil / ketones: 1–3 tbsp/day (C8 emphasis); ketones + antifungal (C8 disrupts *Candida* membranes)

Anti-inflammatory stack: Curcumin, resveratrol, quercetin, boswellia, ginger, omega-3, vitamin D

Other key interventions: Sleep optimization, exercise, stress reduction, sauna, detoxification (heavy metals, mould), oral health, hormones, gut repair

6. **Heart attacks:** Myocardial infarction increases the hazard ratio to 1.35% for vascular dementia.

7. **Cancer** decreases the chance of developing dementia. Cancer survivors have roughly 8–25% lower risk of all-cause dementia, Alzheimer's, or vascular dementia compared to those without cancer history. Smoking related cancers offer even better protection against dementia (possibly because the lungs and brain are in the same vascular system).

8. **Home remedies** which get the highest review scores from (families of) dementia sufferers according to Grok in order of merit are:

1. Vitamin D supplementation
2. Omega 3 fish oil supplementation.
3. Curcumin
4. Lion's mane mushroom/extract

9. After having written the umpteenth redraft of this article: I found myself in a position to ask Grok the right question:

"Is there a correlation between immunocompromised people and dementia?"

The microbial hypothesis would predict a positive correlation. Here is his answer...

"Yes, there is a positive **correlation** between being immunocompromised and an increased risk of dementia, though the relationship is complex and depends on the cause and severity of the immunosuppression.

Key Evidence

A. **HIV/AIDS** and HIV-Associated Neurocognitive Disorder (HAND).

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Untreated or advanced HIV (with low CD4 counts) is strongly linked to dementia-like symptoms (HIV-associated dementia or HAND). Even with modern antiretroviral therapy, milder cognitive impairment remains more common in people with HIV than in the general population. Severe immunosuppression allows direct viral effects on the brain and opportunistic infections [26,27,28,29,30,31,32,33,34].

B. **Organ transplant recipients on immunosuppressants**

Mixed findings. Some studies show **higher** dementia risk in kidney transplant patients (especially older recipients with diabetes or long dialysis history). However, certain immunosuppressants (calcineurin inhibitors like tacrolimus)

have shown **protective** effects against Alzheimer's disease in some cohorts, possibly due to anti-amyloid or neuroprotective properties. Overall, chronic immunosuppression appears to increase risk in many cases, particularly for vascular contributions to dementia [35,36,37,38].

C. Severe or recurrent infections and immune dysregulation:

Hospital-treated infections (a marker of immune vulnerability) are associated with a 1.5–2-fold increased risk of subsequent dementia. This holds even after adjusting for comorbidities. Repeated infections or sepsis appear to accelerate cognitive decline [39,40,41].

D. Immune-mediated diseases

Conditions involving chronic immune activation or dysregulation (e.g., rheumatoid arthritis, inflammatory bowel disease) are linked to modestly higher dementia risk in large UK Biobank analyses [42,43,44].

E. General infections and dementia

Severe infections (pneumonia, urinary tract infections, sepsis) are consistently associated with higher long-term dementia risk, independent of other factors. This supports the idea that periods of significant immune compromise can accelerate brain ageing or unmask underlying vulnerability [45,46,47,48]."

So the microbial hypothesis for Dementia correctly predicted the positive nature of correlations detailed above by Grok. When the immune system is fighting another invasive infection, it cannot fight dementia so effectively, is the finding.

That fits with the microbial hypothesis. It suggests that Dementia is an invasive infection actually. The comparison with cancer is stunning. Cancer assists the immune system in fighting dementia whereas sepsis dilutes the immune response to dementia is how we would interpret these correlations. In particular they indicate that cancer is very different from an invasive infection.

Logical Analysis of the Evidence

There are several fascinating features of the evidence above. Firstly the Vitamin D protection profile strongly suggests microbial/infectious causation or autoimmune attack on the brain. But the discontinuity once dementia is established suggests VDR suppression, which is known for certain microbes.

The next astonishing finding is that cancer correlates inversely with dementia — particularly among those who recover from cancer. People with a history of cancer generally show a lower subsequent risk of developing Alzheimer's or vascular dementia. This suggests that successfully fighting and surviving cancer may train the immune system in ways that also protect against dementia (rather like a vaccination). Immunity boosting is known to occur through Vitamin D: Higher levels are associated with reduced risk of both cancer and dementia. Together, these correlations paint cancer and dementia as related — both are

diseases of immunocompromised or immunocompromising states. In simple terms: If your immune system is strong enough to beat cancer, it is probably strong enough to ward off dementia as well.

The Omega 3 supplementation and the lion's mane neuron growth factor and choline acetyl transferase and acetylcholine and cholinergic capabilities are all useful in building back brain atrophy and offer hope that dementia patients can fight back if given the correct building materials for the reconstruction. The same should be true also of Alpha GPC, because it is also cholinergic.

The effectiveness of curcumin strongly indicates microbial attack as the cause of dementia, since curcumin is antibacterial and antifungal (and antioxidising).

Then we come to the role of the nanoplastics. Obviously putting 20-100 trillion nanoplastic shards with 5-7 year half lives into ones brain is a really dangerous result of modern living. And more investigation needs to be done to determine what damage they do.

Campen found that these shards congregate at the microglia where the immune battle is raging. This suggests that microbes are using them for defence. Microbes can be eaten by phagocytosis. Microplastics cannot. So if a fungus secretes a matrix and makes a biofilm out of some nanoshards, it becomes indigestible to white cells.

Bacteria and funghi are known build defences for themselves when an infection becomes established. Ideally (for the microbes) those defences should be impenetrable to the immune system. But that is precisely what 'forever' nanoplastics are. If a fungus or a bacteria was to make a fort out of them (or a tank) it would survive all immune assault. Bacteria are 500-5000 nm in length and fungi are larger at 2000 - 10,000 nm and yeast are 3000-8000 nm. So perhaps they would need several nanoshards for total protection or perhaps the white cell would spit them out with only one nanoshard? However you look at it, nanoshards may be a godsend to an opportunist pathogen seeking to build an unphagocytosible shield.

Now we consider the type2 correlation. Microbes love sugar (as does cancer). So this also fits with a microbial causation for dementia. High sugar also thickens the blood which restricts blood circulation which encourages microbial attack.

Then we consider the stoke evidence. If a part of the brain dies then it is a softer target for an invading microbe colony to form a base inside. Especially if the microbes involved are the same ones that eat the brain in the morgue after we die.

Possible causes for brain atrophy in dementia

1. Microbial ingestion
2. Auto immune self ingestion

3. Brain cell apoptosis due to some kind of toxicity.
4. Early autolysis

Microbial ingestion is the only one that fits all the evidence above. The other 3 fit some but not all. So the filter of correlation narrows the field down to one candidate as far as we can see.

Hypothesis

The type2 correlation, the VDR suppression, the nanoplastic correlation, the inverse cancer survival correlation (which is a positive immune strength correlation), the Bredesen DECODE protocol success, the positive review scores for building back brain tissue and attacking microbes and the stroke correlation and the immunocompromised correlation all point to microbial attack. But which microbes and how do we get to causation?

Well we already have post mortem causation from everybody who died and was left outside a fridge for too long before being buried or cremated. Brain decomposition starts with enzymatic breakdown of the brain (autolysis). But after a few days the microbes of death take over and eat the brain. We know which microbes eat the brain post mortem (see below). We therefore propose that what happens quickly post mortem in the absence of any immune defence also happens much more slowly pre mortem in the absence of a robust immune defence. This would be a pre mortem decomposition model in which opportunistic pathogens (fungi, bacteria, spirochetes) chronically consume neural tissue when immune clearance fails or is too slow.

We argue that the same microbial classes that decompose post mortem are decomposing pre mortem but at a slower rate in dementia. It is not as if the pathogens form an orderly queue outside the coroner's office waiting for a death certificate to be signed.

Indeed in stroke victims, they will be eating dead brain tissue in a living host.

Corpse eating pathogens liquify the brain in days or weeks when circulation stops. We propose that they 'liquify' it in years when circulation is impaired by strokes, type2 hyperglycosylation, or PFAS invasion and when the immune system is compromised by sepsis, by AIDS, by recurrent infections or vitamin D deficiency.

The microbes which eat the brain after death are present on and in the body before death. In non immunocompromised people they are ineffective. We propose that they are slightly and very slowly effective in immunocompromised people.

A strong indicator for the microbial hypothesis is the VDR suppression observed when dementia is established. There is a reason for the VDR suppression. We propose that this reason is to enable microbes to eat brain cells without too much interference from the immune system [12,13,14,15,16,17].

So here we are using a kind of meta analysis of known correlations to indicate causation. We already have the causation in the case of corpses. The leap we are making, powered by the filter of multiple correlation and the logic above, is that the causation established after death is also the causation before death in dementia. We do know that microbes can cause 100% brain atrophy after death (along with autolysis). We do *not* know whether they can cause the 20% or so cerebral atrophy seen in dementia in living patients with compromised immune systems of type2 diabetes or compromised cardio vascular systems. But we do know that they have that capability and that track record after death.

If you visit a dementia ward, we contend, that what you see, is people who are effectively experiencing a living brain death. Their brains are suffering post mortem type pathogenic decomposition (but without the autolysis) inside a living body.

The really worrying thing about this idea is that it is perfectly possible that all of us after 50/60 suffer from some degree of dementia. There may be an equilibrium between brain tissue being eaten and the brain fighting back by regrowing itself, if enough fish and cholinergic foods are being eaten. This is rather like the idea that cancer is always present but normally our immune system clears it before any noticeable damage is done.

Towards a cure

1. Eliminate plastics from proximity to food. Reverse Osmosis filter all drinking water for people over 50 as much as possible - particularly in hospitals and care homes.

2. Vitamin D receptor agonists could be crucial to dementia recovery, if they can overcome the VDR suppression.

3. Broad spectrum antibiotics delivered intravenously to avoid targeting the gut, and/or antifungals such as some of the polyenes and the azoles should slow Dementia progression significantly if the microbial hypothesis is correct. Further research needs to be done to try to attempt to identify the cerebral equivalent to Helicobacter Pylori.

4. Here therefore is the Grok-Ritchie Dementia Protocol:

1. 4000-8000IU per day of Vitamin D3 only before Dementia becomes serious then cease.

2. 15 grams of fish oil per day (75% of Eskimo level)

3. 10,000 mg of curcumin (95% extract) with piperine twice a week (shock the system repeatedly)

4. 1kg of fried lion's mane mushrooms (hericium erinaceus) per week.

5. Prebiotic oligosaccharides (galacto and fructo - inulin) 5 grams of each per day in water to lower blood sugar and fix the gut and improve immunity.

6. As much sunlight as possible. Do not stay indoors in the dark. If the VDR receptors are turned off, use the sun, it provides more than just D3.
7. No drinking from plastic bottles or cans which are all plastic lined.
8. No sugar, no white bread, no commercially processed food at all. No inflammatory fats, only cold pressed organic rapeseed oil (high in Omega3) and butter and a small amount of olive oil.
9. Only organic free range eggs no other type.
10. No food containing palm oil. No cheese except feta or cottage. No mouldy cheese (antifungal diet).
11. Plenty of fibre and spices and herbs.
12. Essentially a ketogenic antifungal prebiotic diet.
13. Prioritize turkey, organic free range chicken, lean lamb and fish for meat. No beef, no pork. Some veal if desired.
14. No pastries, cakes, biscuits, doughnuts, pretzels, chocolate (except the occasional 80%+ cacao dark chocolate). Do have potatoes, popadums, white chia seed (not black), well soaked washed and drained carb reduced rice and washed and drained carb reduced pulses.
15. Alpha-GPC supplementation for improving ChAT (Choline Acetyltransferase) and ACh (AcetylCholine) levels - 600 mg per day.
16. Almonds, Brazil nuts, Pumpkin seeds, 80%+ cacao dark chocolate, spinach, leafy greens, prunes, raisins, chickpeas (for essential brain minerals),

Mind you, I can tell you, with this or any other protocol: The difficulty is not only designing something that works. It is the hours and hours necessary to cajole demented people into eating it - at the start of the intervention!!

Further Research Recommendation

The microbes of early brain putrefaction are known to be:

Proteobacteria: Escherichia, Pseudomonas, Acinetobacter, Ochrobactrum, Cupriavidus, Agrobacterium — high in early PMI (first 24–48 hours) in brain samples [20,21].

Fungi: Candida, Aspergillus, Malassezia [4, 5, 20, 21]

So the suggested study would be to compare the early Post Mortem thanatobiomes of the brains of two corpses, one who died of dementia and the other who had no dementia on the assumption that the dementia causing microbes have a head start in the demented brain, and would multiply to the point of easy detection in the first hours after death.

Conclusion

Our hypothesis that Dementia is microbial ingestion of brain tissue fits all the correlations we have been able to identify. We point out that causation is known to be microbial in the case of corpses and we point out that a corpse is fully immunocompromised whereas demented people are statistically partly immunocompromised (by Type2, strokes, vitamin D levels, AIDS, and invite the reader to draw their own conclusions from that.

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