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## ALCOHOL HEALTH RISKS AND BENEFITS HEART DISEASE

The risks and benefits of alcohol consumption have been of interest for decades. The question of how much consumption is safe, if any is an epidemiologist's nightmare which results from the difficulty of acquiring valid consumption data by asking people questions since the natural tendency is to minimize what is admitted. Yet this is the only way to make the connection between amount consumed and the associated disease risks. It appears widely recognized that the collection of data results in underestimation of consumption. The frequently cited example is that in France comparison with national consumption figures suggested a five-fold under estimation.<sup>1</sup> Also a recent study from Korea has come under criticism for under estimation, also by a factor of five compared to World Health Organization data.<sup>2,3</sup>

One level of consumption that is probably least subject of distortion is abstaining. Abstainers are a special category for alcohol researchers because in many studies, moderate drinkers frequently have a significantly lower risk of the disorder at issue than abstainers.

Guidelines for maximum prudent intake from different countries also vary significantly as can be seen from the table below. Daily amounts are given in grams per day. A typical drink contains 10-15 grams of alcohol which depends on the type of drink from dry wines to liquors to the large variety of cocktails commonly consumed. Practitioners give advice by taking these guidelines seriously while ignoring underestimation. Recommended maximum intakes in several countries are presented in the table below. The amounts are in grams per day.<sup>4</sup>

COUNTRY	MEN	WOMEN
Australia	40	20
France	60	35
Italy	40	30
Sweden	47	38
UK	24-30	24
US	28	14

Obviously, the variation is large with a range of about a factor of two. These intakes presumably take into account various health issues but how each is weighted probably varies.

Let's look at the most recent studies.

### **THE CHINA STUDY**

A very large study recently reported.<sup>5</sup> It is prospective cohort study of 84,000 adult Chinese free from heart disease at baseline who were followed for 10 years. Self-reported alcohol consumption covered a wide range from 0 to >750 g/week (a typical bottle of wine at 14% by volume alcohol contains 84 grams of ethyl alcohol or 6 drinks of 14 g each). Alcohol consumption was stratified into 0, 1-25, 26-150, 151-350, and 351-750 and >750 g/week. Incident, CVD events, and mortality information were obtained from medical records.

Consistent with many studies, it was observed that abstainers had higher risks of cardiovascular events than non-abstainers. This is interpreted as alcohol being protective (lower risk than abstainers), and in fact this is seen frequently. The study reference for no risk was taken at 1-25 g/week. Twenty-five grams per week is equivalent to approximately one drink of 14 grams alcohol every 3 days. As consumption increased from the reference, risk slowly increased, but only for consumption of > 750 g/week did the risk as measured by hazard ratios corrected for confounding exceed the abstainer risk. This was true for the following endpoints: composite CVD mortality, CVD alone, and all-cause mortality. Intake > 750 g/week is equivalent to 107 g/ day which is about twice the highest risk thresholds given in the above table as guideline acceptable. In fact, not much risk increase was found even between 151-350 and 351-750 for any of the above listed endpoints.

These results contrast dramatically with commonly heard advice that optimum health requires abstaining, and one should not exceed moderate drinking by setting a limit of 2 drinks per day for women and 3-4 for men (4 ≈ 50-60 g/day). For heart disease and all-cause mortality endpoints presented in the paper the incident rates yield a range of absolute risk increases over 10 years of 0.2% to 0.9% for the increase of alcohol intake of 1-25 to 351-750 g/week.

These results which suggest a very liberal view of alcohol intake are based on only one study. However, the results are not that different than those found in the following studies concerning heart disease.

### **THE EPIC STUDY**

A large prospective study based on a European population (EPIC)<sup>6</sup> examined risk vs. g/day consumption and for both CVD and CHD found pronounced U-shaped curves. The minimums indicating that the highest protection for men were around 15 g/day for CHD and 30 for CVD, and for women 20-25 g/day. The curves never reflected risk greater than that found for 5 g/day with the curves for men going out to 60 and for

women to 30 g/day. The protection was thus pronounced. These results were corrected for confounding factors.

### **THE UK STUDY**

Another study examined the association between alcohol consumption and heart disease in a group of almost 2 million patients using UK databases.<sup>7</sup> The reference was moderate drinkers. Moderate consumption was defined as 3-4 drinks per day for men, 2-3 for women. Non-drinkers, former drinkers, and occasional drinkers had risks greater than the reference and heavy drinkers either had moderately enhanced risk or risk indistinguishable from the reference. Thus, the U-shaped or J-shaped curve. This was seen for various types of heart or atherosclerosis related disease, including angina, heart attack, sudden cardiac death, heart failure, as well as stroke and peripheral artery disease. The measures of risk were corrected for confounding and men and women had the same pattern but for women it was somewhat attenuated.

### **A US STUDY**

In a study with a follow-up of 8 years involved 333,000 participants free of heart disease. The reference was abstinence. Moderate alcohol intake at 30 g/day consistently yielded hazard ratios less than 1.0 (decreased risk vs. reference) and typically in the range of 0.7-0.8 indicating protection for all-cause and cardiovascular related mortality.<sup>8</sup> Heavy drinking was associated with increased all-cause mortality. The results were not stratified by gender. Half the participants were women.

Thus, four large studies from 2017 to 2021 all indicate heart disease protection for individuals free of heart disease at baseline when intake is moderate. Abstainers were at a somewhat elevated risk compared to moderate drinkers.

### **INDIVIDUALS WITH HEART DISEASE**

The studies discussed above all involved healthy individuals. A study has just been published in *BMC Medicine* which examined the effect of alcohol on individuals with cardiovascular disease defined as heart attack/angina together or stroke.<sup>9</sup> Data was from the Health Survey of England and the UK Biobank. The UK Biobank cohort was about 5 times larger than the Health Survey of England. A total of 48,400 men and women were included. Endpoints were all-cause mortality, cardiovascular mortality, and subsequent cardiovascular events. Intake levels were *low-level*,  $\leq 14$  units/week, *medium level*,  $>14$  to  $\leq 35$  units, and *high-level*,  $> 35$  units/week. A unit contained 8 g of alcohol which could be as little as a half a glass. Follow-up was for 9.5 years. The reference was abstinence and hazard ratios used to indicate risk increase or reduction. Outcomes were determined from national registries.

For all three endpoints a protective effect was seen in the intake dependence, either as U-shaped or a J-shaped response curves. The high-level drinkers were also protected even at an intake of  $> 56$  g/day. Almost all the HRs from which these results were derived were statistically significant. The dose response curves also suggest that abstainers were at higher risk compared to drinkers. Graphs of risk vs. consumption were consistent in always indicating protection (HR  $< 1.0$ ) up to 75-85 g/day, the limit of

the study. However, for cardiovascular events, the 95% confidence interval was a band above and below 1.0, i.e., not significant, starting at around 15 g/day. For the other endpoints, the upper confidence interval limits remained below 1.0 until 60-70 g/day.

There is bound to be considerable skepticism among the anti-alcohol experts that an amount of alcohol per day approaching that in a bottle of wine provides secondary prevention for patients with cardiovascular disease for major endpoints. However, the intake levels, self-reported not measured, may in fact represent underestimates.

### **BIOLOGICAL MECHANISMS**

The beneficial effects of alcohol discussed above have been attributed to elevation of HDL-C, lowering triglycerides, improving insulin sensitivity, decreasing inflammatory processes, decreasing platelet aggregation and blood clotting, and epigenetic effects.<sup>5</sup> HDL-C has actually been used as a surrogate maker for alcohol consumption. More than moderate consumption can result in levels of 89-90 mg/dL (2.1-2.3 mmol/L). A serum level of 60 is considered very good. There appears to be no risk of high HDL-C at 90 mg/dL.

### **CONCLUSION**

There are also other important considerations regarding alcohol consumption such as the risk of addiction, accidents, driving under the influence, domestic problems, and interference with normal activities and work. The context here is generally heavy consumption and alcoholism. There is also the issue of alcohol and cancer which is complex because of the variation of risk between cancer types and poor study design. The same protective effect seen for heart disease is observed with most cancer types. This will be discussed in a future issue of IHN.

The early suspicion that alcohol offers protection from heart disease now seems firmly established. In addition, the online calculators of the risks of serious heart disease events are neglecting an important factor. Wine consumption has been suggested as an explanation for the famous French Paradox. The range of intake that appears to not only be of no concern but protective is quite large which may be somewhat surprising. Even more so if study intakes are underestimated. Following the example of those who favor the Mediterranean diet and drink a glass or two of wine with meals may indeed be doing something heart healthy. The results are also consistent with the standard recommendation for prudent alcohol use, a maximum 3-4 drinks a day for men, and 2 for women if not at risk of becoming pregnant.

However, underestimation of intake in trials is serious. Even a factor of 2 or 3 puts the risk of alcohol intake in a completely different light.

## **IMPACT ON HUMAN HEALTH OF THE WEED KILLER GLYPHOSATE, THE ACTIVE INGREDIENT IN ROUNDUP**

Nearly 150,000 tons of this herbicide is spread on crops in the US every year. This is equivalent to one pound of glyphosate per person per year. The essential point is that this is the herbicide to which genetic modified plants grown for food are resistant. Thus, weed control is easily accomplished by aerial spraying. The so-called GMO crops produce food that is potentially contaminated with glyphosate used to keep down weeds.

However, the story gets much worse. Glyphosate is also used for pre-harvest desiccation, i.e., killing the foliage to make machine harvesting easier. The treatment also speeds up ripening or maturing, important in northern areas where the growing season is short. The goal is to force maturity before frost sets in. The practice is widespread and is a very efficient way to introduce glyphosate into the food supply. Crops eligible are of course not GMO. The result is that glyphosate has been allowed to contaminate a vast array of foods from both GMO and non-GMO crops. The former include corn, soybeans, potatoes, summer squash, canola, papaya, and sugar beets.

The widespread use of desiccation results in contaminated non-GMO crops such as barley, wheat, rye, rice, and oats. In oat breakfast cereals the contamination can be as high as several parts per million. These are mass produced agricultural products. They are the raw materials for industrially prepared foods, the staple of the Western diet. The extensive use of glyphosate as a desiccant has resulted in this herbicide turning up in a vast number of foods such as baked goods, pasta, pizza, breakfast cereals, soups and array of snacks, all consumed in large quantities.<sup>10</sup> A quick Google search will reveal the magnitude of this problem. Austria recently banned this use of Roundup and there appears to be growing concern among food companies that use oats. For example, all the various granola bars made by one company were found to contain glyphosates.

The increased use of glyphosate herbicides over the years is reflected in urinary excretion levels. In a study of older individuals from 1993 to 2016, urine levels progressively increased from 0.024 to 0.313 microg/L, a factor of 13.<sup>11</sup>

### **HEALTH ISSUES**

The foremost researcher in this field appears to be Stephanie Seneff, PhD, a senior research scientist at MIT's Computer Science and Artificial Intelligence Laboratory in Boston, Massachusetts. She has an international reputation with over 35 papers published in peer-reviewed journals that concern glyphosate issues relating human disease to toxic exposure. Her just published book *Toxic Legacy. How the Weedkiller Glyphosate is Destroying Our Health and the Environment* makes an important contribution to this field and the risk debate.<sup>12</sup> The official view is that there is no significant risk.

Dr. Seneff first discusses the evidence of harm ascribed to glyphosate in Chapter 1. Then in four chapters she covers in detail with documentation the health threats of

glyphosate in the context of **liver disease, reproduction and early development, neurological disorders, and autoimmunity**. To review this evidence is beyond the scope of this discussion. However, it seems clear that this is a health threat that should not be ignored.

In 2015, Seneff and Samsel published a comprehensive review of the case against glyphosate in the context of cancer.<sup>13</sup> They even were able to show that the rate of increase of six common cancers **exactly** matched the growth of agricultural use of glyphosate over the period 1990 to 2010. However, there is more. In 2015 the International Agency for Research on Cancer located in France (the cancer agency of WHO) classified glyphosate as a probable carcinogen for humans. However, this was disputed by Monsanto. In June 2020 the German chemical firm Bayer which bought the Monsanto Roundup business, announced a settlement to pay \$10 billion to settle over 100,000 lawsuits with plaintiffs who claimed they acquired cancer from using its products but Bayer admitted no wrongdoing or liability.<sup>12</sup>

One of the major issues in the glyphosate-cancer association involves non-Hodgkin lymphoma, a group of blood cancers that includes all types of lymphomas except Hodgkin lymphoma. Two meta-analyses (study which combines studies to improve the statistics) have been published recently concerning this link which come to opposite conclusions.<sup>14,15</sup> Selection of studies with the longest latency combined with the highest dose yielded the strongest cancer association.<sup>16</sup>

A just published study examined the association between urinary excretion of a glyphosate breakdown compound (AMPH) and the risk of breast cancer and found a positive result. When the highest vs. the lowest quintiles of AMPA excretion were compared, there was a 4.5-fold higher risk of developing breast cancer.<sup>17</sup>

Glyphosate's toxicity is related not only to its strong metal chelation ability, but to the fact that it can mimic the amino acid glycine and interfere with glycine-mediated processes, many of which are critical. Related is the strong interaction with molecules containing sulfur such as certain biologically important amino acids. Glyphosate is also a strong chelator and particularly effective in binding zinc, copper, manganese, magnesium, cobalt, and iron. Thus, this toxin can induce mineral deficiencies. In addition, glyphosate chelates aluminum. Two molecules wrap around an aluminum ion and shield its +3 charge, making it easier for example for aluminum to cross the blood brain barrier and enter the brain. The neurological toxicity of aluminum in the brain has been the subject of much controversy.<sup>18</sup> It is in many vaccines.

Thus, a very strong case can be made for avoiding ingestion or inhaling this remarkably diversified and prevalent toxin. However, this is difficult if not impossible. There are way too many foods, especially prepared foods that are contaminated mostly through the widespread use of desiccation. For example, a homemade beef barley soup might sound like a great idea but likely will be contaminated from the barley used. Eating only organic foods is of course one viable but challenging option but not even possible for many. Other than this, it appears that it is necessary to prevent the toxic action by

regular eating of certain foods and taking selected supplements that inactivate the toxic actions or promote the elimination of the toxin. The food and supplement approach are based on the known mechanisms whereby Glyphosate harms humans.

The chances are nil that physicians working with individuals presenting with any of the diseases described above as potentially glyphosate related will make this connection. Furthermore, the US Environmental Protection Agency (EPA) claims glyphosate poses no significant health threat based on current food contamination levels.

The US EPA also sets as an acceptable daily intake that “would not cause adverse effects throughout a lifetime” at 1.75 mg/kg body weight/day. The EU sets the limit more than 5-fold lower at 0.5 mg/kg/day. These are called *Acceptable Daily Intakes* (ADI). In fact, the acceptable daily limit is unknown.

Thus, it is of great interest to inquire as to the data upon which these exposure thresholds are based. For all but cynics, the answer may come as a shock. The data supporting the thresholds were supplied by the manufactures during the product registration process, are considered proprietary, and typically the evidence is not available for independent review.<sup>19</sup> Consider also a long paper just published concerning glyphosate food residues and dietary exposure where 3 of the 4 authors are employed by Bayer.<sup>20</sup> In addition, the 2016 EPA thresholds have remained unchanged. Finally, another twist to the story. Roundup contains not only glyphosate but several other chemicals that contribute to the toxicity of the herbicide preparation. This is rarely considered.

Dr. Seneff also draws attention to the history of the threshold for risk of lead toxicity, especially for children, which over the years as data accumulated, has decreased until reaching zero. In her view, zero should also apply to glyphosate.

For an excellent review of this subject which also nicely summarizes many of the issues Dr. Seneff addresses, here is the link to the free pdf.<sup>19</sup>  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4756530/>

## **TWO RECENT HUMAN STUDIES**

A just published study involving a US multi-site birth-cohort found a significant shortening of the gestation period in exposed women which correlated with the urinary levels of glyphosate and its breakdown product AMPA. Only spontaneous births were considered. The urinary levels corresponded to lower exposure than the EPA safe limits.<sup>21</sup>

Another recent study found in examining sperm samples from eleven individuals which were incubated with glyphosate that there was a negative effect on mitochondrial respiration efficiency which implied sperm dysfunction. This was consistent with prior studies that linked glyphosate exposure to reproductive toxicity, congenital disabilities and reduced sperm production.<sup>22</sup>

## **GLYPHOSATE AND ANTIMICROBIAL RESISTANCE**

Recently the growing presence of antimicrobial resistant bacteria has been found in the intertropical zone (near the equator, also called the doldrums by sailors). This is puzzling since human exposure to antibiotics in this region is minimal. The suggestion that this was due to farm use is not reasonable since antibiotics were not widely used in veterinarian practice. A hypothesis currently being advanced is that the culprit is glyphosate, which is both an herbicide and an antibiotic, the later property not widely appreciated.<sup>23</sup> In fact, Monsanto holds a patent on the antimicrobial activity of glyphosate against a wide range of bacteria, fungi and parasites. The pathway inhibited by glyphosate is a very common target for antibiotics and thus the herbicide/antibiotic can induce resistance to a number of antibiotics. A study of bacteria and fungi on an agar plate prepared with a gradient of glyphosate showed strong toxicity varied with the level of glyphosate.<sup>23</sup> Both GMO corn and rice are widely grown in the intertropical zone with heavy use of Roundup.

The authors point out that the emergence of resistance in bacteria and fungi in the world during the last 40 years correlates with the use of glyphosate.

## **FOODS AND SUPPLEMENTS THOUGHT TO COMBAT TOXIC EFFECTS OF GLYPHOSATE**

Dr. Seneff recommends an anti-glyphosate diet emphasizing organic produce while avoiding both ultra-prepared and merely prepared foods, and a diet that includes foods rich in sulfur containing constituents. This includes animal-based protein, seafood, grass-fed beef, fish, and eggs.<sup>12</sup> Among plants onions, leeks, garlic, broccoli, and cauliflower are recommended. Cruciferous vegetables contain sulforaphane, an unusual organic molecule that contains two sulfur atoms and can bind glyphosate. Broccoli and broccoli sprouts are rich in sulforaphane.

Sulforaphane is also available as a supplement. Amazon carries it. Other supplements recommended by Seneff are N-acetyl cysteine,  $\alpha$ -lipoic acid, methyl sulfonyl methane (MSM), liposomal glutathione, and garlic. Aside from sulforaphane, the supplements are all widely used.

This approach is mostly theoretical since human studies are virtually nonexistent. Effective supplement doses are unknown, but supplements at commonly used doses may be considered as an addition to a diet even with adequate sulfur containing foods.

Consider a glyphosate-heavy day of eating. Breakfast: oatmeal made from non-organic mass-produced commercial oatmeal and a big glass of bottled orange juice, another commonly contaminated product. Lunch: a large serving of beef barley soup with lots of barley plus wheat crackers. Snack: two granola bars. Dinner, pasta based main dish and thick crust piece of pie for desert (glyphosate source-- wheat). Bedtime snack: another granola bar.

## **CONCLUSION**

The principal motivation for pessimism concerning what appears to be a serious threat to health is the willingness to accept the official maximum residue limits for food and water which are subject to bias and Big Agriculture pressure and have a shocking history. Given that the food supply contamination appears under these limits, how does one explain the mountain of evidence of a link between glyphosate non-occupational exposure and adverse health results? The simple answer is the limits are significantly too high.

The health threat of glyphosate is not going to go away. Its use and the GMO crops and agricultural practices it services are well established. Thus, the only current options other than maximizing intake of organically grown food and trying to avoid foods known to be commonly contaminated, may be using antidotes such as selected foods and supplements to combat the ill effects.

## **COGNITIVE ACTIVITY AN INACTIVE BRAIN AND RISK OF DEMENTIA**

The phrase *Use it or lose it* commonly describes a view of the impact of mental activity helping to prevent age related brain function deterioration characterized by the path from normality to mild cognitive impairment to dementia, i.e., Alzheimer's disease. For some if not many, retirement or starting to take it easy can offer opportunities for continued or increased mental activity or provide an underlying contributing cause of what will eventually be diagnosed as age related dementia. The challenge is delaying this condition as long as possible, ideally up to the time of death. There are no effective treatments for AD, only symptom modifications. For delaying onset, one of the popular hypotheses is that cognitive activity enhances cognitive reserve thereby delaying the dementia related pathology. However, studies are lacking which adequately examine the efficacy of this intervention. In addition, effective exercise for the brain is much more complex to organize and carry out than physical exercise, which incidentally is also an important aspect of this problem. Someone who retires from an academic or technical or professional position which required constant solving of challenging problems may have trouble in replacing this activity. In fact, recognizing this should inspire planning for the retirement years.

A relevant study has just appeared online published in the journal *Neurology* which examines the success of cognitive activity to effectively exercise the mind with the endpoint of delaying dementia.<sup>24</sup>

A group of 1900 older persons without dementia at enrollment were evaluated for their frequency of participation in cognitively stimulating activities. Yearly evaluations for dementia extended over 22 years during which 25% developed dementia.

The validity of such a study depends on the assessment of cognitive activity from the start of the follow-up. Seven questions were used. (1) Daily reading time followed by

questions concerning the past year. (2) Library visits. (3) Frequency of reading newspapers. (4) Frequency of reading magazines. (5) Frequency of reading books. (6) Frequency of writing letters. (7) Frequency of playing games like checkers or other board games, cards, solving puzzles, etc. For frequency, the score progressed from 1 to 5 for once a year to almost every day. In scoring time spent, none = 1, less than an hour = 2. One to less than two = 3, two to less than three = 4, three to more than 4 = 5. These scores were averaged and became a continuous measure for all analyses. The authors point out that these cognitive activities involve information processing and there are few physical or social barriers to participation. Note the emphasis on the intensity of the activity. However, the activities obviously have inherent aspects of importance in the context of the goal. Reading novels is certainly very different from reading books concerning deep philosophical questions or complex technical or scientific matters.

Playing chess is vastly different than reading a newspaper. Both in fact can be done online and playing chess, GO or checkers against a computer offers a selectable level of intellectual challenge.

The results were very interesting. Persons with a high level of cognitive activity (90<sup>th</sup> percentile) had a mean age of onset of dementia of 94 years while those with a low level (10<sup>th</sup> percentile) had an age of 89. Thus, dementia attributable to brain exercise was delayed 5 years on average. No confounding by education, sex, cognitive activity prior to old age, prior mild cognitive impairment (MCI) or social engagement was found. The authors comment that a 5-year delay is much more significant than it appears. They cite a study showing that the reduced disease prevalence would be 41% and the costs 40%.

It might be asked, what else can one do? One possibility is to address the brain problem of so-called type 3 diabetes. It has been recognized for forty years that aging is accompanied by impaired brain energetics which is related to neurodegenerative diseases. Translated, this refers to the decline in the ability of brain cells to utilize glucose for fuel. Some call it the starving brain.<sup>25</sup> PET scans for brain metabolism provide convincing evidence. Obviously, an alternative fuel is needed. It exists since brain cells can use ketones. They cross the blood-brain barrier, and do not depend on insulin in order to enter brain cells to be metabolized. Nutritional ketones can be generated by a high-fat ketogenic diet or supplementing with 20-70 g/day of medium-chain triglycerides containing octanoate or decanoate (MCT oil from health food stores) or by readily available but expensive ketone esters (Amazon). The idea of solving a major problem in neurodegenerative disease of the brain with a can or bottle of oil from the local health food store apparently does not appeal to mainstream medicine. However, there is plenty of evidence including highly definitive PET scans revealing benefits in brain metabolism.<sup>26-28</sup> See IHN April 2020 for two articles on medium chain triglycerides and cognitive impairment. Also the book by Mary T. Newport, MD.<sup>29</sup>

## REFERENCES

1. Rey G, Boniol M, Jouglu E. Estimating the number of alcohol-attributable deaths: methodological issues and illustration with French data for 2006. *Addiction* 2010;105:1018-1029.
2. Yoo JE, Shin DW, Han K et al. Association of the Frequency and Quantity of Alcohol Consumption With Gastrointestinal Cancer. *JAMA Netw Open* 2021;4:e2120382.
3. Potter JD. Alcohol and Gastrointestinal Cancers in Korea-Risk, Inactive Genes, and Missing Alcohol. *JAMA Netw Open* 2021;4:e2120775.
4. Poikolainen K. *Perfect Drinking and its Enemies*. Second ed. 2020.
5. Zhang X, Liu Y, Li S et al. Alcohol consumption and risk of cardiovascular disease, cancer and mortality: a prospective cohort study. *Nutr J* 2021;20:13.
6. Ricci C, Wood A, Muller D et al. Alcohol intake in relation to non-fatal and fatal coronary heart disease and stroke: EPIC-CVD case-cohort study. *BMJ* 2018;361:k934.
7. Bell S, Daskalopoulou M, Rapsomaniki E et al. Association between clinically recorded alcohol consumption and initial presentation of 12 cardiovascular diseases: population based cohort study using linked health records. *BMJ* 2017;356:j909.
8. Xi B, Veeranki SP, Zhao M, Ma C, Yan Y, Mi J. Relationship of Alcohol Consumption to All-Cause, Cardiovascular, and Cancer-Related Mortality in U.S. Adults. *J Am Coll Cardiol* 2017;70:913-922.
9. Ding C, O'Neill D, Bell S, Stamatakis E, Britton A. Association of alcohol consumption with morbidity and mortality in patients with cardiovascular disease: original data and meta-analysis of 48,423 men and women. *BMC Med* 2021;19:167.
10. Xu J, Smith G, Smith S, Wang W, Li Y. Glyphosate contamination in grains and foods: An overview. *Food Control* 2019;106:106710.
11. Mills PJ, Kania-Korwel I, Fagan J, McEvoy LK, Laughlin GA, Barrett-Connor E. Excretion of the Herbicide Glyphosate in Older Adults Between 1993 and 2016. *JAMA* 2017;318:1610-1611.
12. Seneff S. *Toxic Legacy. How the Weed Killer Glyphosate is Destroying Our Health and the Environment*. London: Chelsea Green Publishing, 2021.
13. Samsel A, Seneff S. Glyphosate, pathways to modern diseases III: Manganese, neurological diseases, and associated pathologies. *Surg Neurol Int* 2015;6:45.
14. Zhang L, Rana I, Shaffer RM, Taioli E, Sheppard L. Exposure to glyphosate-based herbicides and risk for non-Hodgkin lymphoma: A meta-analysis and supporting evidence. *Mutat Res* 2019;781:186-206.
15. Donato F, Pira E, Ciocan C, Boffetta P. Exposure to glyphosate and risk of non-Hodgkin lymphoma and multiple myeloma: an updated meta-analysis. *Med Lav* 2020;111:63-73.
16. Kabat GC, Price WJ, Tarone RE. On recent meta-analyses of exposure to glyphosate and risk of non-Hodgkin's lymphoma in humans. *Cancer Causes Control* 2021;32:409-414.

17. Franke AA, Li X, Shvetsov YB, Lai JF. Pilot study on the urinary excretion of the glyphosate metabolite aminomethylphosphonic acid and breast cancer risk: The Multiethnic Cohort study. *Environ Pollut* 2021;277:116848.
18. Klotz K, Weistenhofer W, Neff F, Hartwig A, van TC, Drexler H. The Health Effects of Aluminum Exposure. *Dtsch Arztebl Int* 2017;114:653-659.
19. Myers JP, Antoniou MN, Blumberg B et al. Concerns over use of glyphosate-based herbicides and risks associated with exposures: a consensus statement. *Environ Health* 2016;15:19.
20. Vicini JL, Jensen PK, Young BM, Swarthout JT. Residues of glyphosate in food and dietary exposure. *Compr Rev Food Sci Food Saf* 2021.
21. Lesseur C, Pathak KV, Pirrotte P et al. Urinary glyphosate concentration in pregnant women in relation to length of gestation. *Environ Res* 2021;203:111811.
22. Cardona-Maya WD. Re: Herbicides Glyphosate and Glufosinate Ammonium Negatively Affect Human Sperm Mitochondria Respiration Efficiency. *Eur Urol* 2021.
23. Raoult D, Hadjadj L, Baron SA, Rolain JM. Role of glyphosate in the emergence of antimicrobial resistance in bacteria? *J Antimicrob Chemother* 2021;76:1655-1657.
24. Wilson RS, Wang T, Yu L, Grodstein F, Bennett DA, Boyle PA. Cognitive Activity and Onset Age of Incident Alzheimer Disease Dementia. *Neurology* 2021.
25. Newport M. *Alzheimer's Disease. What If There Was A Cure?* Laguna Beach, CA: Basic Health Publications, Inc, 2011.
26. Newport MT. *The Coconut Oil and Low-Carb solution For Alzheimer's Parkinson's and Other Diseases.* Laguna Beach, California: Basic Health Publications, 2015.
27. Cunnane SC, Courchesne-Loyer A, St-Pierre V et al. Can ketones compensate for deteriorating brain glucose uptake during aging? Implications for the risk and treatment of Alzheimer's disease. *Ann N Y Acad Sci* 2016;1367:12-20.
28. Croteau E, Castellano CA, Fortier M et al. A cross-sectional comparison of brain glucose and ketone metabolism in cognitively healthy older adults, mild cognitive impairment and early Alzheimer's disease. *Exp Gerontol* 2018;107:18-26.
29. Mary T.Newport. *The Complete Book of Ketones. A Practical Guide to Ketogenic Diets and Ketone Supplements.* Nashville, Tennessee: Turner Publishing Company, 2019.

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