

Glyphosate's Hidden Threat to Your Brain Health

Analysis by [Dr. Joseph Mercola](#)

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STORY AT-A-GLANCE

- › Glyphosate exposure is linked to increased neuroinflammation and Alzheimer's-like pathology in mice, suggesting a detrimental impact on brain health
- › Aminomethylphosphonic acid, a metabolite of glyphosate, persists in brain tissue long after exposure, indicating long-term effects
- › Over 80% of the U.S. population has detectable levels of glyphosate in their urine, highlighting widespread exposure
- › Fasting for less than 8 hours is associated with higher glyphosate levels, revealing dietary habits influence exposure
- › Children and adolescents demonstrate higher glyphosate levels compared to adults, raising significant concerns about potential long-term health impacts on younger populations

Alzheimer's disease is a progressive neurological disorder that deteriorates your memory, cognitive function and ability to perform everyday tasks. This debilitating condition affects millions of Americans, stripping individuals of their independence and profoundly altering lives.

Characterized by symptoms such as memory loss, confusion and changes in behavior, Alzheimer's disrupts both the lives of those diagnosed and their loved ones. As the disease advances, you may witness a loved one struggle with speaking, recognizing

familiar faces and completing simple activities, leading to a significant decline in their quality of life.

Are Herbicides in the Environment Driving Alzheimer's?

Over 80% of the U.S. population has detectable levels of glyphosate in their urine.¹ These startling statistics highlight the widespread exposure to glyphosate and its implications for neurological health.

The ubiquitous presence of glyphosate in our environment raises concerns about its long-term effects on brain health. With such high levels of exposure, understanding glyphosate's role in accelerating Alzheimer's disease pathogenesis becomes essential for developing strategies to protect public health and mitigate this devastating disease.

Conventional Alzheimer's treatments often focus on managing symptoms rather than addressing the root causes, which is frustrating for those seeking long-term solutions. Medications like cholinesterase inhibitors and memantine are commonly prescribed, but they come with side effects such as nausea, dizziness and confusion. These drawbacks highlight the need for a more comprehensive approach to managing Alzheimer's disease.

The underlying causes of Alzheimer's include genetic, environmental and lifestyle factors. Genetic predispositions, such as mutations in the APP, PSEN1, and PSEN2 genes, play a significant role. Environmental factors like exposure to toxins, including glyphosate, have been implicated in increasing the risk of developing Alzheimer's as well. Lifestyle choices, such as poor diet, lack of exercise and chronic stress, also contribute to the disease's onset.

These underlying causes contribute to the development of Alzheimer's through a series of complex biological processes. For instance, genetic mutations lead to the abnormal production of amyloid-beta plaques, which disrupt communication between brain cells. Environmental toxins like glyphosate exacerbate neuroinflammation, further damaging neurons.

Lifestyle factors such as poor diet and lack of exercise lead to oxidative stress and inflammation, both of which are known to accelerate the progression of Alzheimer's. By comprehending these mechanisms, we can better appreciate how this condition develops and progresses.

Often, Alzheimer's diagnosis relies on a combination of cognitive tests, medical history and brain imaging, which doesn't always provide a clear picture. Misdiagnosis is a risk, as symptoms like memory loss and confusion are attributed to other conditions such as depression or **vitamin deficiencies**. This complexity underscores the importance of thorough and accurate diagnostic procedures.

The limitations of current diagnostic methods lead to delays in treatment and management. Cognitive tests don't always detect **early-stage Alzheimer's**, and imaging techniques like MRI or CT scans often miss subtle changes in brain structure. Additionally, the lack of a definitive biomarker for Alzheimer's complicates the diagnostic process.

Glyphosate Exposure Accelerates Alzheimer's Pathology in Mice

A study investigated the impact of glyphosate exposure on the progression of Alzheimer's disease in mice.² The research focused on understanding how glyphosate influences neuroinflammation, the buildup of amyloid- β and tau proteins, and cognitive impairments, even after a significant recovery period.

The findings revealed that glyphosate exposure exacerbates AD-like pathology and persistent neuroinflammation in the brain, highlighting the herbicide's long-term detrimental effects. The study utilized 4.5-month-old 3xTg-AD mice, which are genetically modified to model Alzheimer's disease, alongside non-transgenic (NonTg) control mice. Only female 3xTg-AD mice were included to ensure consistent neuropathological outcomes observed in females.

The researchers administered glyphosate to these mice over a 13-week period, followed by a six-month recovery phase. Behavioral tests conducted at 12 months of age and

subsequent tissue analyses at 13.5 months provided comprehensive insights into the herbicide's effects on both AD and non-AD mice.

One of the key discoveries was that glyphosate exposure led to significant increases in both soluble and insoluble fractions of amyloid- β ($A\beta$) 40 and 42, as well as an elevated $A\beta$ 42 plaque load. The study found, "Glyphosate exposure increases soluble and insoluble fractions of $A\beta$ 40 and 42, and $A\beta$ 42 plaque load by increasing cleavage products of the amyloid precursor protein despite a recovery period."³

This indicates that glyphosate enhances the processing of amyloid precursor protein, leading to more amyloid plaques. It's important to note, however, that some research suggests **amyloid-beta accumulation is a protective response** in areas of high brain metabolic activity, not the primary cause of Alzheimer's.

Additionally, the research highlighted the accumulation of phosphorylated tau proteins in the brain. Specifically, glyphosate exposure resulted in increased levels of phosphorylated tau at multiple sites, including Threonine 181, Serine 396, and AT8 (Serine 202, Threonine 205).⁴

Phosphorylated tau forms neurofibrillary tangles, another feature of Alzheimer's pathology, disrupting neuronal function and communication. Neuroinflammation was another significant finding. The study observed elevated levels of both pro- and anti-inflammatory cytokines and chemokines in the brains and blood plasma of glyphosate-exposed mice.

It was reported, "We found increased pro- and anti-inflammatory cytokines and chemokines persisting in both 3xTg-AD and NonTg brain tissue and in 3xTg-AD peripheral blood plasma."⁵ This persistent inflammation contributes to neuronal damage and accelerates the progression of Alzheimer's disease.

Glyphosate Crosses the Blood-Brain Barrier, Affecting Brain health

Behaviorally, glyphosate-exposed 3xTg-AD mice demonstrated increased anxiety-like behaviors, as evidenced by heightened thigmotaxia – or the tendency to remain near walls – in the Morris water maze test.⁶ These behavioral changes correlate with the underlying neurological damage caused by glyphosate, further linking herbicide exposure to cognitive deficits associated with Alzheimer's.

Another aspect of the study was the detection of glyphosate and its major metabolite, aminomethylphosphonic acid (AMPA), in the brain tissues of exposed mice even after the six-month recovery period.⁷ This persistence suggests that glyphosate and its metabolites cross the blood-brain barrier and remain in the brain for extended periods, continuously affecting neuronal health.

The study also explored the biological mechanisms through which glyphosate exacerbates Alzheimer's pathology. Once in the brain, glyphosate increases the activity of the beta-secretase enzyme (BACE-1), which plays a role in the production of amyloid- β peptides from amyloid precursor protein. This enzymatic activity leads to a higher accumulation of amyloid plaques.⁸

Moreover, glyphosate-induced neuroinflammation is driven by the imbalance of cytokines and chemokines, which are signaling molecules that regulate immune responses. The study reported, "Glyphosate promoted an imbalance of pro- and anti-inflammatory cytokine and chemokine levels in the brain and periphery of 3xTg-AD mice."⁹

This imbalance results in a chronic inflammatory state, damaging neurons and accelerating the neurodegenerative processes characteristic of Alzheimer's disease. Overall, the study provides compelling evidence that glyphosate exposure significantly worsens Alzheimer's disease pathology in mice. By promoting neuroinflammation and causing neuronal dysfunction, glyphosate plays a detrimental role in the advancement of Alzheimer's disease.

The persistence of glyphosate and its metabolite in the brain underscores the long-lasting impact of exposure, emphasizing the urgent need for further research to understand its implications for human health and Alzheimer's disease progression.¹⁰

Widespread Glyphosate Exposure in the US Population

A separate study sought to determine how widespread glyphosate exposure is among Americans and what factors influence its presence in our bodies. By analyzing urine samples from the 2013-2014 National Health and Nutrition Examination Survey (NHANES), researchers found that an overwhelming 81.2% of the U.S. population aged 6 and older had detectable levels of glyphosate in their urine.¹¹ This extensive coverage highlights the pervasive nature of glyphosate in everyday life.

The study focused on a diverse group of participants, ensuring a representative sample of the U.S. population. Among the 2,310 urine samples examined, it was revealed that both children and adults are significantly exposed to glyphosate through various means. Notably, elementary-school-aged children exhibited the highest average glyphosate levels compared to other age groups.¹²

This finding raises concerns about the vulnerability of younger individuals to related health effects. One of the key discoveries was the association between fasting duration and glyphosate concentration. Individuals who fasted for eight hours or less before sample collection had glyphosate levels that were 1.94 times more likely to exceed the 95th percentile compared to those who fasted longer.¹³

This suggests that recent dietary intake plays an important role in **glyphosate exposure**, likely due to the consumption of **foods treated with the herbicide**. Furthermore, the study highlighted the widespread presence of glyphosate in the environment. Glyphosate was detected in various environmental mediums, including air particulates from rural roads, agricultural soils, water sediments and even house dust.¹⁴

This widespread distribution means that almost everyone is exposed to glyphosate regularly, whether through the food they eat, the air they breathe, or their immediate surroundings.¹⁵ The ubiquitous nature of glyphosate makes it a challenging pollutant to control and mitigate.

Exposure to Glyphosate via Food Is Widespread

Glyphosate's detection in a wide array of food items, such as fruits, cereals and pulses, underscores the herbicide's integration into the food supply chain.¹⁶ These multiple exposure pathways contribute to the high prevalence of glyphosate found in the population.

Dietary habits emerged as a significant factor influencing glyphosate levels. Participants' food consumption patterns were closely linked to the variation in glyphosate concentrations observed.¹⁷ Age also played a role in glyphosate concentration trends. The study found that glyphosate levels decreased from ages 6 to 11 and continued to decline until the age group of 20 to 59. However, there was an increase in glyphosate concentration among individuals aged 60 and above.¹⁸

This age-related fluctuation could be attributed to cumulative exposure over time or differences in metabolism and excretion rates among various age groups. Human exposure to glyphosate primarily occurs through three pathways: dermal contact, inhalation and ingestion via diet.¹⁹

Upon entering the body, most glyphosate is excreted unchanged through feces, accounting for 62% to 69% of the exposure.²⁰ In contrast, only about 1% to 6% of orally ingested glyphosate is rapidly eliminated unchanged in urine,²¹ indicating that the majority of the chemical remains in the body longer.

Children and adolescents showed higher glyphosate levels than adults, highlighting a key area of concern. The elevated levels in younger age groups may result from their dietary choices or higher susceptibility to environmental exposures.²² The study highlights the need for strategies to reduce glyphosate exposure in children, such as encouraging dietary changes and monitoring environmental sources more closely.²³

Dietary habits, specifically the types of foods consumed, significantly impact glyphosate exposure levels as well. The study found that individuals who consumed cereal-containing products had higher glyphosate levels, indicating that certain dietary choices increase exposure to glyphosate.²⁴

Conversely, consumption of conventionally grown fruits, cereals and pulses, which are more likely to be treated with glyphosate, contributed to higher urinary concentrations.²⁵ This correlation underscores the importance of choosing organic foods to minimize glyphosate intake.

Protecting Your Brain from Glyphosate's Effects on Alzheimer's

Since glyphosate exposure is widespread and crosses the blood-brain barrier to accelerate Alzheimer's pathology, you need practical steps to protect your brain and cellular health. The good news is that specific dietary and lifestyle changes reduce your exposure while supporting your body's natural detoxification processes.

- 1. Switch to organic foods** — You'll drastically reduce your glyphosate exposure by choosing organic foods, especially grains, legumes and produce. Research shows people who eat organic have significantly lower glyphosate levels in their bodies. Focus particularly on avoiding conventional wheat products, as farmers often use glyphosate as a pre-harvest drying agent.
- 2. Support your gut barrier** — **Glyphosate damages the gut microbiome** and increases intestinal permeability. Rebuild your gut health by consuming probiotic-rich foods and supporting beneficial bacteria like Akkermansia. However, excessive consumption of polyunsaturated fats (PUFAs) like **linoleic acid** in seed oils and other mitochondrial poisons also disrupt your body's mitochondrial function, reducing cellular energy and further disrupting gut health.

It's important to eliminate all seed oils from your diet for at least six months before starting an Akkermansia supplementation program. This preparatory period allows your body to recover mitochondrial function and create a more hospitable environment in your colon for the beneficial bacteria.

- 3. Boost cellular energy production** — Enhance your mitochondrial function to combat glyphosate's damaging effects on brain cells. Include targeted carbohydrates (200 to 350 grams daily for most adults) to support cellular energy production. This

helps your cells maintain their natural protective and repair mechanisms against toxin-induced damage.

- 4. Reduce environmental exposure** — Beyond food, minimize contact with glyphosate in your environment. Use natural lawn care methods instead of herbicides, filter your drinking water with systems certified to remove glyphosate and wash all produce thoroughly — even organic items due to cross-contamination.

Sources and References

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