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Scientists Discover the Missing Piece of the Brain's Drainage System

Analysis by Dr. Joseph Mercola | September 07, 2026

STORY AT-A-GLANCE

- Researchers in South Korea report finding the missing piece of your brain's drainage system — microscopic holes in the arachnoid membrane that let fluid pass out to the vessels carrying it toward your neck. They named the openings "arachnoid fenestrations"
- The holes are minuscule and sit in only one place. In mice, they measured 2 to 12 micrometers across, and appeared only in a narrow strip above the smell centers. Two monkeys showed the same pattern in the same spot
- When the team blocked the holes with beads too large to fit through, dye stopped reaching the lymph nodes in the neck — evidence, in mice, that suggest the waste fluid leaves this way
- In old mice, the holes were 59% fewer and 51% smaller, and the drainage vessels around them had withered. A growth factor given as a nasal dose brought fluid outflow back to young-adult levels — without repairing a single hole
- A separate study of donated brain tissue from 185 people found waste bundles called wasteosomes piling up along drainage routes in four different neurodegenerative diseases — a pattern the researchers call consistent with a long-underperforming clearance system, while stating plainly that they cannot prove it and that more studies are needed

Cerebrospinal fluid, the clear liquid that cushions your brain and spinal cord, carries away waste, including proteins linked to Alzheimer's disease and Parkinson's disease.[1] Researchers have long known that it eventually drains into the lymph nodes in your neck, but the step they could never account for was how the fluid crosses the arachnoid membrane, which is the protective layer that separates your brain from the lymphatic vessels beyond it.[2]

That gap in the anatomy has stood open for roughly 250 years, ever since lymphatic vessels around the brain were first described.[3]

A research team based in South Korea now reports that it has found the missing exit route, along with evidence that the route deteriorates as animals age and can be functionally restored in aged mice. Their work, which also involved Finnish and American researchers, was published in the journal Cell.[4]

What the Cell Study Showed

The work was done almost entirely on mice, as well as two long-tailed macaque monkeys (whose brain structure sits closer to ours than a mouse's does). The mice were specifically bred so their drainage vessels will glow green under a microscope, which let the researchers observe where fluid went. They also used a scanning electron microscope (SEM) to build a very high-magnification image[5] on tissue from both mice and monkeys.[6,7]

The question that the researchers aimed to answer was how cerebrospinal fluid gets out. Either the drainage vessels connect straight to the fluid-filled space around the brain, or the fluid has to squeeze through gaps in the arachnoid membrane first. Researchers have argued over the two pathways for years. All along, a pathway nobody could find was carrying half the traffic.[8]

- **The openings are real, tiny, and sit in one small patch —** Under the SEM, the researchers found actual holes in the membrane, but only in one place — a strip of tissue lying over the perforated bone behind the bridge of your nose, which

separates the brain from the nasal cavity.

The holes measured 2 to 12 micrometers across. To help you visualize, a micrometer is a thousandth of a millimeter, so about 15 of these openings would fit side by side across one human hair. That count comes from 73 holes across four mice. Nowhere else on the membrane had any, and two monkeys showed the same pattern in the same place. This is the piece that previous researchers had been missing: a physical doorway, in a specific spot, that can now be located and measured.

- **Plugging the holes stopped the drainage —** To test whether the holes actually carry fluid, the team corked them. They injected plastic beads into the fluid around the brain using two sizes: small ones fine enough to slip through, and large ones 50 times bigger, too wide to pass.

  The next day, they injected a traceable dye. In the mice given small beads, and in the untreated controls, the dye reached the neck lymph nodes normally. In the mice whose holes were corked, almost none arrived. Blocking the openings stopped the drainage. That is the strongest evidence in the paper that these holes are the exit pathway.

- **Damaging the nasal end slowed amyloid clearance, too —** If this route matters, damaging it should slow drainage, and it did. The researchers used a chemical delivered into the nose to partially destroy the smell nerves and the drainage vessels beside them.

  Dye clearance into the neck lymph nodes fell by 60% and 40% in the two sets of nodes. Repeating the test with a tagged version of amyloid-beta — the sticky protein that builds up in Alzheimer's disease — clearance fell by 49% and 26%.

  Scans showed no change in the brain's fluid volume or in pressure inside the skull, so the slowdown came from the damaged route rather than from a pressure shift. This is the closest the study comes to the disease question. The route does not just carry dye — it carries amyloid-beta.

- **Every part of the system was smaller in old mice —** Comparing mice aged 85 to 100 weeks with young adults (8 to 12 weeks), the drainage network between the smell centers covered 58% less area, and the vessels that remained looked less healthy.

  The holes themselves were 51% smaller and 59% fewer, with immune cells clinging to the membrane surface. The bone channels the vessels pass through were narrower and fewer. Drainage vessels in the lining of the nose were down by roughly half.

  The decline is not observed in one component only, but across the whole route at once — and it happens over the same stretch of life when neurodegenerative disease becomes most common.[9]

- **A growth factor restored the flow without repairing the holes —** The researchers loaded the gene for a signaling protein called vascular endothelial growth factor C (VEGF-C), which prompts drainage vessels to grow, into a harmless virus and gave it to old mice as a nasal dose. Six weeks later, the vessels between the smell centers had doubled, with large gains in the lining of the nose.

  The holes and the bone channels did not recover at all — they stayed exactly as they were. Fluid outflow returned anyway, back into the range seen in young mice. This is a notable finding because the plumbing downstream was able to make up for damage upstream, which is a more workable target. Note that it happened in mice, though, using an experimental gene therapy that does not exist for people as of this moment.

- **What the researchers say is still unknown —** Their methods cannot rule out other escape routes for very small molecules, and they could not watch the process happen live. They also do not know how long the growth-factor effect lasts, why the bone channels never recovered, or whether any of this improves how an aging brain works.

A Second Study Looked at Human Donor Brains' Waste Clearance Capabilities

While the Cell study traces the route in animals, a separate research, published in Acta Neuropathologica Communications, asked whether failing waste clearance shows up in human brains. Working with donated tissue, they counted structures called wasteosomes — microscopic bundles the brain appears to use as sealed waste containers — and found more of them in people who had died with neurodegenerative disease.[10,11]

- **The definition of wasteosome —** It has a spherical shape measuring between 2 and 50 micrometers across, the largest about three-quarters the width of a human hair. Support cells in the brain build them, and what ends up inside depends on what was swallowed, such as the tau protein in Alzheimer's disease that forms tangles inside nerve cells.

  Once made, they can be released into cerebrospinal fluid, and they have been found in the deep lymph nodes of the neck — the same place the Cell study's fluid ends up. This finding is relevant because if the brain wraps up its waste and ships it out through the drainage system, then counting the packages left behind is one way to ask, indirectly, whether the shipping worked.

- **Limitations of the research —** The team analyzed brain tissue from 185 donors: people who had died with Alzheimer's disease, amyotrophic lateral sclerosis (ALS), and two types of frontotemporal lobar degeneration (FTLD): FTLD with TDP-43 proteinopathy (FTLD-TDP) and FTLD with tau proteinopathy (FTLD-Tau). Donors with no brain disease served as the comparison group.

  They set out to score 28 brain regions, but 21 of them had to be dropped from the analysis because the signal was too faint or the scores clustered too tightly to work with, leaving seven regions to carry every result reported. In addition, one donor fell out of the main calculation, leaving 184.

- **Every disease group had more wasteosomes —** All four disease groups carried more wasteosomes than the comparison group, and the rise was spread fairly evenly across regions rather than piling up in one spot. One statistical test placed every disease group above the comparison group; another test left the ALS group just short of their threshold.

- **Age was an alternative explanation, since wasteosomes accumulate with age and the groups differed sharply in age at death —** The Alzheimer's donors averaged 85 years, and the ALS donors 61 years. So, the researchers retested everything holding age as a constant.

  The gap between the disease and comparison groups mostly held, though the Alzheimer's comparison narrowed to the point where they could no longer call it a clear difference. Age itself still mattered in the model. On that basis, they rule age out as the single shared explanation.

- **The pattern followed the drainage routes, not the diseases —** The extra wasteosomes did not pile up where each disease does its own damage. In the FTLD groups, the frontal lobe showed no increase. In ALS, the memory centers are untouched at the stages included here, yet they showed a rise anyway.

  What the affected regions had in common was positioning — they sit around a deep vein at the center of the brain and along its inner surfaces — places that have been proposed as parts of the main drainage pathway. These observations matter because if wasteosomes built up because of each disease, they should track each disease. Instead, they tracked the plumbing, which hints that clearance could be the problem the diseases might share.

- **The researchers say plainly that they cannot prove it —** There is no way to know what any donor's drainage system was doing while they were alive, so the link stays hypothetical rather than an explicit finding.

Wasteosomes may also be building up in drainage regions the study never looked at, including the nerves that carry smell signals and the smell centers themselves, where earlier work reported wasteosomes are especially concentrated. Ultimately, their own conclusion stays conditional — the findings are consistent with a long-underperforming drainage system in these diseases, and further study is required.

Everyday Habits That Support Cellular Energy While the Science Develops

The featured studies discussed here do not point to any brain-drainage intervention you can use today. What they do highlight is that clearance appears to be an active, energy-dependent process that falls off with age — and the everyday habits that support cellular energy remain the practical ground you can actually stand on. Neither study tested the steps below, but they reflect a broader strategy for supporting cellular energy.

1. **Cut linoleic acid (LA) down to roughly 5 grams a day —** LA, which is the dominant fat in seed oils, accumulates in your tissues over the years and can leak metabolites that may compromise mitochondrial function.

   Replace canola, soybean, corn, sunflower, and similar oils with tallow, ghee, or grass fed butter for cooking, and scrutinize labels on packaged foods, sauces, dressings, and restaurant meals, where these oils hide. Nuts and seeds carry LA as well, and olive oil needs moderating because of its oleic acid content, **which can contribute to metabolic dysfunction when consumed in excess**, similar to LA.

2. **Give your brain the fuel it runs on —** Glucose is the preferred cellular fuel, and your brain alone requires a minimum of 125 grams of carbohydrate per day. For most adults, roughly 250 grams of targeted carbs daily supports cellular energy production, with more for anyone highly active. Whole fruits and white rice are reasonable starting points.

If gas, bloating, pain, or irregular stools show up when you add carbs, that usually points to gut function needing attention first, so build up gradually rather than jumping straight to high-fiber foods, which can raise endotoxin in a compromised gut.

3. **Get outdoors daily, ideally around solar noon —** Sensible sun exposure remains the preferred route to optimal vitamin D, which functions as an epigenetic regulator and supports AMP-activated protein kinase (AMPK) activation.

   If you have been eating seed oils regularly, allow four to six months of reduced intake before pursuing high-intensity midday sun, since high-LA tissue increases susceptibility to sunburn. Pair sunlight with movement whenever you can — an hour of walking a day covers both, and standing more beats long, uninterrupted sitting.

4. **Treat sleep as part of your brain's maintenance schedule —** Sleep has been consistently linked to enhanced glymphatic and lymphatic clearance of brain waste, and the pathway described in the Cell paper is part of that same broader clearance system.[12]

   Create a consistent sleep window, get morning light to help reset your body's master clock, and reduce nighttime electromagnetic field (EMF) exposure in the bedroom, since these invisible waves can disrupt cellular ion balance through voltage-gated calcium channels.

5. **Know your numbers instead of guessing —** A handful of inexpensive labs can tell you more about your metabolic status than any symptom checklist. These include fasting insulin and homeostatic model assessment for insulin resistance (HOMA-IR), 25(OH)D for vitamin D status with an optimal range of 60 to 80 nanograms per milliliter (ng/mL), ferritin with a target of 60 to 75 ng/mL, and HbA1c for longer-term glucose patterns.

   Talk to your health care provider about whether these tests are appropriate for you. Then, retesting a few months after making dietary and lifestyle changes shows you whether the changes are landing.

Frequently Asked Questions (FAQs) About the Brain Waste Clearance System

Q: What exactly is a wasteosome?

A: A: This is a tiny sphere (2 to 50 micrometers across) that the brain appears to use as a sealed container for cellular debris. Support cells build them, and what ends up inside depends on what was swallowed. The Acta Neurologica Communications study counted more of them in all four disease groups than in donors without brain disease.

What that means is still open — nobody can know what a donor's drainage system was doing in life, and 21 of the 28 brain regions had to be dropped from the analysis, leaving seven to carry the result.

Q: Does a fading sense of smell mean my brain isn't draining properly?

A: Nothing in this research supports that. The confusion is understandable — the exit route sits directly above the smell centers, and when researchers deliberately damaged the smell nerves in mice, drainage dropped sharply. But that is damage causing reduced drainage in a mouse experiment, not smell loss signaling a drainage problem in a person, and neither study tested that direction.

A persistent, unexplained loss of smell is still worth raising with your health care provider for other reasons.

Q: What is the difference between the two drainage systems discussed in the featured studies?

A: One works inside brain tissue, moving fluid through it and picking up debris. The other takes over at the brain's outer wrappings and carries that fluid the rest of the way to the lymph nodes in your neck. The Cell paper mapped the handoff between them. The second study looked only at the first, and never measured its flow directly.

Q: Could the growth factor that worked in the aged mice be given to people?

A: No. VEGF-C was not a supplement, a drug, or a nasal spray — the researchers packaged its gene inside a modified virus and delivered that into the noses of mice. It exists only in the laboratory, has never been tested in humans, and is available nowhere. No treatment based on this pathway is available for any condition, and the work remains preclinical.

The mouse result was narrower than it sounds, too. Drainage recovered, but no one checked whether amyloid or tau fell, or whether the animals' brains worked any better.

Q: Can sleep help the brain clear waste?

A: Separate research has consistently linked sleep to better clearance of brain waste, and the route described in the Cell paper is part of that same broader system. Neither study here examined sleep, though — that link comes from other published studies. Still, the practical steps to support brain health remain, namely a consistent sleep window, and morning light to help reset your body's master clock.

Q: Is there a test that shows how well my brain is draining?

A: Not currently. No clinical test of brain drainage exists as a routine assessment. Researchers have proposed that wasteosomes might one day serve as an indirect marker of long-term clearance problems, but they note that direct evidence linking

these structures to drainage is still missing.

This article is for informational purposes only and does not constitute medical advice. Consult a qualified health care provider before making changes to your health regimen.

Sources and References

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