

ADVISORY COUNCIL ON ALZHEIMER'S RESEARCH, CARE, AND SERVICES

Public Comments from Advisory Council Meeting, August 2026

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napa@hhs.gov.

PLEASE NOTE: The Public Comments included here are not an endorsement of the views or information by National Alzheimer's Project Act, its Advisory Council members, the Administration or the federal agencies involved in this project.

M. Sharp | 08-05-2026

Good morning. My name is Matt Sharp, and I'm the Advocacy Manager at the Association for Frontotemporal Degeneration. Thank you for the opportunity to comment.

FTD is the most common form of dementia in people under sixty. It strikes people in the prime of their working and parenting years, and it takes an average of three and a half years to diagnose. Families lose income, insurance, and time they cannot get back while they search for an answer.

I'm here to ask that the National Plan explicitly and consistently name FTD in its goals, strategies, metrics, and implementation activities — and that it recognizes where FTD's needs diverge from Alzheimer's disease. When the Plan says "ADRD" without naming us, our families fall through the gap between the words.

Three priorities.

First, research. We need investment in FTD biomarkers, natural history, and clinical assessment tools that support earlier diagnosis, clinical trial readiness, and disease-modifying treatments.

Second, diagnosis and care. Too few providers recognize FTD. Misdiagnosis is routine. Families need real access to specialists, neuropsychological evaluation, advanced imaging, and genetic testing with counseling. And they need care models built for younger-onset dementia — for behavioral symptoms, communication loss, crisis response, and needs that change as the disease moves.

Third, the people doing the caring. Caregivers need education, respite, mental health care, financial and workplace support, and help navigating systems that were not designed for them. We need home- and community-based services and residential facilities equipped to serve people with complex behavioral needs, and a workforce — clinicians, direct-care workers, first responders — trained to recognize FTD.

Finally, measure what matters to our families: communication, independence, safety, quality of life, and staying connected to the people they love.

S. Sivakumaran | 07-27-2026

Submitted by:

Sudhir Sivakumaran, PhD

Chief Scientific Officer, Lewy Body Dementia Association

Thank you for the opportunity to submit comments on behalf of the Lewy Body Dementia Association.

Lewy body dementia is one of the most common causes of dementia, but it remains underdiagnosed, under-researched, and insufficiently represented in national research, care, regulatory, and public-health strategies. Individuals and families frequently face prolonged and uncertain diagnostic journeys, limited therapeutic options, fragmented care, substantial care-partner burden, and symptoms that extend well beyond cognition. These can include fluctuations in attention and alertness, hallucinations, parkinsonism, sleep disorders, autonomic dysfunction, mood and behavioral changes, and marked sensitivity to certain medications.

Accurate differential diagnosis matters even in the absence of an approved disease-modifying treatment. People with LBD may experience severe sensitivity to antipsychotic medications that are commonly used to manage agitation, psychosis, or hallucinations in dementia. Reactions can include profound worsening of motor and cognitive symptoms, sedation, autonomic instability, and potentially life-threatening complications. Earlier and more accurate diagnosis can therefore influence immediate treatment decisions, reduce avoidable harm, and help families anticipate and manage the complexity of the disease.

The National Alzheimer's Project Act created an essential framework for federal coordination across Alzheimer's disease and related dementias. We appreciate the inclusion of LBD and mixed-etiology dementia in ADRD research planning and disease-specific summits. The next step is to ensure that this recognition is translated into explicit priorities, implementation plans, resources, and measures of progress within the National Plan.

"Related dementias" should not function as a residual category. LBD, frontotemporal degeneration, vascular cognitive impairment and dementia, and other neurodegenerative conditions require disease-specific strategies while also benefiting from shared infrastructure and cross-disease collaboration.

LBDA encourages the Advisory Council to promote an integrated national strategy that connects basic science, biomarker development, clinical outcome assessment, longitudinal data, trial readiness, regulatory science, clinical implementation, care, education, support, advocacy, and the lived experience of people with disease and their care partners.

Biomarker progress must be disease-specific and context-specific

Biomarkers are increasingly central to diagnosis, clinical-trial design, therapeutic development, regulatory decision-making, and clinical care. However, "biomarker" is not a single use category. A biomarker may be intended to detect pathology, support diagnosis, differentiate among diseases, enrich a trial population, stage disease, predict progression, demonstrate target engagement, monitor treatment response, assess safety, or potentially serve as a surrogate endpoint.

The evidence required for each use is different. A biomarker that performs well in one disease, clinical population, disease stage, or intended use should not automatically be assumed to perform equivalently in another.

This distinction is especially important across neurodegenerative diseases. Alzheimer's disease biomarkers have advanced rapidly, including amyloid and tau PET, cerebrospinal- fluid biomarkers, and blood-based measures such as phosphorylated tau. These developments are important and should continue. However, an Alzheimer's biomarker does not become a general dementia biomarker simply because it is measured in a person with cognitive impairment.

In LBD, an Alzheimer's biomarker may identify coexisting amyloid or tau pathology that has diagnostic, prognostic, or therapeutic relevance. It does not, by itself, establish the presence, distribution, severity, or progression of Lewy body pathology. Similarly, a biomarker developed in Parkinson's disease may provide relevant biological information but may not have identical performance, interpretation, or clinical utility in dementia with Lewy bodies, Parkinson's disease dementia, prodromal LBD, or mixed-pathology populations.

The relevant question is not simply, “Does the biomarker work?” It is: “Does this biomarker, measured with this assay or imaging method, in this disease population and disease stage, support this clearly defined clinical, drug-development, or regulatory decision?”

The July 15, 2026, Duke-Margolis workshop, Advancing Biomarker Use in Regulatory Review: Evidence, Engagement, and Ecosystem Opportunities, and its preliminary policy roadmap highlighted that biomarker progress is not limited by scientific discovery alone. Progress also depends on translating biological signals into fit-for-purpose evidence, aligning analytical and clinical validation, defining the intended context of use, obtaining timely regulatory input, and building infrastructure through which data and evidence can be responsibly shared and reused.

The Advisory Council can provide important leadership by reinforcing that federal biomarker strategies must include disease-specific development plans for LBD and other related dementias, rather than relying primarily on advances made in Alzheimer’s disease or Parkinson’s disease.

LBD needs a coordinated biomarker portfolio

A comprehensive LBD biomarker strategy should include multiple complementary modalities.

First, the field needs continued development and validation of alpha-synuclein biomarkers. Emerging seed-amplification assays represent a major scientific opportunity, but substantial work remains to establish analytical comparability, biological interpretation, performance across clinical phenotypes and disease stages, longitudinal behavior, and utility for diagnosis, trial enrichment, progression modeling, target engagement, and treatment-response assessment.

Detection of pathological alpha-synuclein and quantitative measurement of disease burden are not necessarily the same. An assay may help establish that pathological alpha-synuclein is present without showing how much pathology exists, where it is distributed, how rapidly it is changing, or whether a treatment has altered the disease process. These distinctions are critical for clinical and regulatory use.

Second, additional fluid biomarkers are needed in blood, cerebrospinal fluid, and potentially other tissues. These should include markers of alpha-synuclein pathology, neuronal and synaptic injury, neuroinflammation, glial activation, axonal damage, autonomic involvement, and coexisting amyloid and tau pathology. Measures such as neurofilament light, GFAP, amyloid, and phosphorylated tau may contribute valuable information, but their interpretation and clinical utility must be evaluated specifically within LBD populations.

Third, the field urgently needs imaging biomarkers that are more directly linked to Lewy body biology. Dopaminergic imaging can provide useful supportive evidence, but it is

not a complete measure of cortical Lewy body pathology, total disease burden, or progression.

Development of reliable alpha-synuclein PET tracers remains a major unmet need. Progress will require coordinated investment in tracer discovery, selectivity testing, human neuropathological validation, standardized acquisition and analysis, longitudinal studies, and comparisons across dementia with Lewy bodies, Parkinson's disease dementia, Parkinson's disease, multiple system atrophy, Alzheimer's disease, and mixed-pathology populations.

Other imaging modalities, including structural MRI, functional imaging, synaptic-density PET, and measures of neuroinflammation or network dysfunction, may also contribute to a multimodal biological framework. The goal should not be to force the field toward one universal biomarker, but to build a fit-for-purpose portfolio capable of addressing distinct scientific, clinical, and regulatory questions.

Biological classification must be connected to clinical meaning

There is growing interest in biological classification and staging across neurodegenerative diseases. This is an important direction, but biological positivity alone is not sufficient.

LBD is heterogeneous. Individuals may have different combinations of alpha-synuclein, amyloid, tau, vascular disease, neuroinflammation, and other pathologies. The anatomical distribution and temporal sequence of pathology may differ, as may cognitive, motor, psychiatric, sleep, and autonomic manifestations.

Any biological classification framework should therefore be evaluated against practical and clinically meaningful questions:

- Does it improve diagnostic confidence?
- Does it identify biologically coherent and clinically relevant subgroups?
- Does it predict progression?
- Does it improve trial enrichment or reduce heterogeneity?
- Does it help match patients to an appropriate treatment mechanism?
- Does it explain differences in treatment response?
- Can it be implemented across community and specialty settings?
- Does it improve outcomes that matter to patients and families?

The field should avoid developing parallel and competing classification systems that use different terminology, thresholds, assumptions, and datasets without a mechanism for reconciliation. The Advisory Council, NIH, FDA, patient organizations, academic consortia, and industry could help convene a neutral process to compare emerging frameworks, identify areas of convergence, resolve material differences, and work toward an interoperable approach.

A common framework does not require treating all synucleinopathies as identical. It should preserve disease-specific and phenotype-specific distinctions while enabling shared terminology, data standards, and analytical methods where scientifically justified.

Clinical outcome measures remain a major bottleneck

Biomarkers cannot replace the need to measure how a person feels, functions, and survives. A strong therapeutic-development ecosystem requires both biological measures and fit-for-purpose clinical outcome assessments.

Existing scales frequently fail to capture the full complexity of LBD. Measures borrowed from Alzheimer's disease may emphasize memory and cognition while underrepresenting cognitive fluctuations, hallucinations, sleep disturbance, autonomic symptoms, movement impairment, neuropsychiatric burden, medication sensitivity, function, and care-partner impact. Measures borrowed from Parkinson's disease may not adequately capture the cognitive, behavioral, and functional dimensions of LBD.

This creates a serious drug-development problem. A therapy may produce a meaningful benefit in one or more LBD domains, yet a trial may fail to detect that benefit if the selected endpoint is insensitive, poorly targeted, excessively variable, or misaligned with the treatment mechanism and disease stage.

Federal research priorities should support the development, validation, and regulatory advancement of LBD-specific clinical outcome assessments. This should include clinician-reported, patient-reported, observer-reported, performance-based, and digital measures. Priorities should include reliability, validity, sensitivity to change, meaningful- change thresholds, feasibility, cross-cultural and cross-setting applicability, and suitability for clearly defined contexts of use.

Patient and care-partner input must be incorporated early. Families should help identify which changes are meaningful, which symptoms create the greatest burden, what trial procedures are feasible, and how benefit should be interpreted across the disease course.

FDA engagement should also occur before large validation programs or pivotal trials are finalized. Earlier, structured, cross-functional discussions among disease-area reviewers, translational experts, statisticians, assay developers, sponsors, consortia, researchers, and patient communities could help prevent late-stage disagreement and inefficient investment.

Shared data infrastructure is essential

LBD research remains fragmented across academic studies, clinical cohorts, observational programs, biobanks, industry trials, imaging repositories, pathology collections, and health systems. Many datasets are individually valuable but too small,

inconsistently collected, or difficult to access and combine.

The United States needs a coordinated LBD data and evidence strategy connecting clinical, biomarker, imaging, genetic, digital, neuropathological, patient-reported, and care-related information.

This does not require moving every dataset into a single repository. Federated approaches may allow data to remain within contributing institutions while supporting standardized queries and analyses. What is essential is agreement on a minimum dataset, common data elements, metadata standards, variable definitions, diagnostic criteria, outcome measures, specimen handling, imaging acquisition, assay documentation, governance, and consent for responsible future reuse.

Longitudinal data are especially important. Cross-sectional studies may show that a biomarker differs between groups. Drug development requires knowing when the biomarker changes, how rapidly it changes, how it relates to clinical progression, whether it behaves differently across subgroups, and how much change might represent a meaningful treatment effect.

The National Plan should promote standardized cross-study and cross-trial data collection, shared natural-history and longitudinal biomarker datasets, harmonized measurement practices, and prospective consent and governance that allow samples and data to be reused after individual studies conclude.

A durable evidence infrastructure should outlast any individual grant, sponsor, platform, or study.

The first-mover burden must be shared

Foundational biomarker and endpoint work is expensive, time-consuming, and often precompetitive. No single company has a strong incentive to fund all assay validation, natural-history research, data harmonization, longitudinal follow-up, and regulatory groundwork that may ultimately benefit multiple therapeutic programs.

This first-mover problem is particularly severe in LBD, where commercial investment has historically been lower than in Alzheimer's disease or Parkinson's disease.

Public-private partnerships can reduce duplication and distribute risk. NIH, FDA, FNIH, patient organizations, philanthropy, academia, diagnostic companies, imaging developers, and pharmaceutical sponsors should align around a limited number of high-priority evidence gaps and jointly support the work needed to close them.

The objective is not to lower scientific or regulatory standards. It is to generate the evidence needed to meet those standards more efficiently, transparently, and consistently.

Successful biomarker development requires more than an assay or promising publication. It requires biological rationale, standardized measurement, access to well-characterized samples, longitudinal data, appropriate reference standards, linkage to meaningful clinical outcomes, transparent statistical analysis, regulatory engagement, and a path to scalable clinical implementation.

Research progress must translate into better care, support, education, and public policy

Scientific advancement is essential, but it is not sufficient on its own. The success of the National Plan should ultimately be measured by whether people living with LBD and their families experience earlier diagnosis, better care, stronger support, clearer information, reduced burden, and greater access to effective services.

LBD presents a uniquely complex care challenge because it affects cognition, movement, behavior, sleep, autonomic function, and daily independence. Symptoms may fluctuate substantially. Treatment decisions can be complicated by medication sensitivities, coexisting conditions, and the need to coordinate across neurology, geriatrics, psychiatry, sleep medicine, primary care, rehabilitation, palliative care, and community-based services.

Families are often left to navigate this complexity with limited guidance and inconsistent access to clinicians with LBD expertise.

The Advisory Council should support a coordinated national approach that connects research and clinical care more directly. Advances in biomarkers, outcome measures, staging, and therapeutic development should be accompanied by implementation strategies for clinicians, patients, and care partners. This includes guidance on how emerging tests should be interpreted, when they should be used, what their limitations are, and how results should inform care without creating confusion, unnecessary testing, or avoidable access barriers.

Education is a critical part of this work. Many health professionals receive limited training on LBD, contributing to delayed diagnosis, misdiagnosis, inappropriate treatment, and preventable harm. Federal agencies and professional societies should collaborate with patient organizations to expand continuing education, diagnostic guidance, clinical decision-support tools, and interdisciplinary training.

These resources should reflect the full clinical complexity of LBD rather than treating it only as a cognitive disorder or as an extension of Alzheimer's disease or Parkinson's disease.

Support for care partners must also be treated as a core component of the national response. LBD care partners often manage unpredictable symptoms, safety concerns, sleep disruption, behavioral changes, mobility challenges, complex medication regimens, and difficult transitions in care. They need timely education, respite options,

care navigation, mental-health support, workplace resources, safety planning, and access to services that recognize the distinctive burden of LBD.

Advocacy organizations such as LBDA connect families to trusted information, support groups, helplines, clinical experts, research opportunities, and policy resources. They also provide an essential bridge among federal agencies, researchers, clinicians, industry, and the lived experience of the community.

The Advisory Council should recognize patient organizations as strategic implementation partners, not only as stakeholders invited to comment after plans have been developed.

National policy must also improve access across diagnosis, research, specialty care, and emerging technologies. Rural and underserved communities, people without access to major academic centers, and families facing financial, geographic, linguistic, or technological barriers may experience even greater delays.

National strategies should include community-based education, materials responsive to the needs of real patients and families, telehealth and care-navigation models, research recruitment reflective of the full population, and reimbursement approaches that ensure the benefits of research and innovation are accessible to all Americans.

Research, care, education, support, and advocacy should not operate as separate workstreams. They should form a continuous learning system. Questions raised by patients, care partners, and clinicians should shape research priorities. Research findings should be translated into accessible clinical guidance and educational resources. Real-world experience should inform regulatory, coverage, and implementation decisions.

This is the broader opportunity for the Advisory Council: not only to accelerate discovery, but to ensure that scientific progress produces visible and practical improvements throughout the LBD journey.

From recommendations to accountability

LBDA respectfully recommends that the Advisory Council support a more integrated and accountable approach within the National Plan.

1. **Establish explicit and measurable priorities for LBD and each major related dementia.** These should include research, diagnosis, clinical care, care-partner support, professional education, public awareness, and access to services, with identified lead agencies, timelines, and outcomes.
2. **Develop coordinated disease-specific biomarker roadmaps.** These should address discovery, analytical and clinical validation, context of use, regulatory application, reimbursement, clinical implementation, and communication of results to clinicians, patients, and families.

3. **Prioritize the development of LBD-relevant biomarkers.** This should include alpha-synuclein fluid biomarkers and PET tracers, together with multimodal measures of co-pathology, neurodegeneration, synaptic dysfunction, inflammation, progression, target engagement, and treatment response.
4. **Support LBD-specific clinical outcome assessments.** Measures should capture cognition, fluctuations, behavior, movement, sleep, autonomic symptoms, function, care-partner impact, and other outcomes meaningful to people living with LBD.
5. **Build shared longitudinal data and biospecimen infrastructure.** This should include common data elements, interoperable standards, harmonized assays and imaging methods, prospective consent, responsible governance, and mechanisms for reusing samples and data.
6. **Create earlier and more structured regulatory engagement pathways.** Disease communities, researchers, patient organizations, assay developers, and sponsors should be able to engage FDA before major investments are made in biomarker, endpoint, or trial-development programs.
7. **Fund public-private, precompetitive partnerships.** These partnerships should reduce the first-mover burden, support assay and tracer development, enable natural-history and longitudinal studies, and generate reusable evidence across therapeutic and diagnostic programs.
8. **Strengthen professional and public education on LBD.** Federal agencies, professional societies, academic centers, and patient organizations should collaborate on continuing education, diagnostic guidance, clinical decision-support tools, and practical resources.
9. **Improve care coordination and access to specialty expertise.** The National Plan should support interdisciplinary care models, telehealth, care navigation, community-based services, and clearer referral pathways.
10. **Treat care-partner support as a core national priority.** Care partners need disease-specific education, respite services, mental-health resources, workplace support, safety planning, and guidance through transitions in care.
11. **Recognize patient advocacy organizations as implementation partners.** Organizations such as LBDA should be engaged in priority setting, program design, education, research participation, implementation, and translation of science and policy into community benefit.
12. **Increase access for all Americans.** National efforts should address geographic, financial, clinical, technological, and other barriers to diagnosis, research participation, specialty care, services, and emerging technologies.
13. **Create a continuous learning system across research and care.** Questions from patients, care partners, and clinicians should inform research priorities, while scientific findings and real-world evidence should inform clinical, regulatory, reimbursement, and implementation decisions.
14. **Track progress publicly through disease-specific accountability measures.** The National Plan should report measurable progress in diagnosis, biomarker and endpoint development, research participation, therapeutic development, access to knowledgeable care, professional education, care-partner support, and population-level outcomes.

Conclusion

The opportunity before the Advisory Council is larger than accelerating research alone. It is to build a coordinated national system in which discovery, diagnosis, treatment development, care, education, support, and advocacy reinforce one another.

For LBD, the scientific foundation is beginning to strengthen. Alpha-synuclein assays, advanced imaging, fluid biomarkers, digital measures, longitudinal datasets, biological staging approaches, and improved clinical assessments all offer promise. At the same time, families continue to experience delayed diagnosis, fragmented care, limited access to knowledgeable clinicians, inadequate support, and too few meaningful treatment options.

Progress in one part of the system will not be sufficient if the remainder stays disconnected. A new biomarker has limited value if clinicians do not know when to use it, patients cannot access it, results are poorly communicated, or no treatment or care pathway is linked to the information it provides. An improved therapy will have limited reach if diagnosis occurs too late, trial populations do not reflect the broader community, or reimbursement and specialty access remain uneven.

Research progress must therefore be planned together with clinical implementation, professional education, care-partner support, public policy, and access.

LBDA's mission reflects this broader responsibility. Advancing science and therapeutic development is essential, but so is helping people receive an accurate diagnosis, access knowledgeable and coordinated care, understand the disease, connect with support, participate in research, and live as well as possible throughout the course of LBD.

The National Plan should recognize patient advocacy organizations as strategic partners in this work. LBDA and other disease-focused organizations can connect federal agencies, researchers, clinicians, industry, patients, and care partners; identify unmet needs; improve education and research participation; and translate national priorities into practical benefits for families.

People living with Lewy body dementia should not have to wait for progress in another disease to eventually reach them. They deserve a focused, scientifically rigorous, compassionate, and accountable national strategy that addresses the entire journey from discovery to diagnosis, from research to care, and from policy to everyday life.

LBDA stands ready to work with the Advisory Council, federal agencies, researchers, clinicians, industry, and the broader community to make that vision a reality.

S. Frey | 08-03-2026

Statement from Scott Frey
Senior Vice President, Public Policy and Government Relations,
Alliance for Aging Research, On Behalf of Project PAUSE
Before the Advisory Council on Alzheimer's Research, Care, and Services
I am Scott Frey, Senior Vice President of Public Policy and Government Relations at the
Alliance for Aging Research.

On behalf of Project PAUSE, a coalition of patient and professional organizations focused on improving long-term care for individuals who have neuropsychiatric symptoms associated with Alzheimer's and other neurocognitive conditions, I'd like to discuss our longstanding concerns about CMS's Long-Stay Antipsychotic Quality Measure, which we believe is fundamentally flawed and has, for too long, unintentionally denied many individuals from consistently receiving clinically appropriate care.

While intended to reduce inappropriate antipsychotic use, the measure relies on a one-size-fits-all approach that does not distinguish between clinically appropriate and inappropriate prescribing. Instead, it measures, with only three exempt conditions, how many individuals in a facility received an antipsychotic. It overlooks clinically appropriate indications, fails to reflect newer FDA-approved therapies, and incorrectly equates lower prescribing with higher-quality care.

As a result, it creates pressure to reduce clinically appropriate treatment, which leads to involuntary discharges, barriers to nursing home admission, and unnecessary suffering for individuals with neuropsychiatric symptoms and their families.

To address these issues, we respectfully continue to urge CMS to retire the current measure and replace it with targeted, low-burden improvements to the Minimum Data Set that strengthen prescribing documentation, reinforce multidisciplinary clinical oversight, and better distinguish clinically appropriate treatment from misuse.

CMS can modernize the measure, advance person-centered care, and safeguard patients. We look forward to partnering with CMS to advance these reforms.

Thank you.

D. Stufflebam | 08-04-2026

Hi, my name is Dee Stufflebam and we live in St. Louis. I have a 38-year-old daughter, Susie, who has Down syndrome. Susie is great. She lives in an independent residential facility, works at TJ Maxx and has a great social life.

Susie currently shows no signs of Alzheimer's. She has sadly lost a few of the residents in her facility who developed Alzheimer's and had to return to their parents' home, or in 2 cases, to a nursing home. She has felt this loss deeply. The parents of these friends have shared the changes their families have had to make to accommodate caring for an adult child with both Down syndrome and Alzheimer's. Siblings have had to quit their jobs, and aging parents have needed to get more in-home care.

Susie has been participating in Alzheimer's research for adults with Down syndrome for almost 10 years through Washington University. Susie has had MRIs, PET scans, lots of lab work and even 2 lumbar punctures. Cognitive testing has also been done at least every 18 months. Most recently she started a new study that required her to use her cell phone 3 times a day for 7 days to answer questions. She did great (she loves her cell phone). Susie loves the attention she gets from the great people at Washington University but mostly she loves to boast about the money she is paid for participating.

Susie's dad and I have also participated in a parent study. We do not know what the future holds for Susie, but the thought of her "losing her spark," as one of her friend's mom said, is heartbreaking.

We keep Susie active, she takes a GLP-1 to keep her weight under control, wears a CPAP, gets vaccines, and eats mostly healthy and exercises daily, all in an effort for her to keep her thriving. The studies indicate that even adults with Down syndrome who are high functioning, as Susie is, still have a very high incidence of developing Alzheimer's. And often as early as their mid 40's We will continue to encourage Susie to participate in the studies. We have been told that scans typically show indications of Alzheimer's 10 years before symptoms develop, so when that happens, we will be very willing to try medication treatment as deemed appropriate.

I recently was a presenter before the FDA to encourage drug manufacturers to include individuals with Down syndrome in their Alzheimer's drug trials.

Thank you for listening and I encourage you to continue to seek answers to this dreadful disease, for people with Down syndrome, and for the population as a whole.



3 August 2026

Comments delivered live at the meeting by Margot Rhondeau, Chief Programs Officer

My name is Kate O'Neill, and I am the Chief Research Engagement Officer at the National Down Syndrome Society (NDSS). Thank you for this opportunity to raise awareness of the impact that Alzheimer's disease has on individuals with Down syndrome and to reiterate why the Down syndrome community must be included in the work of the Advisory Council on Alzheimer's Research, Care, and Services.

As Dr. Rafii explained earlier in today's meeting, individuals with Down syndrome have a 90% risk of developing Alzheimer's disease in their lifetime, because they are born with an extra copy of chromosome 21. At a devastatingly early age, people with Down syndrome experience the changes in behavior, thinking, and memory loss related to Down syndrome-associated Alzheimer's disease (DS-AD). The average age of onset in individuals with Down syndrome is 54 with an average disease duration of 4.5 years.

Given these realities, the Down syndrome community advocates with a profound sense of urgency on this matter. NDSS, together with other support organizations and experts like Dr. Rafii, is working on multiple fronts to ensure that people with Down syndrome have access to existing and emerging diagnostics, therapeutics, and treatments for Alzheimer's disease.

On June 2, 2026, NDSS convened an Externally-Led, Patient-Focused Drug Development (or EL-PFDD) meeting with the Food and Drug Administration (FDA), bringing the Down syndrome community together to focus on the lived experience of DS-AD in a public forum.

This was an important milestone for our community. More than 160 participants engaged throughout the day, including 20 adults with Down syndrome and caregivers who shared their experiences. They were joined by subject matter experts, all the national Down syndrome organizations, multiple pharmaceutical companies, and the Alzheimer's Association. The meeting drew strong FDA participation, with 28 FDA staff members attending. The follow-on from that meeting will include the publication of the Voice of the Patient Report, a formal record of the findings of the meeting that we look forward to sharing with this Council, and a more technical Scientific Workshop on DS-AD in 2027.

In addition, our organization educates Alzheimer's researchers who are beginning to welcome people with Down syndrome into clinical trials. We recently launched an on-demand, digital training that provides tools and strategies to help research teams adapt to include adults with Down syndrome at their study sites.

NDSS was also proud to work with Congress on the 2024 NAPA Reauthorization Act (P.L. 118-92), which expanded the number of non-federal members on the council from 12 to 15, created a new seat to represent an underserved population with an elevated lifetime risk of developing

Alzheimer's disease, and made explicit reference to the Down syndrome community as such an underserved community. We strongly recommend that a representative of the Down syndrome community be included on this Council.

In light of the body of compelling data, the overwhelming impact this disease has on the Down syndrome community, and the exciting advances in research presented by Dr. Rafii, all evidence points to this pivotal moment when action is critical.

We urge this Council to ensure that people with Down syndrome have a seat at every table and a voice in every decision. By doing so, we can advance equitable research, accelerate progress, and improve outcomes for the Down syndrome community and all those who are affected by Alzheimer's disease.

Thank you.

Kate O'Neill
Chief Research Engagement Officer
National Down Syndrome Society

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D. O'Gara | 08-03-2026

My name is Dawn O'Gara from Massachusetts. I am the daughter of Jim Tobin, who lived with behavioral variant frontotemporal degeneration, or FTD. I also serve as an AFTD Ambassador.

Thank you for the opportunity to speak today.

The progress made through NAPA has been significant, but families living with FTD and other younger-onset dementias are still being left behind by policies that were built primarily around Alzheimer's disease.

My father was diagnosed before the age of 65. Because of his age, many dementia services simply weren't available to him. As our family's caregiving responsibilities increased, so did the financial strain. Families like ours often lose not only one income, but two, while still raising children or helping pay for college. Yet many programs remain tied to age instead of need.

Even when services were available, they were designed specifically around Alzheimer's disease. In his nursing facility, staff treated him like every other resident with dementia. But behavioral variant FTD is different. His memory remained relatively intact, so techniques commonly used for Alzheimer's, such as therapeutic fibbing, only increased his frustration because he knew he was being lied to.

Later, after we brought him home, we sought palliative care. A nurse reviewed his condition and agreed he met the dementia criteria until she learned he had FTD instead of Alzheimer's. She closed her notebook, said he no longer qualified, and walked out of our home without even examining him.

No family should lose access to care simply because their loved one has a different form of dementia.

I respectfully ask the Council to ensure equitable access to dementia services regardless of age or diagnosis, expand education on FTD and other dementias throughout the healthcare system, and continue building a national dementia strategy that recognizes Alzheimer's disease is not the only dementia.

Thank you for your time and for considering the needs of all families living with dementia.

Dawn O'Gara
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D. Kirby | 08-04-2026

To Whom It May Concern,

Thank you so much for the opportunity to voice my concerns and share our story of the heartbreaking reality of young onset dementia. I'm an Ambassador for the AFTD representing Illinois and Indiana BUT most importantly, I'm a mother with a passion to help others receive a timelier diagnosis, understanding and the care they deserve, as they navigate the journey of young onset dementia. I appreciate the work that the Council has done in recent years, though I feel those with "other related disorders" deserve more attention.

Our sweet daughter, Kara, was finally diagnosed after a grueling 2-year journey through misdiagnoses, psychiatric ward stays, unnecessary treatments and mistreatment due to lack of knowledge of young onset dementia. We finally received the Behavioral Variant Frontotemporal Degeneration diagnosis at Mayo Clinic in Rochester, MN. They were able to put a name to the " Monster Under the Bed", the reason our 29-year-old daughter no longer acted like the independent, sweet spirited, sometimes sassy, but caring young lady. We were heartbroken over this diagnosis but now knew what we were dealing with, determined to make the best of everyday we had with her. During this time, I lost my parents and was caring for me MIL with early stages of Alzheimer's.

Our sweet Kara, became totally dependent, doubly incontinent, lack of all executive functioning, mute and even unable to smile or show emotions. Kara's memory was mostly intact even until the end when she became unable to swallow and digest food. She was able to recognize family, hug, hold hands and give the I Love You hand sign. As we say, " Love Needs NO Words". My husband and I were blessed to care for Kara 24/7 for 4 years until FTD took her life at just 33! The financial, emotional, and physical toll was great on both of us but we cherished the time with her as we knew it was limited.

My passion for the very young onset dementia community stems from my love for my daughter, and the anguish we endured as we watched her suffer a devastating illness which was magnified by the lack of awareness, education, care and services. Unfortunately, these failures by the medical community are largely due to her young age vs. her needs.

Justin, Jessica, Mark, Larry, Ben, Christina, Daria, Matthew, Christian, Ashley, Camiya, are just a few of the young adult children of my "Sister Mama Bears" as we call ourselves. We cared for, loved and longed for the best quality of life for our children as we navigated our journeys with challenges due to lack of national awareness, services, and care.

Rare doesn't mean insignificant or unimportant. "The other related disorders " deserve

to be named and recognized. Unfortunately, I have learned that FTD isn't as rare as we probably think, but rarely diagnosed due to lack of awareness and education. When you hear "the other related disorders" think of Kara, Justin, Daria, Jessica, Larry, Christina, Mark, and Ben, all of which were Sister Mama Bear's children that we lost before they were 40.

I am blessed to be given the names and information of other "Mama Bears" that have reached out to the AFTD helpline so I can let them know they are not alone. FTD needs to be more visible so we can help others who are misdiagnosed with psychiatric illnesses. Please Name IT so we can Tame IT.

We donated Kara's brain to Mayo Clinic where she was enrolled in research after her diagnosis. She had the FUS protein pathology though not connected to gene mutation. I strongly believe that brain donation needs to be emphasized as well with those with neurodegenerative diseases.

Thank you so much for allowing me to expand on my public comments in this email. I felt more details to our journey could be helpful to see the impact on these young adults and their families.

Feel free to contact me anytime if my input can be beneficial for your council. I truly appreciate your time, understanding and dedication to the Dementia community.

Thank You,
Dawn Kirby
AFTD Ambassador



J. Wood | 07-27-2026

Public Comment to the NAPA Advisory Council
Submitted on behalf of the Lewy Body Dementia Association

Thank you for the opportunity to provide public comment to the Advisory Council on Alzheimer's Research, Care, and Services under the National Alzheimer's Project Act. On behalf of the Lewy Body Dementia Association, we urge the Council to elevate an urgent and preventable safety issue affecting people living with Lewy body dementia, including

both dementia with Lewy bodies and Parkinson's disease dementia: the continued administration of haloperidol, commonly known as Haldol, and other high-potency dopamine-blocking antipsychotics for psychosis, agitation, or behavioral symptoms in this population.

Lewy body dementia is uniquely associated with severe neuroleptic sensitivity. LBDA clinical guidance warns that a high percentage of patients with dementia with Lewy bodies experience worsening parkinsonism, sedation, immobility, or even neuroleptic malignant syndrome after exposure to antipsychotics, and specifically states that typical antipsychotics such as haloperidol should be avoided. LBDA's emergency-room guidance further estimates that severe sensitivity reactions occur in approximately 25% to 50% of patients with Lewy body dementia given antipsychotic drugs in usual dose ranges; these reactions can include worsening cognition, sedation, acute or possibly irreversible parkinsonism, autonomic dysfunction, and symptoms resembling neuroleptic malignant syndrome, which can be fatal. Sources: LBDA, "Treatment Guidance and Medication Considerations in LBD"; LBDA, "Emergency Room Treatment of Psychosis in LBD."

The federal warning is also clear. The FDA-approved labeling for Haldol injection states that elderly patients with dementia-related psychosis treated with antipsychotic drugs are at increased risk of death; analyses of 17 placebo-controlled trials found death rates of about 4.5% among drug-treated patients compared with about 2.6% among placebo-treated patients over a typical 10-week trial, or approximately 1.6 to 1.7 times higher risk. The same labeling states that Haldol is not approved for dementia-related psychosis and lists Parkinson's disease and dementia with Lewy bodies as contraindications. Source: U.S. Food and Drug Administration, Haldol Injection prescribing information, 2019 label.

Published clinical evidence underscores why these warnings matter. A Lancet report on neuroleptic sensitivity in dementia with Lewy bodies described severe sensitivity reactions—including cognitive decline, parkinsonism, drowsiness, and features of neuroleptic malignant syndrome—in 50% of exposed patients in earlier work, with a three-fold increase in mortality; in the later autopsy-confirmed cohort, 21 of 40 patients with dementia with Lewy bodies had received neuroleptics, and 53% of those exposed experienced neuroleptic sensitivity. Source: Ballard, Grace, McKeith, and Holmes, "Neuroleptic sensitivity in dementia with Lewy bodies and Alzheimer's disease," The Lancet, 1998.

For families, this is not an abstract prescribing issue. A person with Lewy body dementia who is given haloperidol for hallucinations, delusions, agitation, or emergency behavioral symptoms may deteriorate rapidly: becoming more rigid, less mobile, more confused, more sedated, less able to swallow or communicate, and at higher risk for falls, aspiration, hospitalization, institutionalization, or death. These events can transform an already difficult dementia journey into an avoidable crisis for patients and caregivers, erode trust in emergency and acute-care systems, and worsen prognosis by accelerating functional decline and reducing the person's ability to remain safely at home.

LBDA respectfully asks the NAPA Council to recognize haloperidol exposure in Lewy body dementia as a patient-safety priority and to recommend practical safeguards across federal, clinical, and long-term care settings. These safeguards should include: clear education for clinicians, emergency departments, hospitals, long-term care facilities, first responders, and families that haloperidol and other typical antipsychotics should be avoided in dementia with Lewy bodies and used with extreme caution in Parkinson's disease dementia; medication-reconciliation prompts and electronic health record alerts that flag Lewy body dementia, Parkinson's disease dementia, Parkinsonism, and neuroleptic sensitivity; inclusion of Lewy body dementia-specific psychosis guidance in dementia care models and quality measures; and support for research and care pathways that prioritize non-drug approaches, caregiver education, cholinesterase inhibitors when appropriate, and specialist consultation before antipsychotic exposure whenever possible.

The national dementia strategy must address the realities of related dementias, not only Alzheimer's disease. Lewy body dementia patients are commonly seen in emergency, inpatient, and long-term care settings where psychosis or behavioral symptoms may be treated urgently. Without visible, standardized warnings and education, families remain responsible for preventing unsafe medication exposure at exactly the moment when they are most frightened and least able to advocate. The Council can help shift that burden from families to systems by making Lewy body dementia medication safety a core element of dementia-capable care.

We urge the Council to promote a clear national message: haloperidol and other typical antipsychotics can be dangerous, and sometimes fatal, for people with Lewy body dementia. Safer care begins with recognition, diagnosis, family education, clinician training, and systems that prevent contraindicated medications from being administered in moments of crisis.

Selected citations for the record: Lewy Body Dementia Association, "Treatment Guidance and Medication Considerations in LBD"; Lewy Body Dementia Association, "Emergency Room Treatment of Psychosis in LBD"; U.S. Food and Drug Administration, Haldol Injection prescribing information, 2019 label; Ballard C., Grace J., McKeith I., Holmes C., "Neuroleptic sensitivity in dementia with Lewy bodies and Alzheimer's disease," The Lancet, 1998.
