Beyond Blood: Unlocking Alternative Non-invasive Sample Types using NULISAseq™

Translating biofluids with highly sensitive, multiplex proteomics

Most researchers consider plasma and serum the primary biofluids for conducting proteomics and multiplex immunoassays. While venipuncture-based specimens provide valuable insights for systemic protein biomarker analysis, other sample types that are more proximal to diseased tissue can be less invasive and avoid collection challenges. As these samples are often available in limited or dilute quantities, target proteins are present at low abundance, underscoring the need for sensitive assays.

The NULISA™ platform combines industry-leading sensitivity with multiplexing to deliver unmatched biomarker analysis from small-volume biofluids.[1] Sensitivity ensures disease-associated, low-abundance markers are detected and not overlooked. Multiplexing hundreds of proteins in targeted panels maximizes the utility of proteomic data obtained from small, precious samples. Moreover, non-invasive techniques allow for more frequent sampling, enabling longitudinal studies. These designs enhance statistical analysis through repeated measures and chronicle disease and treatment trajectories, which are essential for precision medicine and outcomes-based care.[2]

In addition to blood-based biofluids, NULISA has demonstrated remarkable utility in analyzing cerebrospinal fluid (CSF), both using the NULISAseq Inflammation Panel 250 (e.g., see ref[3]) and the NULISAseq CNS Disease Panel 120 (e.g., see ref[4]). This white paper highlights real-world applications in which researchers have tested alternative non-invasive sample types with the NULISAseq Panels, providing sample-specific preparation considerations and performance data. A summary of sample types and associated protein detectabilities achieved with NULISA is reported **Table 1**.

Table 1. Summary of NULISAseq protein detectability across a broad range of sample types. Note that overall detectability may vary based on the collection method, disease biology, and other variables. For more information about a particular sample type, please email us at support@alamarbio.com.

NULISAseq Inflammation Panel 250			NULISAseq CNS Disease Panel 120		
Sample type	**Detectable targets (250 total)**		**Sample type**	**Detectable targets (124 total)**	
	Number	**Percent**		**Number**	**Percent**
Dried Blood & Plasma Spots*	200–240	80–95	Dried Blood & Plasma Spots*	100–120+	80–95+
Interstitial Fluid	235+	95+	Interstitial Fluid	105+	85+
Saliva	185–215	75–85	Saliva	90–105	75–85
Urine	110–190	45–75	Urine	40–60	35–45
Skin tape	60+	25+	Skin tape	30+	25+
Synovial Fluid	235+	95+	Tear fluid	70–95	60–75
Nasal Swabs	175–200	70–80	Brain lysate	40–55	35–45
Sputum	150+	60+	Plasma-derived EV	60–100	50–80
Stool	25–50	10–20	CSF-derived EV	20–120	15–95

*See NULISAseq™ Analysis of Samples Collected by Microsampling and Dried Blood and Plasma Spot Methods.

Urine: Rapid, Non-Invasive Biomarkers of Kidney Function and Injury

Jamie Lin, M.D., FACN

Assistant Professor
MD Anderson Cancer Center (Houston, TX, USA)
Section of Nephrology

Dr. Lin is a nephrologist studying kidney injury resulting from cancer immunotherapy. She is the co-director of the Nephritis section of MD Anderson's nationally recognized IOTOX research committee in the treatment and management of immuno-oncology-related toxicology.

Recent advances in protein technologies, including NULISA, have made urine an accessible sample type for highly sensitive proteomic detection in large-scale and longitudinal studies. Non-invasive and abundant in volume, urine can be collected from any subject and can provide information on kidney function and disease.

In cancer immunotherapy, approximately 20% of patients receiving immune checkpoint inhibitors (ICI) develop an acute kidney injury (AKI), of which a small subset presents with acute interstitial nephritis (AIN). In patients receiving ICI who develop AKI, the current standard of care is to pause therapy and obtain an invasive biopsy for diagnosis and treatment recommendation. A urine-based biomarker specific to AIN would alleviate the time delay, discomfort, and bleeding risk associated with a kidney biopsy.

To identify prognostic biomarkers of AIN, Dr. Jamie Lin and colleagues profiled urine and plasma samples from patients receiving ICI using the NULISAseq Inflammation Panel 250.

The analysis included 49 total patients with AKI, including 22 and 27 who did or did not develop ICI-AIN, respectively. For urine, 73% of the targets achieved detectability in more than 50% of the samples, compared to 95% in the plasma samples. However, the urine samples provided a richer source of biomarker candidates than plasma, containing 59 differentially expressed proteins in ICI-AIN compared to 22 unique to plasma. **Table 2** presents the top performing proteins in differentiating ICI-AIN from non-AIN.

After applying multiple data filtering strategies, the team identified a two-protein signature comprising the apoptosis receptor FAS and the cytokine IL-5, which optimally predicted ICI-AIN diagnosis with 94% accuracy by area under the curve (AUC) analysis.[5]

This study demonstrates that NULISAseq can reveal non-invasive, highly robust, low abundance biomarkers in urine with good detectability, potentially addressing unmet needs in clinical research.

Table 2. NULISAseq Inflammation Panel 250 in urine identifies proteins with high diagnostic accuracy for ICI-AIN positivity

Protein	AUC	Fold Change	p-value (FDR corrected)
IL5	0.892	28.93	8.47E-05
Fas	0.887	23.99	1.62E-03
TNFSF4	0.842	22.15	1.78E-03
CXCL9	0.832	20.67	1.62E-03
CD274	0.832	15.44	1.39E-03
IL20	0.830	14.81	1.50E-03
TNFSF15	0.827	10.52	1.58E-03
TSLP	0.820	10.15	4.02E-04
TREM1	0.815	9.51	1.62E-03
CCL1	0.811	8.54	1.39E-03
IL5 + Fas	0.941	-	-

AUC = Area Under the Curve; FDR = False-Discovery Rate
Data were reported in Long et al (2025) and reflect urine markers with AUC > 0.8, FDR < 0.01, and fold change > 8.0.[5]

Tear Fluid: Non-Invasive Markers of CNS and Ocular Disease

Marlies Gijs, Ph.D.

Assistant Professor
Maastricht University (Netherlands)
University Eye Clinic Maastricht

Dr. Gijs is a pioneer in ophthalmology, assessing the utility of tear fluid as a source of non-invasive biomarkers for ocular and systemic conditions. Her accomplishments include the detection of neural markers in tear fluid, founding the Tear Research Network to harmonize the field and to accelerate clinical adoption of tear fluid biomarkers, and managing a tear fluid biobank at Maastricht.

Tear fluid represents a powerful source of non-invasive biomarkers for ocular and neurodegenerative diseases, including Alzheimer's disease (AD) and Huntington's disease. Levels of critical neurodegenerative protein biomarkers in tear fluid extracts, such as total tau, phosphorylated tau, and amyloid-beta peptides, correlate with disease severity and clinical dementia.[6]

Dr. Gijs's lab developed a quantitative tear fluid collection method that relies on Schirmer's strips and maximizes protein extraction while minimizing protein degradation.[7]

To determine the detectability of NULISAseq CNS Disease Panel 120 proteins, human tear fluid from healthy subjects was collected as previously described.[7] In this pilot study, 88 of the 124 targets (71%) achieved detectability in more than 50% of samples (**Figure 1A**). Phosphorylated tau species pTau-181 (100%), pTau-217 (83.3%), and pTau-231 (100%) were above the limit of detection (LoD, defined as the mean signal + 3 * SD) in most samples, demonstrating the high sensitivity of the NULISA technology to detect these important biomarkers at baseline levels.

The overall detectability of CNS disease targets, expressed as NULISA Protein Quantitation (NPQ) values above the LoD, is presented in **Figure 1B**. These data demonstrate how diverse protein biomarkers can be readily identified in tear fluid samples due to the high sensitivity of NULISA assays. A larger cohort study involving healthy and disease-state donors would further establish the feasibility and translatability of routinely measuring markers of interest.

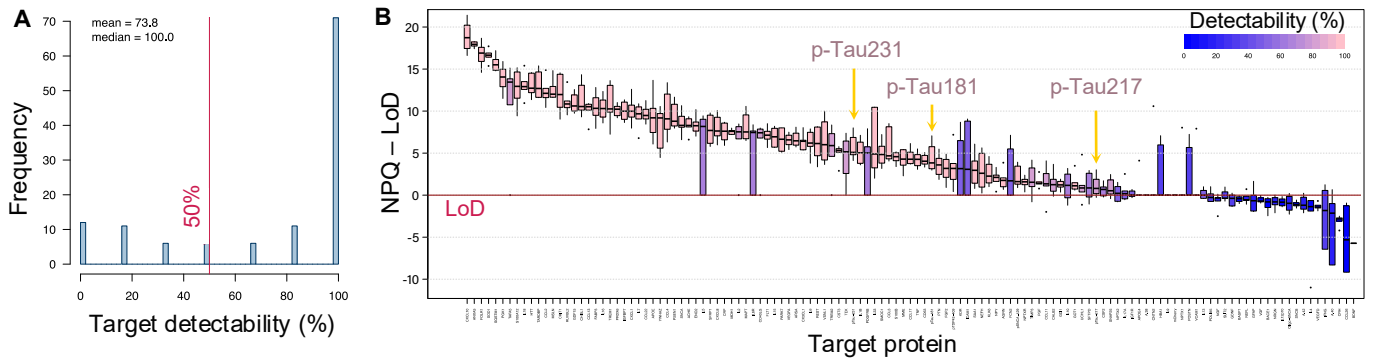


Figure 1. NULISAseq CNS Disease Panel 120 achieves 71% detectability in tear fluid. (A) Target detectability across n=6 samples from healthy controls, (B) Detectability plot for each protein in the panel; high and low detectability are shaded pink and blue, respectively. Phosphorylated tau species are indicated. The limit of detection (LoD) is delineated in magenta. NPQ = NULISA Protein Quantification.

Extracellular Vesicles: Tissue-Specific, Ultra-Low-Abundance Proteins in Biofluids

Amanda Cano, Ph.D.

Head of Molecular Biology & Biomarkers Program, Ace Alzheimer Center (Barcelona)

Dr. Cano investigates the role of novel biomarkers and pathways involved in Alzheimer's disease pathophysiology using proteomic, lipidomic, and metabolomic analyses in human biospecimens.

Extracellular vesicles (EVs) broadly encompass the heterogeneous lipid bilayer-encased particles released by all cell types. Interest in EVs as a source of liquid biopsies stems from their role in intercellular communication and their biomarker cargo, which could aid in the diagnosis and monitoring of pathological conditions. The ability to isolate cell-specific EVs by immunocapture based on their unique surface marker profiles can also be provide insights into the molecular events and pathways mediating disease pathogenesis at the cellular level (**Figure 2A**).[8]

As EVs constitute 50 nl per 1 ml of human plasma (0.00005%), their protein cargos are present in strikingly low abundance.[9] Moreover, EV concentrations in CSF are lower than in plasma and technically more challenging to isolate.[10] Consequently, highly sensitive techniques with low volume requirements, such as NULISA, may be ideal for the proteomic analysis of EVs derived from both CSF and plasma.

To test the feasibility of the NULISAseq CNS Disease Panel 120 in EVs, Alamar collaborated with Dr. Amanda Cano of the Ace Alzheimer's Center Barcelona. Total EVs were isolated by ultracentrifugation of the CSF from three individuals with mild cognitive impairment (negative for amyloid, tau, or neurodegeneration pathology, A-T-N-) and three individuals with AD dementia (A+T+N+). Of the 124 targets, 113 (91.1%) achieved detectability in more than 50% of samples (**Figure 2B**). The amyloid beta peptides (Aβ38, Aβ40, and Aβ42) and phosphorylated tau species (p-Tau181, p-Tau217, and p-Tau231) were detected in all six samples tested (**Figure 2C**). Other prominent markers of neurodegeneration, including neurofilament chains NEFL and NEFH, several cytokines, interleukins, chemokines, TREM1, and TREM2, were detected in all samples.

Blood-based collections are considered less invasive than lumbar puncture, and Alamar has also analyzed plasma-derived EVs using the NULISAseq CNS Disease Panel 120. Many of these EVs were purified based on tissue-specific antibodies that indicate their brain anatomical source. Detectability ranged from 14–77% (17–94) of the Panel's 124 targets, depending on the abundance of the specific EV (**Table 3**). These data support NULISAseq utility in identifying multiple low-abundance biomarkers from precious CNS-derived and plasma EVs.

Table 3. Summary of EVs tested with the NULISAseq CNS Disease Panel 120

EV Sample Type	% of Detectable Targets
CSF-derived EVs	15-95%
Plasma-derived EVs	65-75%
Cell-derived EVs	25-40%

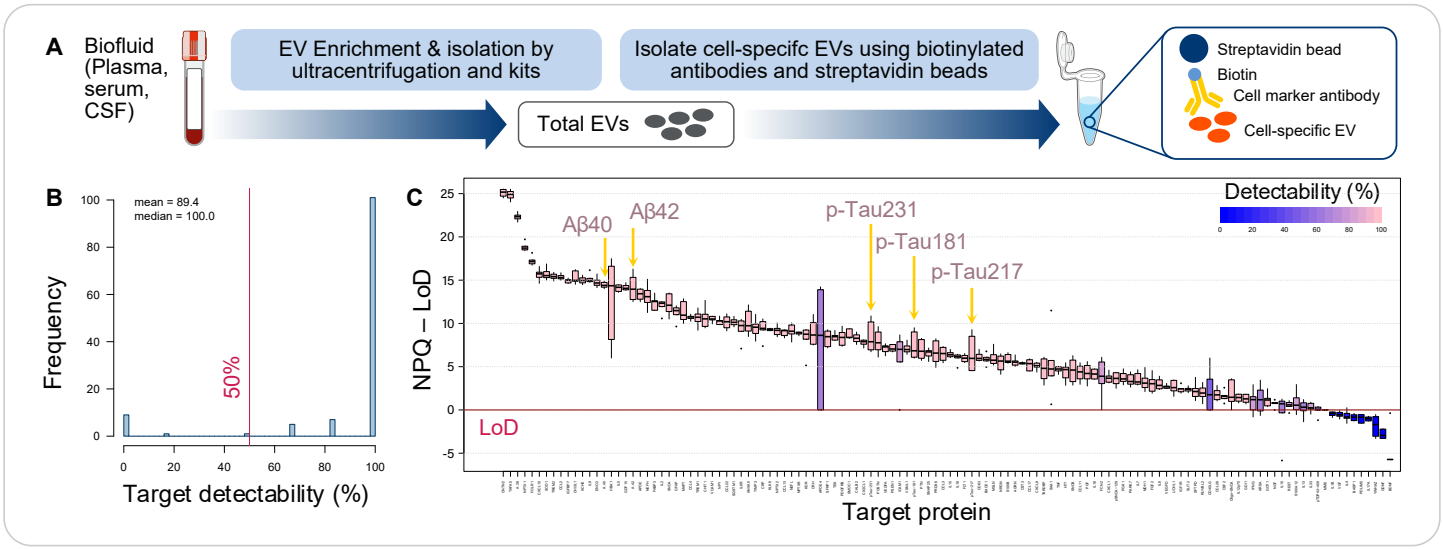


Figure 2. NULISAseq CNS Disease Panel 120 achieves >91% detectability in CSF-derived EV specimens. (A) Overview of total EV and cell-specific EV isolation.[8] (B) Target detectability across six samples, (C) Detectability plot for each protein in the panel; high and low detectability are shaded pink and blue, respectively. Amyloid beta peptides and phosphorylated tau species are indicated. The limit of detection (LoD) is delineated in magenta. NPQ = NULISA Protein Quantification.

Saliva and Nasal Swabs: Replete with Respiratory Markers

Kevin Matthew Byrd, D.D.S., Ph.D.

Assistant Professor, Virginia Commonwealth University School of Dentistry
Philips Institute for Oral Health Research

Dr. Kevin Matthew Byrd leads the Lab of Oral & Craniofacial Innovation at Virginia Commonwealth University. The lab develops non-invasive and spatially-resolved diagnostic approaches to uncover how immune, stromal, and epithelial networks regulate health, inflammation, and disease susceptibility. By integrating AI-driven analytics with human biospecimens and engineered tissue models, the group aims to translate molecular insights into scalable tools for precision diagnostics, early disease detection, and therapeutic monitoring across immune and mucosal disorders.

Saliva collections through passive drooling and nasal swab extractions offer appealing options for identifying proteomic biomarkers associated with upper respiratory infections, including COVID-19, as well as oral conditions such as periodontitis. Although these noninvasive sampling methods eliminate the need for needle sticks or specialized collection devices, they contain proteins at notoriously low abundances, particularly the host-specific (as opposed to bacterial) proteins that inform on immunological parameters.[11]

Alamar collaborated on a project led by Brittany Rupp in Dr. Kevin Matthew Byrd's lab at the ADA Science and Research Institute to assess the feasibility of the NULISAseq Inflammation Panel 250 in interrogating saliva and nasal swab specimens. Matched saliva and nasal swabs were collected from healthy adults aged 20–40 years for multimodal analysis, highlighting the requirement for low-volume inputs. Prior to NULISAseq analysis, saliva samples were centrifuged to pellet cells, and nasal swabs were resuspended in a buffer containing PBS.

Both sample types showed over 70% protein detectability on the NULISAseq Inflammation Panel 250. **Figure 3A** illustrates the detectability in saliva, where 76% (189 of 250) of proteins were above the LoD in more than 50% of samples. Likewise, 72% (181 of 250) of the panel proteins were detected above the LoD in more than 50% of nasal swabs (**Figure 3B**). These results highlight how NULISA's ultra-high sensitivity in samples with limited volumes can support biomarker discovery and translation in non-invasive oral and nasal specimens.

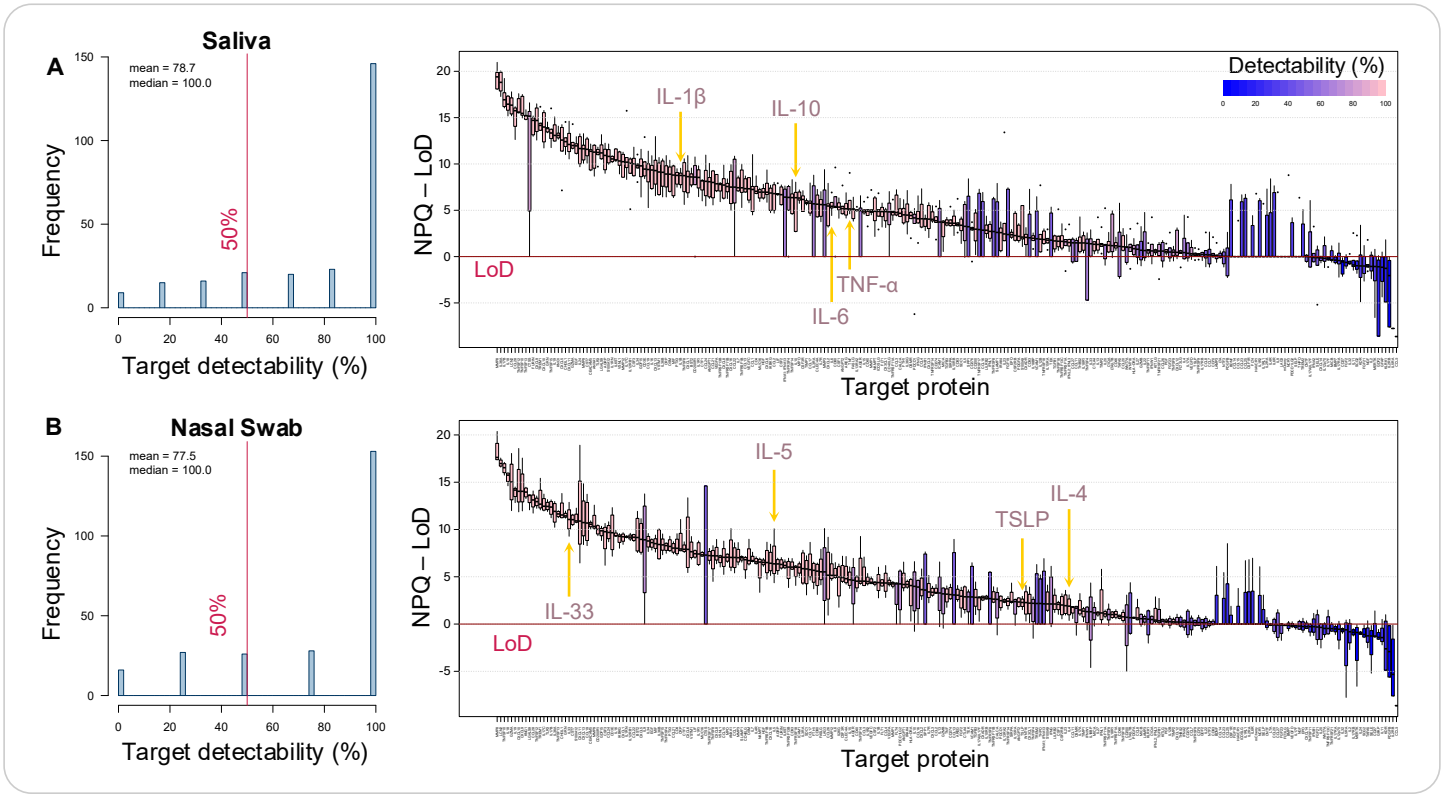


Figure 3. NULISAseq Inflammation Panel 250 achieves > 72% detectability in saliva and nasal swab specimens. Matched adult (A) saliva (n=6) and (B) nasal swab (n=6) samples. Proteins with high and low detectability are shaded pink and blue, respectively. Cytokines of interest with typically low-abundance in saliva or nasal extractions are indicated. The limit of detection (LoD) is delineated in magenta. NPQ = NULISA Protein Quantification.

Stool Extracts: Linking the Gut-Brain Axis with Proteomics

Barbara Bendlin, Ph.D., MA

Professor
University of Wisconsin, Madison School of Medicine
Wisconsin Alzheimer's Disease Research Center

Dr. Bendlin is mapping the multifaceted factors contributing to brain aging, including prediabetes, lifestyle choices, gut microbiome, and socioeconomics. She combines MRI and PET imaging with fluid biomarker analysis to pinpoint brain changes that occur in early-stage Alzheimer's disease.

In addition to her research, Dr. Bendlin leads UW-Madison efforts in neuroscience public policy and is an associate editor of the Journal of Alzheimer's Disease.

The gut-brain axis has been implicated as a physiological driving mechanism in several neurological disorders, including autism spectrum disorder, epilepsy, and Alzheimer's disease. Fecal material thus represents a promising sample matrix for identifying diagnostic biomarkers and drug discovery targets. Furthermore, endoscopic methods for diagnosing inflammatory bowel disease (IBD) are invasive, and stool-based biomarker approaches have found clinical success.[12] Conversely, the complexity of fecal samples, associated matrix effects, and bacterial proteolytic activity complicate fecal proteomics, which is typically measured with less sensitive 2D-DIGE and mass spectrometry.[13] Sensitive and specific methods are needed to robustly identify host-specific proteins of interest.

Prof. Bendlin's group at the University of Wisconsin studies how the link between gut microbiota, inflammation, and gut barrier integrity can contribute to age-related diseases like AD. Calprotectin (i.e., the heterotetramer of S100A8 and S100A9) is an established protein marker of gut inflammation, and the group is interested in measuring additional inflammatory proteins in human feces. The NULISAseq Inflammation Panel 250 was applied to fecal samples from 10 patients with AD and 10 matched controls.

Extracts of 5 µl were chosen to maximize signal while minimizing matrix effects (see page 8, "Tips and Best Practices").

Overall detectability on the NULISAseq Inflammation Panel 250 was 18% with 45 targets detected above the LoD in more than 50% of samples. Given the complexity of fecal samples, such low detectability was not surprising. Nevertheless, 13 proteins achieved 100% detectability, including the calprotectin component S100A9.

A linear regression was applied to the NULISAseq data to identify differential protein markers among AD patients and matched controls. Fecal levels of fibroblast growth factor 2 (FGF2) and the anti-inflammatory cytokine IL-13 were significantly lower ($p<0.05$) in AD patients compared to controls (**Figure 4**). FGF2 and IL-13 are implicated as protective factors in Alzheimer's and dementia.[14,15] Conversely, fecal levels of the pro-inflammatory cytokine IL-16 and extracellular matrix degrader MMP-9 were significantly higher in AD than in control. Circulating IL-16 levels have been shown to moderately differentiate AD patients from controls, while increased fecal MMP-9 and other matrix metalloproteases have emerged as potential diagnostic markers of IBD.[16,17] Notably, S100A9 levels were similar between the two groups.

Despite the challenges of obtaining fecal proteomics data, these results suggest that the NULISA platform can provide biologically meaningful and translational analysis in non-invasive stool samples, reporting on host gut inflammatory status related to neurological, gastrointestinal, and microbiota-influenced disease states.

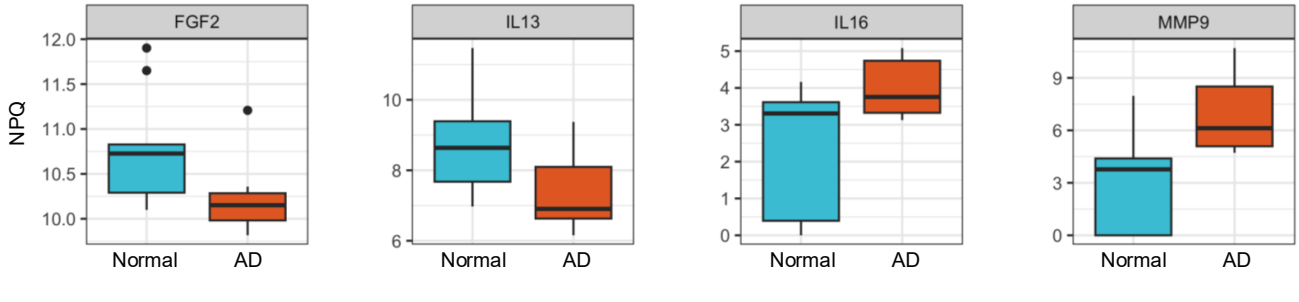
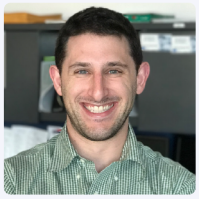


Figure 4. NULISAseq Inflammation Panel 250 fecal proteins with significantly different levels among normal and AD subjects. NPQ = NULISA Protein Quantification.

Brain Homogenates: Optimal Protein Extraction for Biomarker Elucidation



Jonathan D. Cherry, Ph.D.

Assistant Professor, Boston University School of Medicine
U.S. Department of Veterans Affairs

Dr. Cherry focuses on how neuroinflammation and traumatic brain injury contribute to chronic traumatic encephalopathy (CTE). He is particularly interested in how microglia and pro-inflammatory cytokines contribute to hyperphosphorylated tau accumulation and spread.

As a research scientist with the Boston VA, Dr. Cherry also supports the VA-BU-CLF brain bank. He is the director of the digital pathology hub for the BU CTE center, pioneering new methods for postmortem tissue analysis.

Brain tissue is a highly invasive sample type, yet it is routinely collected post-mortem in human and mouse studies.[18] Extracting proteins in aqueous solution from fatty brain tissue poses a challenge, which is addressed by homogenization in buffers that typically include detergents. The commonly used radioimmunoprecipitation assay (RIPA) buffer contains the detergents Triton-X-100, SDS, and the bile acid sodium deoxycholate to solubilize the proteins of interest. While RIPA is a relatively mild buffer, stronger chaotropic agents, such as GHCL, can extract insoluble proteins that are challenging to extract.[19]

To determine the optimal conditions for brain homogenate extraction and analysis using the NULISAseq CNS Disease Panel, a pilot study was conducted in collaboration with Prof. Cherry's research group at Boston University, utilizing 12 post-mortem human specimens. Since the amount of brain tissue can vary, normalization of lysates across different samples based on total protein concentration is essential for a successful study design (see page 8, "Tips and Best Practices"). A standard BCA assay was performed to measure the total protein concentration, and the samples were first normalized to the lowest protein level observed in the BCA assay. Multiple sample dilutions were tested to find the optimal input concentration for the assay. The two extraction buffers exhibited similar overall protein detectability, with 45% (56 of 124) of proteins detected in over 50% of the samples.

Hierarchical clustering analysis revealed that the two buffers produced distinct proteomic profiles, suggesting differences in extraction efficiency. Overall, GHCL extracted insoluble proteins associated with CNS pathology more efficiently, including amyloid beta peptides, phosphorylated tau species, and both monomeric and oligomeric alpha-synuclein (SNCA). Differential expression analysis with false discovery rate adjustment was performed to identify the proteins most effectively extracted into either RIPA or GHCL buffer (**Figure 5**).

Based on these results, the NULISAseq CNS Panel 120 can detect key translatable proteins with high sensitivity and selectivity in brain tissue homogenates. The choice of extraction buffer depends on the markers of interest (see **Figure 5**). Alamar recommends conducting the assay on homogenates diluted to 0.01–0.2 mg/ml total protein.

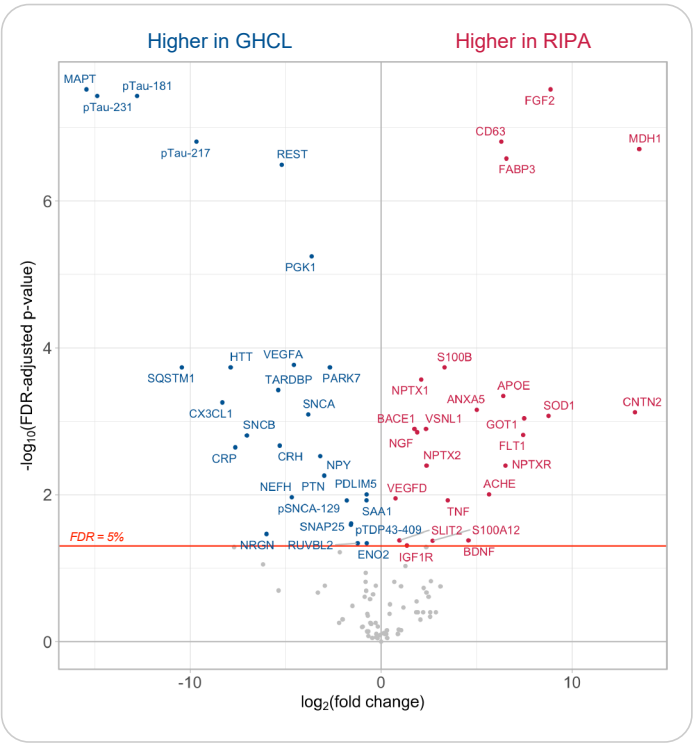


Figure 5. Volcano plot illustrating the extraction efficiencies of GHCL and RIPA buffer for the NULISAseq CNS Panel 120 targets. Proteins labeled in blue displayed higher levels in GHCL, while those labeled in red exhibited higher levels in RIPA. The FDR-corrected p<0.05 cutoff is represented by a red line.

Summary

The NULISA technology has proven highly valuable in plasma, serum, and CSF biomarker studies. While blood-derived sample types are most commonly associated with proteomic analysis, alternative sample types provide additional contexts and non-invasive options for identifying, validating, and tracking translatable biomarkers. However, some of these sample types present significant challenges to efficient and reproducible sample preparation due to numerous variables that need testing and optimization (**see Tips and Best Practices**). This report broadens the range of sample matrices accessible to the NULISA platform and describes experimental parameters to improve study success.

As a new platform, NULISA's capabilities are expanding rapidly. For questions about NULISA technology or its application in specific sample types, contact us at info@alamar.com.

Tips and Best Practices

While the NULISAseq Panels are primarily tuned for plasma and serum, the best-in-class attomolar sensitivity and seven-plus logs of dynamic range make NULISA the ideal platform for measuring alternative specimens. Working with sample types other than plasma and serum poses novel challenges due to the overall lower abundance of proteins, variable amount of starting material, and complex characteristics of the matrix. Please consider the following when using these types of samples.

- **Matrix effects:** Heterogeneous biospecimens contain a multitude of components that can cause assay interference and impede antibody binding to protein targets. "Matrix effects" are particularly challenging for complex matrices such as stool, and working with these samples requires optimizing input volumes to maintain high detectability and minimize interference. With the high sensitivity and low volume requirements of the NULISA platform, testing a dilution series is recommended when working with novel sample types for the first time to determine the optimal input volume for maximizing data quality.

- **Hook effect:** In an immunoassay where the concentration of the analyte significantly exceeds that of the antibody, the apparent assay dose response is reduced, resulting in a lower-than-expected ligand concentration and a "hook"-shaped dose-response curve. Alamar overcomes hook effects by using "cold" antibodies and tuning the panel for highly abundant proteins, thereby maintaining a wide dynamic range of two logs above endogenous levels and one log below endogenous levels for all targets in plasma and serum. For other sample types, the dilutional linearity of the assay should be determined empirically to ensure detection of analytes within the linear range without reaching the "hook" or saturation.

- **Normalization:** Non-traditional biofluids or tissues may contain variable densities of material; therefore, normalization is crucial for accurate analysis and comparison across different conditions. Normalization can be based on total protein concentration (e.g., brain lysates discussed above), as measured by a BCA assay, EV counts through nanoparticle tracking analysis, or the volume of biofluid used for isolation and extraction (e.g., EVs and tear fluid).

- **Have additional questions?** Please contact our support team at *support@alamarbio.com*.

References

1. Feng, W. et al. NULISA: a proteomic liquid biopsy platform with attomolar sensitivity and high multiplexing. Nat Commun 14, 7238 (2023).
2. Dodig-Crnković, T. et al. Facets of individual-specific health signatures determined from longitudinal plasma proteome profiling. EBioMedicine 57, 102854 (2020).
3. Molfetta, G. Di et al. Inflammation biomarkers and Alzheimer's disease: A pilot study using NULISAseq. Alzheimers Dement 17, e70079 (2025).
4. Ibanez, L. et al. Benchmarking of a multi-biomarker low-volume panel for Alzheimer's disease and related dementia research. Alzheimers Dement 21, e14413 (2025).
5. Long, J. P., Singh, S., Dong, Y., Yee, C. & Lin, J. S. Urine proteomics defines an immune checkpoint-associated nephritis signature. J Immunother Cancer 13, e010680 (2025).
6. Gijs, M. et al. Association of tear fluid amyloid and tau levels with disease severity and neurodegeneration. Sci Rep 11, 22675 (2021).
7. Gijs, M. et al. Pre-analytical sample handling effects on tear fluid protein levels. Sci Rep 13, 1317 (2023).
8. Kumar, A. et al. MicroRNA expression in extracellular vesicles as a novel blood-based biomarker for Alzheimer's disease. Alzheimers Dement 19, 4952–4966 (2023).
9. Nieuwland, R. & Siljander, P. R. M. A beginner's guide to study extracellular vesicles in human blood plasma and serum. J Extracell Vesicles 13, e12400 (2024).
10. Sattarov, R. et al. Phosphorylated tau in cerebrospinal fluid-derived extracellular vesicles in Alzheimer's disease: a pilot study. Sci Rep 14, 25419 (2024).
11. Moreno, E. et al. Proteomic snapshot of saliva samples predicts new pathways implicated in SARS-CoV-2 pathogenesis. Clin Proteomics 21, 37 (2024).
12. Vitali, R. et al. Proteomic Analysis Identifies Three Reliable Biomarkers of Intestinal Inflammation in the Stools of Patients With Inflammatory Bowel Disease. J Crohns Colitis 17, 92 (2023).
13. Devan, S. B. S., Ramli, R., Alshehade, S. A., Lim, S. Y. M. & Mamat, N. Comparison of the techniques for isolating immunoassay-suitable proteins from heterogeneous fecal samples. Anal Biochem 698, 115748 (2025).
14. Zhai, W. et al. The fibroblast growth factor system in cognitive disorders and dementia. Front Neurosci 17, 1136266 (2023).
15. Morimoto, K. et al. Expression profiles of cytokines in the brains of Alzheimer's disease (AD) patients, compared to the brains of non-demented patients with and without increasing AD pathology. J Alzheimers Dis 25, 59 (2011).
16. Soomro, S. et al. Predicting disease course in ulcerative colitis using stool proteins identified through an aptamer-based screen. Nat Commun 12, 3989 (2021).
17. Campanelli, L. et al. Blood levels of cytokines highlight the role of inflammation in Alzheimer's disease. Heliyon 11, e41725 (2025).
18. Wagner, J. et al. Medin co-aggregates with vascular amyloid-β in Alzheimer's disease. Nature 612, 123 (2022).
19. Wang, S. et al. A vaccine with Aβ oligomer-specific mimotope attenuates cognitive deficits and brain pathologies in transgenic mice with Alzheimer's disease. Alzheimers Res Ther 9, 41 (2017).

Alamar Biosciences, Inc.
47071 Bayside Parkway Fremont, CA 94538
T +1 (510) 626-9888 | E info@alamarbio.com

Visit **AlamarBio.com** to learn more.

ALAMAR BIOSCIENCES