

Autism Medical & Research Summit 2026 (AMRS)

Translating Biology
into Care for the Most
Complex Autism Profiles



November 5-7
2026
Hotel Meliá,
Sitges
Barcelona
SPAIN

SCIENTIFIC PROGRAM

November 5, 2026 - THURSDAY

Day 1 — From Definitions to Clinical Reality

Measuring severity, understanding complexity, and recognizing crisis drivers in high-support autism

“High support Autism: What We Know, What We Assume, and What We Must Demonstrate”

Abstract: Severe autism represents a heterogeneous but underserved segment of the spectrum (30%+), marked by high support needs, minimal verbal abilities, complex comorbidities, and under-representation in research. This keynote outlines the current conceptual instability around definitions and severity metrics, highlights structural biases in cohorts, and maps the central scientific gaps—biomarkers, mechanisms, longitudinal trajectories, and translational endpoints. The lecture sets the stage for a conference focused on rebuilding methodological foundations and establishing research priorities for high-support autism.

14:00–14:20 — OPENING SESSION

- Welcome & scientific rationale
- Why a meeting focused on severe autism
- Overview of the state-of-the-art paper
- Film screening

14:20–15:00 — OPENING KEYNOTE

“Severe Autism: What We Know, What We Assume, and What We Must Demonstrate”

15:00–15:45 — SYMPOSIUM 1

Rethinking Severe Autism: From ‘high support’ to Profound Autism throughout Definitions, Taxonomy & Measurement”

Abstract: This session examines why the field still lacks a stable and operational definition of “high support/severe/profound” autism. Speakers will address the coexistence of administrative (e.g., Lancet Commission) versus clinical definitions, the scarcity of validated severity cut-offs, and the exclusion of individuals with intellectual disability or minimal verbal ability from most studies and trials. Strategies for building inclusive cohorts, calibrating severity measures, and reporting recruitment flows transparently are proposed. This session provides the conceptual foundation for the entire congress, clarifying why definitions and measurement choices directly shape biological stratification, trial design, and clinical translation.

Talks:

1. **What Do We Mean by “Severe Autism”?** Nosology & Operational Thresholds
2. **Underrepresented Profiles:** Why Minimally/Non-Verbal & ID Individuals Rarely Enter Research Cohorts
3. **Beyond Scores:** Domain-Specific Trajectories & ADOS-CSS Limitations

15:45–16:00 — Transition

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16:00–17:00 — SYMPOSIUM 2

“Clinical Complexity: Pain, Sleep, Sensory Overload & Comorbidities”

Abstract: High support autism frequently overlaps with hidden pain, autonomic dysregulation, aberrant sleep micro-architecture, epilepsy, gastrointestinal symptoms, and medical emergencies that shape functional severity. This symposium integrates findings from neurology, sleep medicine, autonomic neuroscience and internal medicine to illustrate how these factors alter behavior, learning, and adaptive functioning. Approaches for measurement, early detection, and research design are discussed.

Talks:

1. **Invisible Pain & Autonomic Dysregulation:** When Behavior Masks Medical Distress in High-Support Autism
2. **Sleep as a Neurodevelopmental Regulator in Autism:** arousal Instability, and Functional Outcomes
3. **Epilepsy:** Unrecognized Drivers of Functional Severity
4. **ENT Disorders and Upper Airway Infections in Autism:** From Hidden Inflammation to Neuroimmune Triggers

17:00–17:25 — Coffee Break

17:30–18:15 — SYMPOSIUM 3

“Severe Behavioral Phenotypes: Neurobiology of Self-Injury, Autonomic Crises & Aggression”

Abstract: Severe self-injurious behavior (SIB), aggression, and autonomic crisis episodes represent some of the most medically urgent and least understood phenotypes in high-support autism. This symposium examines converging neurobiological pathways—nociceptive dysfunction, endogenous opioid signaling, autonomic instability, neuroinflammatory triggers, and sensory-motor integration abnormalities—that drive extreme behavioral crises. Presentations will explore biomarkers of arousal dysregulation, the role of interoceptive pain abnormalities, early-warning physiological signatures, and the interface between medical distress and behavioral escalation. The session proposes mechanistically anchored frameworks for assessment and outlines research strategies capable of distinguishing behavioral, autonomic, and medical causes of crisis behavior.

Talks:

1. **Pathophysiology of Self-Injury:** Pain, Opioid Systems & Arousal Dysregulation
2. **Autonomic Instability & Aggressive Outbursts:** Mechanistic Underpinnings
3. **Sensory-Motor Drivers of Crisis Behavior:** Early-Warning Signals & Biomarkers

18:15–18:20 — Transition

18:20–19:05 — ASSOCIATION ANCHORING SESSION

“Why Biomedical Research Matters: Perspectives from Autism Organizations”

Abstract: Autism organizations outline the lived impact of diagnostic delays, unmet medical needs, sensory and behavioral crises, and fragmented services. Their perspective frames why mechanistic, biologically grounded research is essential to improve both care and long-term outcomes. The session anchors the congress in real-world priorities and underscores the urgency of translational science for high-support autism.

Talks:

1. **Clinical Complexity Through Lived Experience:** Patient and Caregiver Perspectives

19:05–19:10 — Transition

19:10–19:35 — POSTER AREA OPENING & NETWORKING

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November 6, 2026 - FRIDAY

Day 2 — Biology Becomes Study Design

From mechanisms to stratified trials and data standards

Morning — Neuroimmune Foundations

09:00–09:30 — PLENARY LECTURE

“Neuroimmune & Redox Mechanisms in high support Autism: Microglia, BBB Dysfunction”

Abstract: Emerging evidence highlights altered microglial activation, cytokine shifts, blood–brain barrier permeability changes, mitochondrial dysfunction, and oxidative stress signatures in subsets of individuals with severe autism. This plenary synthesizes neuroimmune, metabolomic and neuropathological findings, clarifying what is replicable and what remains speculative, and pointing toward mechanistic axes amenable to biomarker development and therapeutic targeting.

09:30–09:35 — Transition

09:35–10:35 — SYMPOSIUM 4

“Inflammation & Oxidative Stress: Linking Peripheral Signatures to CNS Pathophysiology”

Abstract: The session explores how peripheral immune and redox markers relate—or fail to relate—to central neuroinflammation. Speakers will examine inconsistencies across cytokine studies, the role of mitochondrial/oxidative pathways, and the challenge of integrating peripheral signatures with CNS imaging or CSF data. Methodological routes to strengthen periphery-to-brain inference are presented.

Talks:

1. **Microglia & Cytokine Dysregulation:** Robust Evidence vs Unknowns
2. **Mitochondrial Dysfunction & Oxidative Stress:** Mechanistic Pathways
3. **Blood–Brain Barrier as a Gatekeeper of Neurodevelopment:** Inflammation, Immune Trafficking, and Barrier Instability in Autism
4. **The Glymphatic system:** Emerging Hypotheses, Sleep-Dependent Clearance and Neurodevelopmental Vulnerability

10:35–11:00— Coffee Break

11:00–12:15 — SYMPOSIUM 5

“Neuroimmune Dysregulation, Autoimmunity & Encephalopathies in high support Autism”

Abstract: Increasing evidence suggests that a substantial subset of individuals with high-support autism exhibit neuroimmune abnormalities, autoimmune mechanisms, or intermittent encephalopathic processes. This symposium addresses the growing recognition of immune-mediated phenotypes—ranging from autoimmune encephalitis to systemic inflammatory states—that manifest as regression, agitation, catatonia-like features, cognitive fluctuation, and profound behavioral change. Speakers will review diagnostic red flags, immunological biomarkers, neuroimaging findings, and the role of microglia, astroglia, and cytokine dysregulation. The session discusses clinical decision pathways for MRI/EEG/CSF/autoantibody workups and clarifies current evidence regarding molecular mimicry, PANS/PANDAS overlap, and immune-triggered neurobehavioral deterioration.

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Talks:

1. Inborn errors of immunity present with neuropsychiatric symptoms: **overlap with autistic behavioral symptoms**
2. Autoimmune & Neuroinflammatory Encephalopathies: Red Flags & Diagnostic Workflows
3. Systemic Immune Dysregulation, Microglia & Astroglia: Behavioral & Cognitive Impact
4. Molecular Mimicry, Antibodies & Immune-Triggered Regression: Evidence & Gaps
5. Autoimmunity and Arthritis in Youth with Autism and Suspected Post-Infectious Deteriorations

12:15–12:20 — Transition

12:20–13:00 — DIAGNOSTIC PANEL

“When Immune Disorders Present as Behavioral Crises in Severe Autism”

Behavioral crises in severe autism often mask underlying medical or immune-driven processes. This expert panel examines cases in which immune dysregulation, neuroinflammation, autonomic instability, or metabolic encephalopathy present primarily as acute agitation, aggression, mutism, regression, or catatonia-like episodes. Through interdisciplinary perspectives from neurology, psychiatry, immunology, emergency medicine, and developmental pediatrics, the session outlines actionable diagnostic pathways that distinguish behavioral from medical emergencies. Panelists will discuss when to initiate MRI/EEG/LP evaluations, which laboratory markers to prioritize, how to interpret atypical presentations, and how emergency protocols should adapt for minimally or non-verbal individuals. Practical recommendations for acute stabilization and follow-up care will be provided.

Panelists: Neurology, Psychiatry, Immunology, Emergency Medicine, Developmental Pediatrics

Topics:

- Immune-triggered agitation & regression
- Autonomic crises & catatonia-like states
- When to escalate to MRI, EEG, LP, autoantibodies panels
- Emergency protocols

13:00–15:00 — Lunch & Poster Viewing & networking

Afternoon — Therapies, Trials & Translation

15:00–15:45 — SYMPOSIUM 6

“Immunomodulatory & Anti-Inflammatory Treatments: Evidence, Gaps & Trial Standards”

Abstract: This symposium reviews current evidence for immunomodulatory and anti-inflammatory interventions in high-support autism, including steroids, IVIG, metabolic and redox-modulating agents, and microbiome-based therapies. Special attention is given to Microbiome Transfer Therapy (MTT/FMT), supported by a growing body of research—including completed Phase 1 and Phase 2 clinical trials and an ongoing Phase 3 study—demonstrating benefits across gastrointestinal, immune, metabolic, and behavioral domains. The session examines mechanistic rationales, trial outcomes, responder subgroups, and the biomarkers needed to guide inclusion criteria, harmonize outcome measures, and ensure safety and reproducibility in next-generation clinical trials.

Talks:

1. **IVIG in Severe Autism:** Signals, Inconsistencies & RCT Requirements
2. **Anti-Inflammatory & Immunomodulatory Phenotypes:**
Steroid Responsiveness + NAC, Sulfuraphane, Minocycline, Omega-3
3. **Microbiome Transfer Therapy (MTT/FMT):** GI–Immune Mechanisms & Trial Lessons

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15:45–15:50 — Transition to Omics session

15:50–16:35 — SYMPOSIUM 7

"Genomics, Epigenomics & Global Data Resources: from variants to stratification"

Abstract: Although many cases are labelled "non-genetic," advances in whole-exome/genome sequencing and epigenomic profiling increasingly reveal oligogenic, regulatory, and rare variant contributions. This symposium clarifies the porous boundary between idiopathic and genetically mediated autism, the relevance of CNVs and non-coding architecture, and the role of gene–environment interactions. Implications for classification and mechanism-based trials are discussed.

Talks:

1. Rare variants, CNVs and non-coding architecture in high-support autism
2. Epigenetics & gene–environment interaction (maternal immune activation, exposome)
3. Building a global genetics resource: harmonisation, FAIR metadata and phenotype linking

16:35–16:40 — Transition

16:40–17:10 — SYMPOSIUM 8

"Pharmaco-Omics & Long-Term Safety in Severe Autism"

Abstract: Individuals with severe autism often receive complex combinations of antipsychotics, antiepileptics, and sedative agents, with variable efficacy and heavy adverse events. This session introduces pharmacometabolomics, therapeutic drug monitoring, and polypharmacy network models that can improve prediction of response and metabolic safety. Clinical and ethical challenges in this population are highlighted.

Talks:

1. Psychotropics & Antiepileptics: Heterogeneous Response Profiles
2. Polypharmacy Networks & Drug–Metabolome Interactions

17:10–17:15 — Coffee Break

17:15–18:00 — SYMPOSIUM 9

"Metabolomics: The Metabolic Dimension of High-Support Autism"

Abstract: This symposium explores metabolomics as a translational tool to identify systemic metabolic signatures associated with high-support autism, with a particular focus on the gut–brain–immune–metabolic axis. Speakers will examine how gastrointestinal dysfunction and altered nutrient processing shape circulating metabolic profiles, influence inflammatory tone, and modulate neurodevelopmental trajectories. The role of **anti-inflammatory dietary patterns** and targeted nutritional strategies will be discussed as supportive interventions capable of modifying metabolic and inflammatory states, while acknowledging current evidence gaps and methodological challenges. Overall, the session frames metabolism not merely as a downstream consequence, but as a **modifiable biological layer** that connects environment, gut function, inflammation, and neurodevelopment, with direct implications for research design, clinical monitoring, and precision-oriented supportive care.

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Talks:

1. **When Diet Reflects Gut Dysfunction:** Restrictive Eating, Gut-brain axis and Metabolic Consequences in Severe Autism
2. Anti-Inflammatory Nutrition as a Supportive Strategy: **Dietary Modulation of Inflammation and Metabolic Balance**
3. Metabolic Signatures of High-Support Autism: **From Systemic Profiles to Phenotypic Stratification**

18:00–18:05 — Transition

18:05–18:50 — SYMPOSIUM 10

“AI in High-Support Autism From Real-World Data to Clinically Actionable Biomarkers”

Abstract: In high-support autism, clinical complexity may exceed the capacity of traditional analytic approaches. Artificial intelligence offers powerful tools to integrate heterogeneous data, yet current models frequently fail to translate into clinical benefit due to biased cohorts, limited interpretability, and exclusion of the most complex phenotypes. This symposium explores a clinically grounded approach to AI in autism, focusing on real-world data, biologically informed stratification, and meaningful outcomes. By connecting AI methods to neurodevelopmental mechanisms, neuroimmune and metabolic pathways, and longitudinal trajectories, the session outlines a roadmap toward robust, explainable, and clinically actionable AI. The goal is not “more AI,” but better AI—capable of supporting patients, diagnosis, stratification, and trial design in the most underserved autism populations.

PLENARY LECTURE — “AI You Can Trust: From Mechanism-Based Cohorts to Predictive Biomarkers”

Talks:

1. **Minimal Datasets, Maximum Insight:** Leveraging Real-World Data to Build Trustworthy Clinically Useful AI in Autism
2. **When Data Are Too Much for Humans:** Neural Models, Biomarkers, and Clinical Stratification in Complex Neuroimmune Disorders

18:50–18:55 — Transition

18:55–19:30 — RESEARCH INCUBATOR

(Non-competition session): Overview of multicentre opportunities and how to join upcoming consortia (no pitches).

Abstract: A structured overview of open multicenter initiatives, novel datasets under development, and collaborative opportunities. Speakers will outline how research groups can join, share standardized data, and contribute to next-generation mechanistic trials.

MiniTalks:

1. **From research idea to scientific article:** keys to researching ASD from a clinical perspective

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November 7, 2026 - SATURDAY

Day 3 — Clinical Translation & Future Directions

Turning Complexity into Actionable Pathways

09:00–09:30 — PLENARY LECTURE

“Severe Autism Across the Lifespan: Neurology, Medicine & Predictive Care”

Abstract: This lecture reconnects pediatric, adolescent, and adult trajectories, focusing on medical vulnerability, risk prediction, emergency pathways, and long-term health outcomes. It integrates neurological, medical, and service-level data to propose a life-course framework for high-support autism

09:30–09:35 — Transition

09:35–10:35 — SYMPOSIUM 11

“Adult Outcomes & Aging in Severe Autism: Medical Trajectories and Coordinated Models of Care”

Abstract: As individuals with severe and high-support autism transition into adolescence and adulthood, clinical complexity often increases, while access to coordinated medical care decreases. Adult outcomes in this population are shaped not only by neurodevelopmental features, but by the cumulative impact of metabolic vulnerability, endocrine changes, chronic inflammation, sleep disorders, long-term pharmacological treatments, and barriers to healthcare access. This symposium addresses the medical trajectories of aging in severe autism, moving beyond a purely neurodevelopmental framework to a lifespan-oriented medical model. Speakers will examine emerging evidence on cardio-metabolic risk, highlighting the interplay between oxidative stress, sleep disruption, psychotropic and antiepileptic medications, and chronic immune activation. The role of endocrine health—from puberty through adulthood—will be explored as a frequently overlooked determinant of functional outcomes, behavioral stability, and medical morbidity. The session will further discuss the evolving needs of adults with high support requirements, emphasizing the importance of coordinated, multidisciplinary models of care that bridge pediatric and adult services. Particular attention will be given to emergency medicine in non- and minimally verbal individuals, where atypical presentations, communication barriers, and diagnostic delays contribute to increased risk and preventable morbidity.

Talks:

1. **Metabolic Risk in High-Support Autism** *Oxydative stress, Medications, Sleep and Inflammation Across the Lifespan*
2. **From Puberty to Aging:** *Why Endocrine Health Becomes a Hidden Determinant of Outcomes in Severe Autism*
3. **Beyond Neurodevelopment:** *Adults with High Support Needs*
4. **Emergency Medicine for Non-/Minimally Verbal Individuals**

10:35–10:40 — Transition

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10:40–11:10 — PANDAS and PANS as neuroimmune model & PANS/PANDAS documentary

Abstract: Pediatric Acute-onset Neuropsychiatric Syndrome (PANS) and Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections (PANDAS) describe acute neuropsychiatric syndromes characterized by sudden onset or rapid exacerbation of behavioral and cognitive symptoms following infectious or inflammatory triggers. While distinct from autism spectrum disorder, these conditions provide a valuable neuroimmune model to explore mechanisms of acute functional decompensation in vulnerable neurodevelopmental systems. This talk examines what PANS/PANDAS can teach us about high-support autism phenotypes, particularly those marked by abrupt behavioral worsening, regression, or crisis presentations. By focusing on overlap and boundaries, we aim to highlight shared biological themes. The session will discuss how PANS/PANDAS exemplify a dynamic, time-sensitive model of severity—where acute immune-mediated perturbations translate into rapid loss of function, often misinterpreted as primary psychiatric or behavioral deterioration. These insights are especially relevant for minimally verbal and intellectually disabled individuals, in whom pain, infection, and inflammation frequently manifest as behavioral escalation.

Talks:

1. **Lessons from PANS/PANDAS for High Support Autism Phenotypes: Neuroimmune Overlap, Acute Decompensation, and Conceptual Boundaries**
2. **PANS/PANDAS talk (Tbd)**

11:15–11:45— Coffee Break

11:45–12:25 — PANS/PANDAS PANEL

- Clinicians
- Researchers
- Associations
- Policy/health system reps

12:30–13:30— CLOSING PANEL

“What Should Change Tomorrow? Priorities for Severe Autism Medicine & Research”

- Clinicians
- Researchers
- Associations
- Policy/health system reps

Abstract: This closing panel brings together clinicians, researchers, associations, sponsors and health-system leaders to define the most urgent next steps for transforming medical care and research in severe autism. Panelists will identify high-impact opportunities in biomarker development, neuroimmune and multi-omic research, cohort design, trial methodology, safety monitoring, emergency medicine, and coordinated care models. Discussions will focus on actionable strategies—what can be implemented immediately, what requires structural change, and what long-term investments are needed to shift outcomes for individuals with the highest medical and support needs. The session aims to synthesize conference insights into a unified roadmap for scientific, clinical, and health-policy progress.

13:35 –14:00 — BEST ABSTRACT/POSTERS AWARDS

Recognition of the most impactful posters based on scientific rigor, innovation, and translational potential.