

INTERNATIONAL EDITION

# Autism Medical & Research Summit

**AMRS 2026**

**November 5-7 2026**  
Barcelona, Sitges, Spain



# Autism Medical & Research Summit

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## SCIENTIFIC PROGRAM

November 5th, 2026 - FRIDAY

### Day 1 — From Definitions to Clinical Reality

*Redefining severity in high support autism and examining clinical complexity, comorbidities and crisis drivers*

#### “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

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#### 14:20–15:00 — OPENING KEYNOTE

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

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#### 15:00–15:30 — 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. **Diagnosing High-Support Autism in Practice:** Clinical Approaches, Constraints, and Unmet Needs
2. **Underrepresented Profiles:** Why Minimally/Non-Verbal & ID Individuals Rarely Enter Research Cohorts

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15:30–15:35 — Transition

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15:35–16:55 — SYMPOSIUM 2

*“Clinical Complexity: Pain, Sensory Impairments & 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, 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. **Psychiatric Symptoms or Neuroimmune Dysregulation?** A Clinical Framework for Interpreting Behavioral Change in Severe Autism
2. **Sensory Impairment in Autism**
3. **Epilepsy:** Unrecognized Drivers of Functional Severity
4. **ENT Disorders and Upper Airway Infections in Autism:** From Hidden Inflammation to Neuroimmune Triggers

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17:00–17:25 — Coffee Break

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17:30–18:10 — 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.

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### Talks:

1. **Self-Injurious Behavior and Pain in Severe Autism:** Current Hypotheses on Pain Perception and Defensive Responses
2. **Behavioral Crises in Severe Autism:** Hypothesized Links to CNS Hyperresponsivity and Sensory Dysregulation

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18:10–18:15— Transition

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### 18:15–19:35 — 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:** Caregiver Perspective
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18:35–18:40 — Transition

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### 18:40–19:20 — POSTER AREA OPENING & NETWORKING

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## SCIENTIFIC PROGRAM

November 6th, 2026 - FRIDAY

### Day 2 — Biology Becomes Study Design

Exploring neuroimmune, genetic and metabolic mechanisms and biomarker identification

Morning — Neuroimmune Foundations

#### 09:00–09:25 — 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.

#### 09:25–09:30 — 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
- 5.

#### 10:50–11:20— Coffee Break

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### 11:20–13:00 — 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.

#### Talks:

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

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### 13:10–13:15 — Transition

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### 13:15–13:45 — 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

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### 13:45–15:00 — Lunch & Poster Viewing & networking

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### 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:

**IVIG in Severe Autism:** Signals, Inconsistencies & RCT Requirements

**Anti-Inflammatory & Immunomodulatory therapies**

**Microbiome Transfer Therapy (MTT/FMT):** GI-Immune Mechanisms & Clinical Trials Lessons

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#### 15:45–15:50 — Transition

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#### 15:50–16:50 — SYMPOSIUM 7

##### *"Genetics & Epigenetics in High-Support Autism: From Variants to Stratified Mechanisms"*

**Abstract:** While many individuals with high-support autism are historically classified as "non-genetic," advances in whole-exome/genome sequencing and epigenomic profiling reveal oligogenic, regulatory, and rare variant contributions that inform disease mechanisms. This expert panel will discuss the blurred boundary between idiopathic and genetically mediated autism, the role of CNVs, non-coding regulatory regions, and gene–environment interactions, including maternal immune activation and environmental exposures. Panelists will explore how these insights can inform classification, stratified trial design, biomarker discovery, and global research collaboration. Harmonization, FAIR metadata, and phenotype linking strategies for building a worldwide genetics resource will also be addressed.

**Panelists:** Geneticist, Clinical geneticist, molecular biologist, developmental Pediatrics

#### Panelists / Topics:

- Epigenetic Mechanisms & Gene–Environment Interaction (maternal immune activation, exposome)
- Building a Global Genetics Resource: Data Harmonization, FAIR Metadata, and Phenotype Linking
- Implications for Classification, Stratification, and Mechanism-Based Trials

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16:50–17:20 — Coffee Break

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17:20–18:20 — SYMPOSIUM 8

*“Metabolomics: The Metabolic Dimension of High-Support Autism”*

**Abstract:**

*This symposium proposes metabolism as a central, integrative biological dimension of high-support autism rather than a secondary or epiphenomenal finding. The session opens with a state-of-the-art lecture synthesizing current evidence on systemic metabolic alterations in autism, including amino acid dysregulation, energy metabolism abnormalities, redox imbalance, and gut-derived metabolic signals, with a focus on findings most relevant to individuals with severe and high-support needs. The lecture is followed by an expert panel discussion addressing key unresolved and clinically relevant questions in the field. Topics include lessons learned from rare but treatable inborn errors of metabolism presenting with autism-like phenotypes (such as BCKDK deficiency), the reproducibility and specificity of reported metabolic signatures in autism, the role of mitochondrial dysfunction in neurodevelopmental vulnerability and functional severity, and competing hypotheses linking metabolism to immune activation, neuroinflammation, and brain energetics. Panelists will critically examine methodological challenges, sources of heterogeneity, and the limits of current evidence, while identifying metabolic pathways that may serve as biomarkers, stratification tools, or targets for supportive and disease-modifying interventions.*

**Talks:**

1. **Metabolism in High-Support Autism:** Current Evidence, Replicable Findings and Open Questions

17:40–18:20 — EXPERT PANEL

*Abstract: An expert discussion on lessons from inborn errors of metabolism (e.g., BCKDK deficiency), autism-associated metabolic signatures, mitochondrial dysfunction, and competing models linking metabolism, immunity, and neurodevelopment.*

*Panelists:*, Pediatrics, pediatric neurologist, Biomedical scientist, nutritionist.

18:30–18:45

**Talk:**

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

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## SCIENTIFIC PROGRAM

November 7th, 2026 - SATURDAY

### Day 3 — Clinical Translation & Future Directions

*Translating emerging evidence into clinical care models, emergency pathways and research priorities.*

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#### 09:00-09:30 — PLENARY LECTURE

##### *Rethinking Clinical Service Models in Severe Autism: From Symptom Management to Outcome Optimization*

**Abstract:** This lecture examines how current clinical service models for individuals with severe and high-support autism can be restructured to move beyond reactive symptom management toward **proactive, outcome-oriented care**. It addresses gaps in continuity across pediatric, adolescent, and adult services, with attention to medical complexity, care coordination, crisis prevention, and long-term health trajectories. By integrating neurological, medical, and service-level perspectives, the session proposes **conceptual frameworks for redesigning care pathways**, including anticipatory monitoring, risk stratification, and cross-disciplinary collaboration, with the goal of improving clinical outcomes and quality of life across the lifespan.

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#### 09:30-09:35 — Transition

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#### 09:35-10:15 — SYMPOSIUM 10

##### *Rethinking Clinical Service Models in Severe Autism: From Symptom Management to Outcome Optimization*

**Abstract:** This lecture examines how current clinical service models for individuals with severe and high-support autism can be restructured to move beyond reactive symptom management toward proactive, outcome-oriented care. It addresses gaps in continuity across pediatric, adolescent, and adult services, with attention to medical complexity, care coordination, crisis prevention, and long-term health trajectories. By integrating neurological, medical, and service-level perspectives, the session proposes conceptual frameworks for redesigning care pathways, including anticipatory monitoring, risk stratification, and cross-disciplinary collaboration, aiming to improve clinical outcomes and quality of life across the lifespan.

#### 09:35-10:35 — EXPERT PANEL

##### *Optimizing Clinical Care for High-Support Autism: Emergency Management, Hidden Medical Drivers, and Systemic Solutions*

**Abstract:** Individuals with high-support autism frequently present with acute medical and behavioral crises, yet current healthcare systems are often ill-equipped to respond effectively. This expert panel combines insights on emergency medicine for non-/minimally verbal individuals and the recognition of hidden medical drivers of severity. Panelists will discuss systemic gaps, limitations, and innovative strategies from diverse healthcare settings across Europe and the USA, including hospital-based and outpatient models. Key themes include autism-informed

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*emergency protocols, multidisciplinary coordination, risk anticipation, continuity of care, and scalable approaches to improve clinical outcomes for this underserved population.*

**Panelists:** Podiatrist, neurologist, immunologist, psychologist

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### 10:15–10:20 — Transition

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### 10:20–11:00 — PANDAS and PANS as neuroimmune model

**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 with autism, Acute Decompensation, and Conceptual Boundaries
2. **Recognizing PANS/PANDAS Flares in Severe Autism:** Clinical Red Flags, Atypical Presentations, and Diagnostic Challenge

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### 11:05–11:35 — Coffee Break

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### 11:35–12:15 — PANS/PANDAS PANEL

**Abstract:** This expert panel uses real-world clinical cases to examine how PANS and PANDAS present, evolve, and are managed across different healthcare settings. Through case-based discussion, panelists will explore acute neuropsychiatric deterioration, diagnostic uncertainty, and the overlap with severe autism phenotypes, particularly in minimally verbal or intellectually disabled individuals.

The session will highlight key clinical red flags, atypical presentations, and common pitfalls in recognizing immune-mediated neuropsychiatric syndromes. Experts from clinical, research, and advocacy backgrounds will discuss

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decision-making processes around diagnostic workups (including infection triggers, immune markers, and neuroimaging), as well as variability in treatment approaches and access to care across regions. In addition, the panel will address broader system-level challenges, including lack of consensus guidelines, disparities between countries, and barriers to timely diagnosis and intervention.

*Panelists: Podiatrist, neurologist, immunologist, psychologist, researchers, caregivers.*

### 12:20–13:15— CLOSING PANEL

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

**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.

*Panelists: Podiatrist, neurologist, immunologist, psychologist, researchers, advocates, caregivers.*

### 13:20 –14:00 — BEST POSTERS AWARDS & CLOSING

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