**Interview with Dr. Ken Alibek**

**For Romanian families who may not know you yet, could you briefly introduce yourself and tell us how you came to work with children with autism and neurodevelopmental disorders?**

I am a physician, microbiologist, and researcher with many years of experience studying infectious diseases, immunology, and the biological mechanisms of complex chronic illnesses. Early in my career, I focused primarily on dangerous infectious agents and biodefense. Over time, my attention shifted from acute infections to persistent infections, chronic inflammation, immune dysfunction, and their possible roles in cancer, neurological disorders, and other long-term conditions.

I have also held senior medical and academic leadership roles. In my home country of Kazakhstan, I led large oncology centers and served as head of a National Medical Holding that included six therapeutic and diagnostic centers. I also served as vice dean for medical research at Kazakhstan's leading university medical school.

My involvement with autism began many years ago, after my daughter was diagnosed with autism and received inappropriate care at one of the most prominent university medical schools in the United States. During that experience, I encountered many children whose behavioral diagnoses did not fully explain their medical problems.

Many of these children had recurrent infections, gastrointestinal disturbances, immune abnormalities, inflammatory markers, sleep disorders, fatigue, allergies, developmental regression, or neurological symptoms. As I began evaluating them more comprehensively, I concluded that autism—at least in a substantial subgroup—cannot always be understood solely as a behavioral or purely genetic condition.

**Approximately how many children have you evaluated or treated so far, and what does your practice look like today? Do you also work with international patients through online consultations?**

Over the years, I have evaluated or participated in the management of more than 1,500 children with autism and related neurodevelopmental conditions. My broader clinical databases contain information from an even larger group of children, although not every child received the same level or duration of treatment.

I have worked with families from the United States, Western and Eastern Europe, Central Asia and other regions. Much of this work has been conducted through telemedicine and online consultations, particularly when families live in countries where access to specialized neuroimmune evaluation is limited

**When a child diagnosed with autism comes to you, what do you investigate beyond the behavioral diagnosis?**

I begin by asking whether the behavioral diagnosis adequately explains the child's entire clinical picture. Autism describes a pattern of communication, social and behavioral differences, but it does not necessarily identify the biological processes producing those symptoms.

I review the prenatal and birth history, developmental trajectory, timing of regression, previous infections, vaccination and medication history, gastrointestinal symptoms, sleep, allergies, skin problems, seizures, motor abnormalities, recurrent fever, pain and changes following illness.

Depending on the individual case, laboratory investigation may include blood counts, liver and kidney function, nutritional and metabolic markers, thyroid and other endocrine parameters, immunoglobulins, lymphocyte populations, inflammatory markers, selected cytokines, autoimmune markers and appropriately chosen infectious studies. The investigation must be targeted. I do not believe that every child should automatically undergo every available test.

**How do you understand autism today, based on what you have seen in your clinical practice?**

I see autism as a heterogeneous syndrome rather than one disease with one cause. Different biological pathways may eventually produce a similar behavioral diagnosis.

In some children, genetic or developmental factors may predominate. In others, prenatal inflammation, congenital or later infection, immune dysregulation, autoimmunity, metabolic dysfunction, microbiome disruption, nutritional deficiency or environmental exposure may contribute. Several of these factors can coexist and reinforce one another.

My clinical hypothesis is that a biologically identifiable and potentially treatable subgroup exists within the much larger autism spectrum. This does not mean that infection or inflammation explains every case, and it does not mean that autism should be reduced to a single infectious cause.

**What are the most common medical or biological abnormalities you find in the children you evaluate?**

The patterns vary, but frequently observed abnormalities include recurrent or persistent infections, altered immunoglobulin levels, atypical lymphocyte populations, inflammatory changes, autoimmune markers, gastrointestinal dysbiosis, nutritional deficiencies, oxidative stress and disturbances in thyroid, adrenal or other metabolic functions.

Clinically, I often see gastrointestinal problems, disturbed sleep, chronic fatigue, unusual reactions to infections, frequent respiratory or urinary infections, allergies, skin manifestations, irritability, anxiety, obsessive-compulsive symptoms, tics, attention difficulties and episodic worsening of behavior.

None of these findings is specific to autism. Their importance lies in the pattern they form in an individual child and whether that pattern corresponds to clinical history.

**Infections appear to play an important role in your approach. Which infections do you investigate most frequently and why?**

Testing depends on the child's history and geographical exposure. I may consider herpes-family viruses such as cytomegalovirus, Epstein–Barr virus, herpes simplex viruses, varicella-zoster virus and HHV-6. In selected cases, I also investigated *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, streptococcal infection, *Toxoplasma gondii*, *Borrelia* species and other regionally relevant pathogens.

I pay particular attention to congenital infections and infections associated with neurological injury, immune dysregulation or postinfectious autoimmune phenomena. However, screening must be clinically directed. A long panel of positive antibodies without a compatible history does not establish causation.

**Your interpretation of infectious testing sometimes differs from conventional practice. Why can IgG or IgM antibodies be clinically relevant to you even when PCR is negative?**

PCR and serology answer different questions. PCR attempts to detect nucleic acid from the organism in the particular sample collected at a particular moment. A negative blood PCR means that the target was not detected in that specimen; it does not always establish that the organism is absent from every tissue or that no infection occurred previously.

IgG usually indicates previous exposure and can persist for many years. IgM may suggest recent infection, but it can also persist, cross-react or occasionally produce a false-positive result. Therefore, neither IgG nor IgM should be interpreted in isolation.

Serology becomes potentially informative when it is consistent with the clinical history, organism biology, exposure, symptom pattern and other laboratory findings. It may also guide further investigation, but it does not automatically justify antimicrobial treatment.

**How do you distinguish antibodies reflecting a past infection from those that may indicate an ongoing, persistent or reactivated infection? What makes an IgG or IgM result clinically meaningful enough to treat?**

This is one of the most difficult areas of clinical interpretation. A single positive IgG result generally establishes exposure, not active infection. I look for additional evidence: changes in antibody levels in paired samples, antibody avidity when appropriate, antigen or molecular testing, compatible organ involvement, inflammatory or immune changes, recent clinical deterioration and exclusion of more probable explanations.

The meaning also differs substantially among pathogens. For some infections, serology is useful; for others, it is unreliable for determining activity. Persistent IgM must be interpreted cautiously because it may reflect prolonged immune response, cross-reactivity or assay interference rather than active infection.

I would not recommend treatment solely because an IgG value is high. Treatment requires a coherent clinical and laboratory picture, an organism for which effective therapy exists, a reasonable expectation of benefit and a favorable assessment of risk.

**When you consider an infection clinically relevant, how do you decide how to treat it and for how long?**

The choice depends on the organism, suspected site of infection, acute versus persistent presentation, immune status, age, body weight, organ function, previous treatment and medication safety. I also consider whether there is recognized clinical guidance for that infection.

Treatment duration cannot be standardized across all children or pathogens. Some infections require a defined conventional course; others require reassessment after an initial phase. I monitor clinical response, blood counts, liver and kidney function and other medication-specific safety parameters.

If evidence supporting ongoing infection is weak or the risks exceed the expected benefit, I do not believe prolonged antimicrobial therapy should be continued simply because a child has autism.

**What happens when infection is only part of the problem and the immune system itself has become dysregulated? How do you identify these cases?**

Sometimes the initiating infection is no longer the only problem. The immune system may remain activated, suppressed or poorly regulated after the original trigger. In these cases, eliminating or suppressing a pathogen may not be sufficient.

I look for recurrent infections, unusual inflammatory reactions, autoimmune manifestations, abnormal immunoglobulins, altered lymphocyte subsets, persistent inflammatory markers, allergies and symptom exacerbations following infections. The history may reveal a relapsing pattern in which neurological or psychiatric symptoms worsen each time the child becomes ill.

These cases often require collaboration among pediatrics, immunology, infectious disease, neurology and, when appropriate, rheumatology.

**How do you approach autoimmunity when the immune response is directed against the body's own structures, particularly the nervous system?**

First, I try to establish whether autoimmunity is genuinely likely rather than assuming that every neurological symptom is autoimmune. I review the timing of symptoms, infection-related exacerbations, neurological examination, validated autoimmune markers and evidence of systemic inflammation. When necessary, I recommend consultation with a pediatric neurologist, immunologist or rheumatologist.

Treatment should address any identifiable trigger while also controlling a harmful immune response. The strength of the evidence and the severity of the child's condition determine how aggressive the intervention should be. More

intensive immunotherapies require specialist supervision and careful risk–benefit assessment.

**What forms of immunomodulation do you use in your practice, and what determines which approach is appropriate for a particular child?**

Immunomodulation is a broad concept. It may begin with correcting vitamin and mineral deficiencies, improving sleep and gastrointestinal health, reducing inflammatory triggers and treating confirmed infections. In selected children, anti-inflammatory, antihistamine or other prescription immunomodulatory approaches may be considered.

The choice depends on whether the immune system appears overactivated, suppressed or misdirected; whether autoimmunity is suspected; and whether an infectious trigger remains present. Potent therapies such as corticosteroids, intravenous immunoglobulin or other specialist immunotherapies should be considered only for appropriately selected cases under qualified medical supervision. They are not routine treatments for autism itself.

**How do you balance treating an infectious trigger with modulating the immune response when both appear to be involved?**

The order and intensity of treatment matter. Excessively suppressing immunity in the presence of uncontrolled infection can be dangerous, while treating only the infection may be insufficient if a self-sustaining inflammatory or autoimmune process has developed.

I try to identify the dominant process and choose the safest starting point. Treatment may proceed step by step, or carefully selected interventions may be combined. Close monitoring is essential, because changes in symptoms or laboratory results may require adjustments to the plan.

**Where do PANS/PANDAS, autoimmunity and neuroinflammation fit into your understanding of children diagnosed with autism or other neurodevelopmental disorders?**

PANS and PANDAS demonstrate that infections and immune responses can be associated with abrupt neuropsychiatric changes in susceptible children. They are particularly relevant when symptoms such as obsessive-compulsive behavior, food restriction, tics, anxiety, emotional instability or developmental regression appear suddenly or worsen dramatically after an infection.

These conditions should not be used as universal explanations for autism. However, a child with a pre-existing neurodevelopmental diagnosis can also develop PANS, PANDAS or another postinfectious neuroimmune condition. A sudden change from the child's previous baseline deserves medical investigation rather than being automatically attributed to autism.

**What does a typical treatment protocol look like in your practice? What roles can antimicrobials, immunomodulation, anti-inflammatory interventions and nutritional support play?**

There is no single protocol for every child. Treatment usually begins with identifying the most clinically important and treatable abnormalities. These may include a documented infection, immune deficiency or dysregulation, inflammation, gastrointestinal dysfunction, nutritional deficiency, sleep disturbance or an endocrine problem.

Antimicrobials may be considered when there is sufficient evidence of a clinically relevant infection. Anti-inflammatory and immunomodulatory measures may be used when inflammation or immune dysfunction is supported by the overall clinical picture. Nutritional support helps correct deficiencies and support normal metabolic and immune function, but supplements should not be presented as substitutes for diagnosis or medical treatment.

Whenever possible, interventions are introduced in a structured manner. If too many treatments begin simultaneously, it becomes difficult to determine which one helped or caused an adverse reaction.

**How individualized are your protocols? Can two children with the same diagnosis require completely different approaches?**

Absolutely. Two children may both meet the diagnostic criteria for autism while having very different developmental histories and biological abnormalities.

One child may have prominent gastrointestinal and nutritional problems; another may have recurrent infection and immune deficiency; a third may have autoimmune features or a clear postinfectious regression. Their treatment plans should therefore not be identical.

Personalized medicine means treating the child's documented abnormalities, not treating the diagnostic label with a universal package of medications or supplements.

**How do you evaluate whether treatment is working? Which clinical changes and laboratory findings matter most to you?**

**Clinical progress comes first.** I place more weight on meaningful improvement in the child's daily functioning than on the normalization of any single laboratory value. The most important changes include:

- communication and receptive language;
- social engagement, attention, anxiety, and repetitive behaviors;
- sleep, gastrointestinal function, and motor abilities;
- self-injury, school participation, and daily independence.

**Multiple sources of observation matter.** Parents' observations are essential, but whenever possible they should be supported by standardized developmental or behavioral assessments, input from therapists and teachers, and objective medical findings.

**Laboratory results must be interpreted in context.** Monitoring depends on the abnormality being treated and on the safety requirements of the therapy.

Treatment should not be considered successful simply because an antibody level changes; it should be judged by whether the child improves clinically.

**What kinds of improvement have you observed in your patients, and what have the children with the most significant improvements taught you about the biology behind their original diagnosis?**

I have observed improvements ranging from better sleep and gastrointestinal function to reduced hyperactivity, anxiety, aggression, repetitive behavior and frequency of infections. Some children have shown better attention, communication, social interaction, learning and independence.

The strongest improvements have taught me that at least some symptoms considered inseparable from autism may be intensified by treatable medical processes. When a child's functioning improves after a documented biological abnormality is addressed, this provides an important clinical signal.

However, an individual response does not by itself prove causation or establish that the same treatment will help other children. These observations should generate carefully designed clinical studies.

**Have you seen children improve to the point where they no longer meet their previous diagnostic criteria? If so, how do you interpret these cases?**

Yes, I have seen children improve substantially, and some were later reported to no longer meet their previous diagnostic criteria. Such cases must be interpreted carefully. Diagnostic status can change because of maturation, early developmental intervention, correction of medical problems, initial diagnostic uncertainty or a combination of these factors.

I do not interpret these cases as proof of a universal cure for autism. I interpret them as evidence that the autism spectrum contains biologically different subgroups and that some children possess a greater capacity for functional recovery when contributing medical abnormalities are identified and treated early.

**What do you believe conventional medicine currently overlooks when evaluating children with autism or neuropsychiatric symptoms?**

Modern medicine is highly specialized. A gastroenterologist may evaluate the intestine, an immunologist the immune system, a neurologist the nervous system and a psychiatrist the child's behavior. What is often missing is the integration of these findings into one biological model.

Behavioral and educational therapies are important, but they should not prevent physicians from investigating pain, inflammation, infection, immune dysfunction, sleep disorders, seizures, gastrointestinal disease, nutritional deficiencies or endocrine problems.

The central question should not be only, "Which diagnosis describes this behavior?" We should also ask, "Which biological processes may be contributing to this child's condition?"

**Many psychiatric and neurodevelopmental diagnoses are still defined primarily by symptoms and behavior. Do you think this will change as we learn more about the biological mechanisms behind these conditions? What do you think diagnosis will look like in the future?**

Yes. Behavioral diagnosis will remain useful, but I believe it will increasingly be supplemented by biological characterization.

Future diagnosis may combine clinical phenotype with immune, inflammatory, metabolic, microbiome, electrophysiological, imaging and genetic or epigenetic information. Instead of saying only that a child has autism, physicians may identify a more precise subtype—for example, a predominantly genetic-developmental subtype, a neuroimmune or postinfectious subtype, a metabolic subtype or a mixed form.

Before biomarkers enter routine practice, however, they must be validated in large, well-controlled studies. We must avoid replacing one oversimplification with another.

**Do you think conditions we currently call autism, ADHD, OCD or other psychiatric and neurodevelopmental disorders will eventually be divided into biological subtypes with different causes and different treatments? If so, what could this mean for the future of psychiatry and personalized medicine?**

I believe this is very likely. These diagnoses describe clusters of symptoms, but similar symptoms may arise through different biological pathways. Conversely, one biological process—such as neuroinflammation—may produce different psychiatric or developmental manifestations in different people.

Identifying biological subtypes could transform psychiatry from predominantly symptom-based management toward mechanism-based and personalized treatment. It could help physicians predict which patients are more likely to respond to antimicrobial, immunological, metabolic, neurological, behavioral or conventional psychiatric interventions.

The future will probably not eliminate current diagnoses immediately. It will add a second layer: the behavioral phenotype together with the biological mechanism.

**Finally, what would you tell a parent who has just received an autism diagnosis for their child and is wondering whether there could be more to investigate medically?**

**Begin with hope, not blame.** I would first tell parents not to lose hope and not to blame themselves. An autism diagnosis describes a child's current developmental profile; it does not define the child's entire future.

**Start support early and evaluate medically.** Families should begin evidence-based developmental, educational, and communication support as soon as possible. At the same time, the child should receive a careful medical evaluation, especially when symptoms would deserve attention in any child, such as:

- developmental regression, seizures, or loss of skills;

- recurrent infections, gastrointestinal pain, or severe sleep disturbance;
- unusual fatigue, sudden psychiatric changes, or nutritional problems;
- signs of inflammation or immune dysfunction.

**Be cautious and look for thoughtful care.** Parents should be wary of anyone who promises a universal cure or recommends prolonged medication based on a single unconfirmed laboratory result. The goal is not to chase every theoretical abnormality, but to identify clinically meaningful, treatable problems and help the child achieve the best possible health and development.

Ideally, families should work with physicians who are scientifically responsible, clinically curious, and able to integrate evidence-based medicine with careful clinical reasoning in complex cases.