A Collaborative Update on Autism Spectrum Disorder

Exploring the Role of Vector-Borne Infections in a Global Health Crisis

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Executive Summary

Autism spectrum disorder (ASD) now affects over 7 million Americans, with 1 in 31 children diagnosed, a 384% increase since 2000 (CDC, 2023; TACA, n.d.). Despite billions invested in genetic and behavioral research, no definitive cause or cure has been found. Meanwhile, families face lifelong disability, emotional hardship, and financial strain, often with no answers.

This white paper presents a working hypothesis based on clinical observations, published case reports, decades of overlooked research, and emerging scientific evidence:

> **That chronic, potentially congenital infections, particularly from bartonellosis, babesiosis, and borreliosis (Lyme disease), may be a root cause of autism in a significant subset of children.**

Among the three infections, bartonellosis is considered by some physicians and research to be the most neurologically active and symptom-producing, particularly in cases involving neuropsychiatric and cognitive symptoms. It has been associated with encephalopathy, seizures, ADHD, rage, anxiety, OCD, and developmental regression, potentially making it the most significant direct contributor to autism presentations (Breitschwerdt et al., 2020, 2025).

This hypothesis is biologically plausible, testable, and aligns with real-world clinical patterns seen by physicians and families across the world. It offers a unifying explanation for six long-standing mysteries in autism research:

- Why so many autism families have autoimmune disease
- Why children with autism exhibit symptoms at an early age
- Why autism can develop suddenly or gradually after immune stress, including vaccines
- Why standard genetic and environmental models cannot explain the exponential rise
- How real-world transmission, through congenital, pet-related, vector-borne, and sexual pathways may be sustaining and spreading infection
- Why some children with autism show substantial improvement when treated with long-term antimicrobial therapies

Numerous case reports exist, and word-of-mouth has led many families to seek out more accurate testing from specialty laboratories, often uncovering evidence of these infections. In some cases, parents have witnessed dramatic improvements in their children when appropriate treatment was initiated.

By any scientific standard, this hypothesis warrants urgent research. If even a fraction of cases could be explained or improved by addressing hidden infections, the impact on children, families, and public health would be historic.

> The question may no longer be, “What causes autism?”
> It may now be, “What if we’ve been overlooking an infection all along?”

2. Background and Problem Statement

ASD has become one of the most rapidly rising and least understood medical conditions in modern history. According to the CDC, autism affected 1 in 150 children in 2000. As of 2024, that number has surged to 1 in 31 (CDC, 2023). This increase has been consistent across states, demographics, income levels, and globally, defying the notion that diagnostic expansion or awareness alone can explain the trend.

Current scientific models fall short:

- Genetics cannot change fast enough to explain a 384% increase in just two decades. Human genetics evolve over millennia, not decades

- Autism rates have risen steadily and uniformly across countries, climates, and communities, defying the expectations of any environmental theory. If environmental exposures like industrial chemicals, pesticides, heavy metals, or pollutants were the primary cause of autism, we’d see more hot spots or localized surges tied to specific toxins or triggers.

- Improved diagnosis and awareness explain only a portion of the rise, and does not match the 25-yr long exponential curve seen in national surveillance data

Meanwhile, clinical observations have pointed to something missed by commonly quoted research: an infectious hypothesis.

A Hidden Infectious Driver?

Three vector-borne pathogen species, *Bartonella*, *Babesia*, and *Borrelia* (Lyme disease), are increasingly recognized by researchers, as well as physicians with advanced training in vector-borne disease and chronic illness. These infections are known to:

- Cross the placenta during pregnancy in animals and suspected in humans (congenital transmission) (Breitschwerdt et al., 2010a, 2010b, Behnke et al., 2001)

- Persist in hosts for years without detection (Cheslock & Embers, 2019, Bush et al., 2024)

- Invade the brain, immune system, joints, and gut (Breitschwerdt et al., 2010c)

- Cause neuropsychiatric, neurological, and autoimmune symptoms in both children and adults (Atladóttir et al., 2009, Breitschwerdt et al., 2020, 2025, Comi, 1999, Wu et al., 2015, Bransfield, 2018, Bransfield et al., 2020). While once thought rare, these infections may be far more common, and far more impactful, than previously believed

Realistic Transmission Pathway

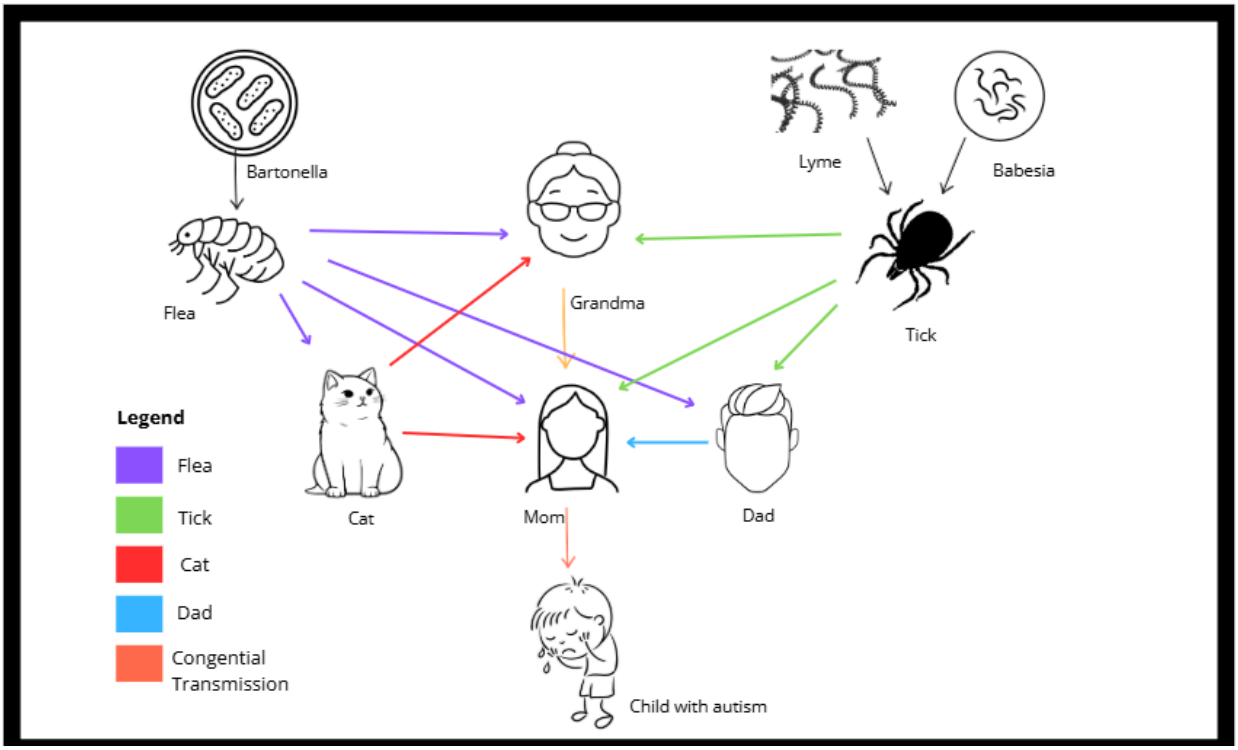
These infections can spread through:

- Congenital transmission (mother to fetus) (Siewert, 2022, Tołkacz, 2018, Zerbo et al., 2015)

- Vector exposure. *Bartonella* has the highest number of vectors (fleas, ticks, mites, lice, bed bugs, biting spiders, sand flies, red ants, mosquitoes) (Chomel, 2006; Billeter, 2008; Breitschwerdt, 2019). *Borrelia* and *Babesia* spread through ticks

- Animal contact with an infected cat or dog can transmit *Bartonella* through scratches, bites, or potentially licks on an open wound

- Possibly sexual transmission, based on animal models and human case reports



These routes provide a realistic and worldwide model for how the infections could silently pass from one generation to the next, matching the autism curve's slow rise (1970-1998), then exponential rise (1999-2024).

Prevalence and Occupational Exposure

Bartonellosis is more common than previously recognized, particularly among individuals with frequent animal contact. Studies have found that:

- In humans, seroprevalence among veterinarians and animal workers ranges from 24% to 55%, with one study finding 28% of symptomatic veterinarians tested positive via PCR (Lantos et al., 2014).
- Up to 28% of symptomatic veterinarians tested positive for Bartonella DNA in their blood, compared to 0% in healthy controls (Oteo et al., 2017)

These findings suggest that *Bartonella* poses a substantial risk to those in animal-related professions, yet the prevalence in the general population, especially those with pets, remains unknown, highlighting a critical gap in public health surveillance.

3. Core Evidence Supporting the Infectious Hypothesis

A. Overlap Between Autism Symptoms and Stealth Infections

The core symptoms of ASD, social withdrawal, anxiety, irritability, sensory issues, sleep disturbances, GI dysfunction, tics, OCD, ADHD, hypermobility (EDS), and seizures, are all documented in individuals with stealth infections, especially bartonellosis, which appears to be the most neuroinvasive and symptomatically aligned with autism, though all three infections cause important symptoms. (Alibek & Muehlenbein, 2022, Breitschwerdt et al., 2008, 2020, 2025, Offutt & Breitschwerdt, 2023, TACA, 2025, Bransfield et al., 2008, Bransfield, 2012, Kuhn et al., 2014)

Key Point: These infections can produce a neuropsychiatric and developmental picture nearly indistinguishable from autism, and are rarely tested for.

B. Animal Reservoirs and Vectors

These infections are not only clinically significant, they're epidemiologically widespread.

Domestic animals and their parasites serve as key reservoirs and transmission sources for *Bartonella*:

- Cats: Up to 24% of domestic cats carry *Bartonella henselae* in their bloodstream, and over 50% show antibodies, often without symptoms (Jameson et al., 1995)

- Dogs: General seroprevalence of *Bartonella* in dogs appears relatively low, but transmission from a pet is still possible. In a study of over 15,000 dogs from North America, 3.26% tested seropositive for at least one *Bartonella* species, with 2.13% positive for *B. henselae*, 2.39% for *B. koehlerae*, and 1.42% for *B. vinsonii* subsp. *berkhoffii* (Lashnits et al., 2018)

- Fleas: A Florida-based study found that 11.3% of pooled flea samples from dogs tested positive for *Bartonella* DNA, highlighting the potential for fleas to act as vectors for *Bartonella* species in the United States. (Lashnits et al., 2014)

This widespread animal and vector presence, combined with underdiagnosis in humans, makes it plausible that *Bartonella* exposure is far more common than currently believed.

C. Autoimmunity in Families

Research shows significantly elevated rates of autoimmune conditions in autism families, with studies finding that 16-21% of mothers and first-degree relatives of children with autism have autoimmune disorders, compared to 2-4% in control families (Comi et al., 1999). Additionally, 46% of autism families have two or more members with autoimmune disorders, representing a substantial increase over general population rates of approximately 4.6% (Abend et al., 2024).

These conditions include thyroid disease, rheumatoid arthritis, lupus, fibromyalgia, diabetes, inflammatory bowel disease (IBD), and multiple sclerosis. (Atladóttir et al., 2019, Comi et al., 2018, Wu et al., 2015). Increasing evidence suggests that many of these autoimmune conditions may in fact be caused or mimicked by persistent bartonellosis which is known to induce chronic inflammation and immune dysregulation that resembles autoimmune pathology (Breitschwerdt et al., 2012, Maggie et al., 2012). Rather than spontaneous immune dysfunction, this clustering of autoimmune diseases may reflect undiagnosed, inherited or newly acquired infections that the immune system is persistently fighting.

Key Point: These may not be autoimmune conditions at all, but rather unidentified infections mischaracterized as autoimmunity.

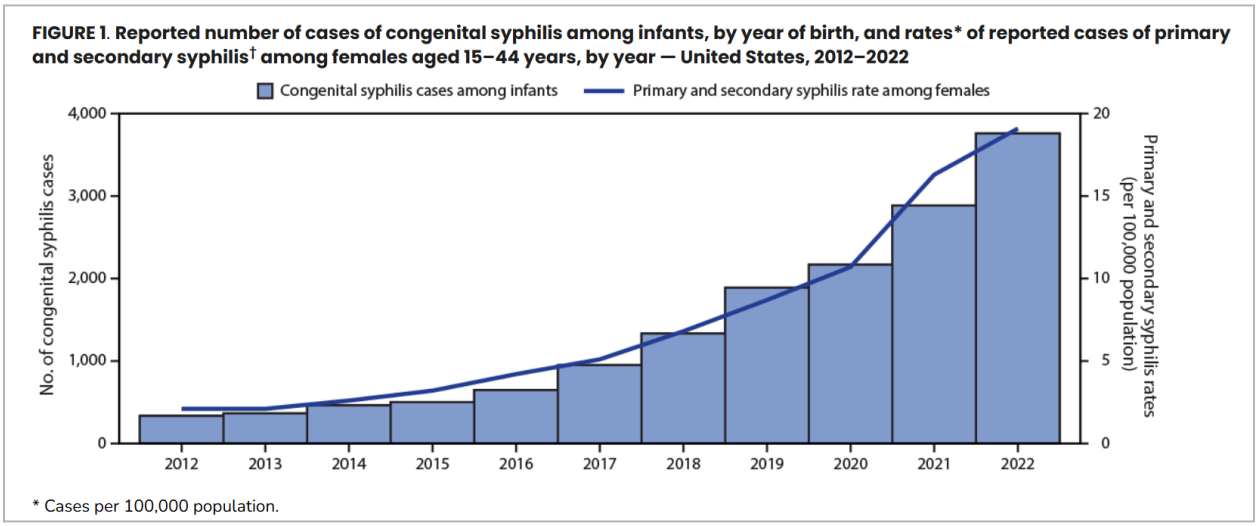
D. Congenital Transmission & Generational Modeling

Lyme disease associated with ASD has been documented to have been transmitted in three generations (Bransfield, 2009). Infections like bartonellosis are now known to transmit vertically from mother to child in animals, and based on clinical evidence, are strongly suspected to do so in humans (Behnke et al., 2001; Breitschwerdt et al., 2010a, 2010b). If a parent is infected, the pathogens may silently pass to the fetus in utero. That child may not develop autism but may experience other symptoms such as PANS, ADHD, and neuropsychiatric disorders commonly associated with these stealth infections. If left untreated, the infection may persist for life, and be transmitted again to the next generation. The autism risk may be especially pronounced in male offspring, who are diagnosed with autism at significantly higher rates than females.

So what would a congenital transmission curve look like?

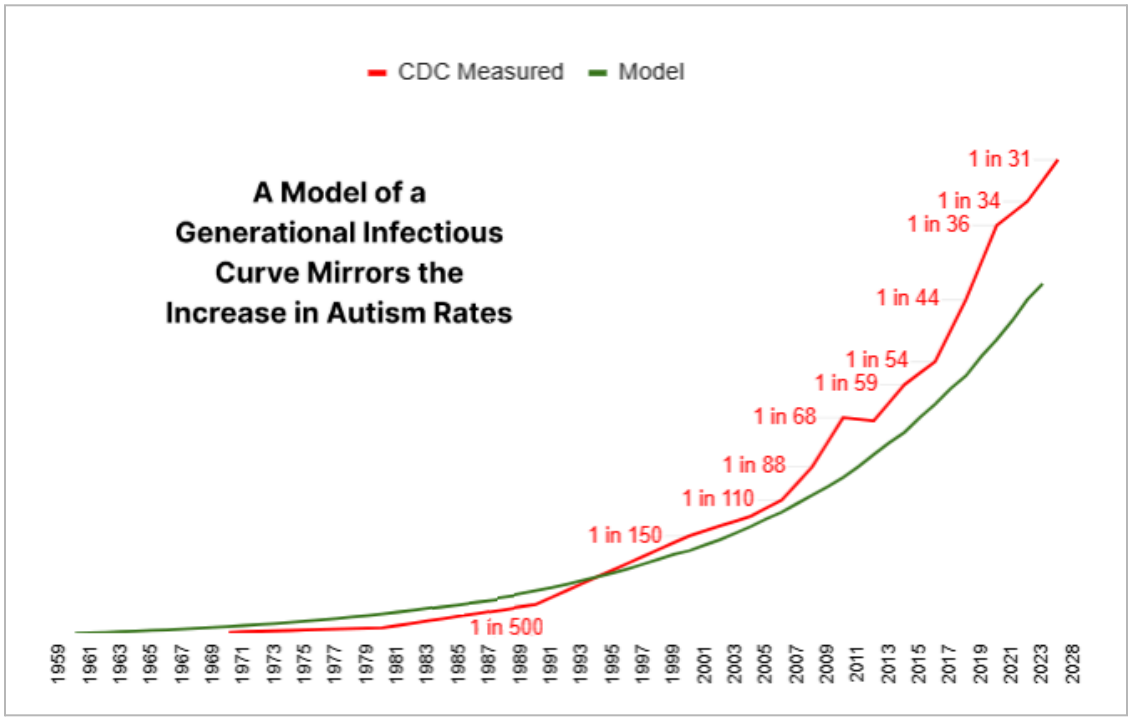
The shape of the congenital syphilis curve below is unmistakable. It's the classic trajectory of an infectious disease spreading through vertical (mother-to-child) transmission. What makes it so striking is how closely it mirrors the autism prevalence curve: a long period of slow growth followed by a sharp and accelerating rise. And this is for a disease that is easily screened and cured during pregnancy. In contrast, *Bartonella*, *Babesia*, and *Borrelia* are more difficult to detect, rarely screened for, and still not recognized by the NIH as causes of chronic illness. If even a subset of autism cases are caused by missed congenital infections, we are already in the midst of the next potentially preventable public health crisis.

Figure 1



Source: Centers for Disease Control and Prevention (CDC). Sexually Transmitted Infections Surveillance, 2022. Published April 30, 2024. Figure 1: Reported number of cases of congenital syphilis among infants, by year of birth, and rates of reported cases of primary and secondary syphilis among females aged 15–44 years, by year — United States, 2012–2022.

Figure 2



Source: CDC Autism and Developmental Disabilities Monitoring (ADDM) Network, 2000–2022; NIH and peer-reviewed literature for historical estimates (1970–1990); 2024 estimate from TACA based on CDC trend analysis.

Modeling Autism as a Congenital Infectious Disease (Description)

- Includes known and suspected transmission routes: Flea and cat exposure, CDC-estimated Lyme disease infection rates, Potential sexual transmission, based on existing case reports and animal models
- Factors out overlapping infectious contributors to isolate the potential impact of Bartonella and related pathogens
- Incorporates probabilities for: Congenital transmission from infected mothers to offspring, Autism development in children exposed in utero
- The curve shown represents the moderate scenario, based on mid-range estimates for all variables. The model also includes conservative and aggressive cases to represent a realistic range of possible outcomes

  Note: This model is not yet published and will be undergoing review by a statistician.

A Congenital Transmission Model That Matches the Autism Curve

In a congenital infection model, spread doesn't occur over days or weeks like a viral model, but across generations. A child is born with a congenital infection. As they grow, they may struggle with anxiety, OCD, fatigue, or neurological symptoms. These symptoms are rarely connected to infection, and may often be misdiagnosed as neuropsychiatric disorders. Eventually, they become parents, and the infection is silently passed on. One or more of their children may develop autism.

This pattern repeats. At first, the rise is barely noticeable. However, across two or three generations, the numbers begin to accelerate. The curve steepens. That's what we've modeled, a generational transmission curve, which closely mirrors the real-world autism prevalence data from the CDC, NIH, and TACA.

Transmission doesn't just occur vertically. Women can acquire these infections directly from pets, vectors, or potentially through sexual contact, as with syphilis, and then potentially pass them on in utero. The result is a compounding cycle of congenital and acquired transmission.

E. Vaccine-Triggered Symptom Onset

Some parents report a sudden or gradual onset of autism symptoms after routine vaccinations, especially around 12–18 months (CDC, 2022). This has fueled controversy about the safety of vaccines, but a more plausible explanation is emerging: vaccines may challenge the immune system in children harboring latent infections, triggering a flare in symptoms. While this could be related to the vaccine, it might instead reflect the infection itself or the immune activation it triggers.

This same phenomenon has been documented in adults with bartonellosis who develop "Long COVID symptoms" after immune triggers like COVID-19 or COVID-19 vaccination. (Dong et al. 2024, Aubry et al., 2024, Yale News, 2025, de Sá, 2024). Developing Long COVID following the COVID vaccine may be the first time *adults* suffered debilitating bartonellosis symptoms after a vaccine, lending more credence to the possibility in children. This strengthens the plausibility of a similar mechanism in children, except in children, bartonellosis may involve deeper or more widespread brain involvement beginning in utero, during critical stages of neurodevelopment, leading to the specific autism symptoms typically seen in children.

To avoid future transmission, since Long COVID symptoms closely mirror bartonellosis symptoms in adults, doctors should consider either screening their Long COVID patients using sensitive dPCR testing from specialty labs or referring their patients to a vector-borne disease specialist with advanced training.

F. Clinical Improvements with Antimicrobial Treatment

Some case reports describe children with autism symptoms who tested positive for stealth infections and showed significant improvement following long-term treatment with broad-spectrum antimicrobial protocols. However, more research is needed to understand how common these infections are in the autism population.

Symptom improvements include:

- Returned or improved speech
- Reduction in rage, anxiety, tics, and OCD

- Better sleep and eating

- In some cases, exit from special education or social skills classrooms

These are not isolated anecdotes; they reflect a pattern observed by many physicians and families from around the world in online support groups (Kuhn et al., 2006, Bransfield et al., 2007, Bransfield et al., 2008, Offutt & Breitschwerdt, 2023).

G. Why We're Discussing Three Infections, Not One

Unlike most infectious diseases that focus on a single pathogen, this hypothesis centers on three infections, bartonellosis, babesiosis, and borreliosis because they are frequently found together in chronically ill patients when an experienced physician evaluates the patient with these potential issues in mind.

These stealth pathogens:

- Share common vectors (e.g., ticks, fleas) (Chomel, 2006; Billeter, 2008)

- Have overlapping symptoms (neurological, psychiatric, inflammatory) (Bransfield et al., 2007, Bransfield et al., 2008, Tomasiewicz et al., 2016)

- Evade detection and often go undiagnosed (Cheslock & Embers, 2019, Bush et al., 2024)

Clinical studies and specialty lab data show that when one is present, there's a 50% likelihood of one or more co-infections (Halos et al., 2005, LymeDisease.org, n.d.). These infections may act synergistically, worsening severity and complicating diagnosis and treatment.

For that reason, this paper considers all three as part of a poly-microbial model which is a more realistic framework for how these pathogens may contribute to autism and neurodevelopmental disorders.

4. Multifactorial Nature of the Disease

While bartonellosis, babesiosis, and borreliosis are often referred to as "stealth infections" due to their ability to persist undetected, they are also best understood as components of a multifactorial disease process. Two complicating comorbidities, mast cell activation syndrome (MCAS) and mycotoxin related illness or chronic inflammatory response syndrome (CIRS), frequently accompany these infections, and both can significantly impact symptom severity and treatment outcomes.

Patients with these vector-borne infections often experience treatment resistance, unexplained symptom flare-ups, or slow progress until these secondary factors are addressed. In fact, in

most integrative or functional medicine practices, the standard protocol is to first stabilize the patient by addressing mycotoxin illness and MCAS-related inflammation. Doing so often leads to faster symptom relief and improved response and tolerance to antimicrobial treatment.

MCAS

MCAS is increasingly recognized as a comorbid condition closely linked to bartonellosis, babesiosis, and borreliosis. It occurs when mast cells, immune cells involved in allergic and inflammatory responses, release excessive or inappropriate amounts of histamine and other chemical mediators.

Common symptoms of MCAS include:

- Flushing, itching, or hives
- Gastrointestinal upset (nausea, diarrhea, abdominal pain)
- Brain fog, anxiety, or mood instability
- Low blood pressure, dizziness, or heart palpitations
- Shortness of breath or chest tightness
- Sensitivity to smells, foods, or medications

Treating MCAS can bring much-needed symptom relief and allow the immune system to respond more effectively to antimicrobial therapy. However, treatment is not one-size-fits-all. Most patients require a trial-and-observe approach, testing various combinations of H1 and H2 blocking antihistamines, mast cell stabilizers, and in some cases, beta-blockers. Finding the right combination often involves evaluating multiple permutations, essentially a Cartesian product of options, to discover what works best for that individual.

Fortunately, awareness of MCAS is growing rapidly in the medical community, especially among allergists. However, this condition is often still underdiagnosed in traditional settings. Every patient diagnosed with MCAS should be screened for underlying vector-borne infections by a qualified and knowledgeable physician, as these infections are often the upstream drivers of mast cell dysregulation.

Mold Toxicity / Mycotoxin Illness

Mycotoxin illness is another major complicating factor in patients with vector-borne infections. Mold exposure, especially to mycotoxins from water-damaged buildings, can trigger severe immune dysregulation, contribute to chronic inflammation, and cause flare-ups of existing symptoms, particularly neuropsychiatric manifestations such as anxiety, mood swings, OCD, rage, or cognitive dysfunction.

In patients with bartonellosis, babesiosis, or borreliosis, mold exposure can worsen:

- Chronic fatigue
- Migrating joint pain
- Rages, panic attacks, or OCD
- Light and sound sensitivity
- Gastrointestinal distress
- Sleep disruption
- POTS-like symptoms

One little-known factor is that modern paints no longer contain lead, which used to offer resistance to water penetration. Today's low-VOC and water-based paints provide minimal water resistance, making homes more vulnerable to mold growth after even minor water leaks. Mold can hide behind drywall, under carpets, and in HVAC systems, often undetected until it's causing significant harm.

Proper identification and treatment of mold exposure, can result in dramatic symptom relief and allow patients to tolerate and respond better to antimicrobial treatments targeting the infections themselves.

5. A Unifying Hypothesis That Fits the Facts

The current scientific hypotheses of genetic and environmental impacts have failed to explain the full autism picture. In contrast, the infectious hypothesis provides a testable, biologically plausible model that addresses multiple facets known about this condition, and a clinically observed explanation for the most persistent autism questions:

Mystery	Explained by Stealth Infections
High rates of autoimmune disease in families	Chronic infection triggering long-term immune activation; misdiagnosed as autoimmunity Atladóttir et al., 2019, Breitschwerdt et al., 2012, Comi et al., 2018, Maggie et al., 2012, Wu et al., 2015)

Symptom overlap with other disorders	Symptoms like anxiety, OCD, tics, GI problems, ADHD and seizures are frequently reported in stealth infections, especially bartonellosis, which shows the greatest neuropsychiatric overlap with ASD (Breitschwerdt et al., 2008, 2020, 2025)
Sudden or regressive onset of symptoms after vaccines	Immune challenge, not the vaccine itself, may trigger flare of a latent infection already present in the child (CDC 2022)
Exponential rise in autism rates	Matches the spread pattern of a slow-moving congenital infection epidemic across generations (CDC, 2024)
Inconsistent effectiveness of autism treatments	Standard therapies don't address the infectious root cause (Lord et al., 2018)
Reports of dramatic symptom improvement with antibiotics	Seen in multiple case reports and physician practices when treating stealth infections (Offutt & Breitschwerdt, 2023, Rolain et al., 2004)
Family clustering and affected siblings (Ozonoff et al., 2024, Zerbo et al., 2015)	Reflects shared congenital and environmental exposures (including pets, vectors)

In his blog *The Tick Detective*, Dr. Richard Horowitz discusses how infections such as Bartonella, Babesia, and Borrelia, along with immune dysregulation and other downstream effects, may contribute to autism spectrum disorders in some individuals. These effects range from mitochondrial dysfunction and chronic neuroinflammation to autoimmune reactions against brain tissue that profoundly influence cognition, mood, and behavior. He also notes that between 60 and 86 percent of individuals with ASD experience chronic insomnia, which can significantly worsen cognition, behavior, and quality of life.

This hypothesis may not explain all cases of autism, but if it explains even a subset, the implications for diagnosis, treatment, and prevention are profound.

Why Testing and Treatment Is Difficult and a Cure Is Urgently Needed

The Testing and Treatment Gap

These infections are not only incredibly difficult to treat, they're incredibly difficult to diagnose.

Standard commercial lab tests are often ineffective for detecting *Bartonella*, *Babesia*, and *Borrelia* in their chronic or congenital forms. (Ericson, et al., 2024) These stealth pathogens:

- Hide inside tissue, cells, biofilms, and macrophages, evading immune detection
- Persist at low levels, especially in chronic cases
- Do not reliably trigger antibody production, leading to false negatives

As a result, chronically infected patients often test "negative" on standard panels and are dismissed as having behavioral, psychological, or idiopathic conditions, if they are tested at all.

At the same time:

- The NIH does not formally acknowledge the existence of bartonellosis, babesiosis, or borreliosis, or understand these are a significant contributor to chronic disease, let alone as possible drivers of autism
- The NIH has sponsored the LymeX competition to develop accurate diagnostics for *Borrelia.* However, there are no federal guidelines or funding for the equally important improved diagnostics for *Bartonella* and *Babesia*
- Most physicians receive no training in identifying, screening, ordering, or interpreting advanced panels from specialty laboratories, where more sensitive testing is available
- Primary care physicians in conventional medical settings would refer patients to infectious disease specialists in vector-borne disease if it was suspected that there might be a significant infection causing symptoms in a patient. However, only 89,000 cases of Lyme disease were reported in the United States in 2023, while the CDC estimates that 476,000 cases occur annually. This disease is not a high priority in the minds of outpatient physicians. Only 1,762 cases of *Babesia* across 27 reporting states are noted in the CDC archives with the most recent data over 10 years old, from 2013. Even for *Bartonella henselae*, only about 20,000 cases are diagnosed annually, and these are primarily acute cases. These are obviously underestimates of unknown proportion since physicians are not trained to look for these potential underlying infectious etiologies in chronic cases.

Without accurate identification and testing, there is no diagnosis. Without diagnosis, there is no treatment. Without recognition by institutions like the NIH, families are left to navigate these infections alone, paying out of pocket, often spending tens of thousands of dollars for specialty testing, medications, and specialty care not covered by insurance, with less than desirable results.

> **This is not just a scientific gap. It's a systemic failure that is delaying care that could change lives.**

The MSIDS Model, A Tool for Diagnosing Complex Illness

Developed by Dr. Richard Horowitz and co-author, the MSIDS model (Multiple Systemic Infectious Disease Syndrome) offers a structured way to diagnose and treat this complex, chronic illness that involves overlapping infections and immune dysfunction.

MSIDS recognizes that many patients, especially those with autism, PANS/PANDAS, Long COVID, or autoimmune symptoms suffer from more than one underlying etiology. The model includes infections like bartonellosis, babesiosis, borreliosis, plus co-occurring issues such as:

- Immune dysregulation
- Mitochondrial dysfunction
- Mold and toxin exposure
- Mast Cell Activation Syndrome (MCAS)
- Hormonal imbalances

This broad-based approach helps doctors identify obstacles to healing, personalize treatment, and recognize when stealth infections may be driving psychiatric or neurological symptoms.

Dr. Horowitz's MSIDS model is described in peer-reviewed publications and in his books, *Why Can't I Get Better?* and *How Can I Get Better?*

Treatment is equally challenging. Bartonellosis, babesiosis, and borreliosis are not fully recognized by many as persistent pathogens. As a result:

- The NIH provides no education or guidance on these chronic infections.
- There are no standardized, approved treatment protocols, nor protocols that are known to cure, so relapse is frequent
- Doctors who treat these infections must rely on clinical experience and long-term antibiotic strategies

- Medications may need to be compounded, and doctors visits are often cash-pay only because insurance won't cover the long appointments needed for complicated medical cases

Families are left to shoulder the burden often costing tens of thousands of dollars over years, with no roadmap, no insurance support, and no cure.

This system is not only unsustainable. It's unjust. That's why diagnostics, treatment access, and cure research must be priorities in any response.

> **It's no longer a question of whether this hypothesis is worth testing. It's a question of how many lives we miss helping if we don't.**

The Silent Progression of Stealth Infections

These infections are often insidious. Following exposure, often unrecognized due to the tiny size of vectors or unrecognized problematic contact with pets, symptoms may emerge slowly, ranging from headaches, anxiety, and obsessive behaviors to joint, muscle, or nerve pain, thyroid conditions, dizziness, or chronic fatigue. At this stage, many never seek medical care, or if they do, physicians typically treat the symptoms without considering infectious causes. These infections are rarely considered, or if they are, standard tests often fail to detect *Bartonella*, *Babesia*, or *Borrelia*. Patients are often misdiagnosed with an autoimmune or neuropsychiatric diagnosis, or dismissed.

Over time, the infection can persist and worsen, leading to visits across specialties such as psychiatry, rheumatology, and endocrinology without resolution (the chronic health crisis). Untreated, these stealth infections may contribute to one or more autoimmune and neuropsychiatric conditions, metastatic cancer, or Alzheimer's disease (Allen et al., 2021, Miklossy, 2011). Worse still, they may be passed to offspring through congenital transmission, manifesting as autism, PANS, or autoimmune disease as adults. Yet few in the medical community recognize how these pieces may fit together. So, the cycle continues.

6. Clinical Case Example

> "I searched online for answers to my son's OCD. This led me to a functional doctor who diagnosed my son with PANS, then screened for vector-borne infections. I was shocked to discover all three of my boys and myself were infected."
> — mother of autistic son

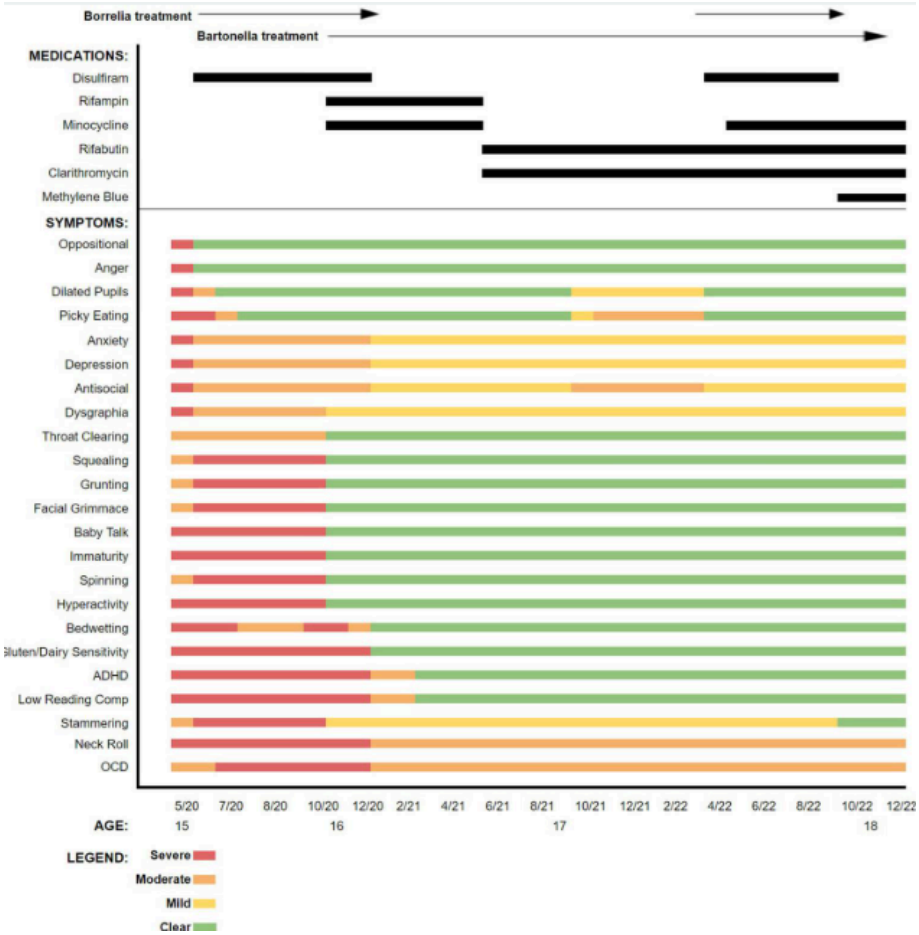
In a clinical case study, this young teen, diagnosed with autism at thirteen, struggled with ADHD, oppositional defiance, OCD, tics, anxiety, food selectiveness, and severe educational limitations (Offutt & Breitschwerdt, 2023). For years, he received standard behavioral and psychiatric care with limited success.

At age 16, he was tested using specialty laboratory panels and found to be positive for:

- *Bartonella henselae*
- *Babesia microti*
- *Borrelia burgdorferi*

After receiving targeted long-term antimicrobial treatment under physician supervision, he experienced significant improvements:

- Resolution of oppositional behavior and anxiety
- Normalized sleep and eating
- Six-point increase in IQ
- Mainstreamed out of special education and social skills classes for the first time in his life in his senior year of high school



More than twenty chronic symptoms improved or disappeared with long-term antibiotic treatment and without psychiatric medications.

This boy's story reflects what large numbers of parents and qualified functional and integrative medicine doctors are now reporting: When stealth infections are diagnosed and treated, autism symptoms can improve.

7. Conclusion: A Hypothesis Worth Urgent Action

The evidence presented in this white paper supports a testable, biologically plausible hypothesis: that congenital vector-borne infections, particularly bartonellosis, may be a hidden driver of autism spectrum disorder in a significant subset of children.

This white paper has presented a unified hypothesis that connects:

- Rising autism rates to a pattern of congenital infectious disease spread
- Widespread autoimmune illness in families in both adults, and in their children as they age
- Neuropsychiatric autism symptoms consistent with stealth congenital infections
- Transmission patterns that mirror infectious disease, and are consistent globally
- Real-world improvements seen with antimicrobial therapy

If even a fraction of autism cases are caused or worsened by congenital infection, then urgent research is not optional, it is morally and medically required.

We now have:

- A biologically plausible cause
- A realistic, testable transmission model
- A trackable symptom profile
- A pathway to diagnosis and treatment

It opens a door to something we haven't had before: a pathway to prevention, intervention, and possibly recovery.

We Must Act Now

Urgent, well-funded research is needed to:

- Conduct large-scale infection screening in ASD populations and their mothers and a healthy cohort of children and their mothers, along with unvaccinated children and their mothers
- Develop accurate diagnostic tests for all infections that are available at standard labs
- Find a cure for all three vector-borne infections: bartonellosis, babesiosis, and borreliosis
- Conduct medical training and public awareness campaigns to encourage screening and reduce exposure, especially from infected pets

Screen Where It Matters Most

If this hypothesis is correct, lives could be changed through simple, scalable changes to screening practices:

- During pregnancy (as done today for syphilis, CMV, and TORCH) (CDC, 2001)
- At birth, when early intervention could make the biggest difference
- Before vaccinations, children harboring latent infections can be healed and consider an appropriate vaccine schedule.
- Screen the national blood supply for *Bartonella* and *Borrelia* to prevent transmission through blood transfusions. Today, the FDA only requires screening for *Babesia* in 18 states. (FDA, 2019).

The Cost of Inaction

Autism currently costs the U.S. over $223 billion annually, with lifetime care per child ranging from $1.4 to $2.4 million. Families often face financial devastation and emotional exhaustion (Buescher et al., 2014).

If research proves even a fraction of autism cases are preventable or treatable by addressing stealth infections, the savings to families and to society would be staggering. The reduction in suffering would also be immeasurable and significant.

A Lesson from History

Bartonellosis and other sub-acute infections may mirror the medical mystery of syphilis a century ago: a stealthy, multi-systemic infection with neurological, psychiatric, and severe congenital consequences that went unrecognized for millennia. Syphilis was eventually understood, tested for, and screened during pregnancy, transforming outcomes for millions. Bartonellosis and other vector-borne infections, like syphilis, hide in tissues, persist chronically, and mimic other conditions. Recognition could similarly shift the trajectory of countless lives.

This hypothesis is not fringe. It is informed by science, observed in clinical settings, and aligned with patient outcomes.

We cannot afford to ignore this hypothesis. We must act with the urgency shown in COVID-19 to fund the research, develop the diagnostics, and find a cure.

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