L1-79 — and Why It Is Only Part of the Answer

A plain-language guide for parents and families

Based on the Biology of Autism (IDA) Framework

Decoding Autism Now · decodingautismnow.com · July 2026

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SECTION 01

What is happening inside your child's body

To understand why any autism treatment works — or why it stops working — you first need to understand what is actually going wrong inside the body. Not at the level of behavior. At the level of chemistry.

The brain uses a chemical called somatostatin to keep itself calm and organized. Somatostatin is produced by a special group of brain cells called SST-14 interneurons — the brain's traffic controllers. Their job is to prevent the brain's signaling system from running too fast and too loud. When they are working properly, the brain can filter sensory input, focus social attention, and coordinate the release of the hormones that make social connection feel rewarding and meaningful.

In children with immune-derived autism, these traffic controller cells cannot produce enough somatostatin because the instructions to make it are being blocked at the genetic level. The gene that codes for somatostatin cannot be read because the chemical machinery needed — which depends on a healthy methylation cycle — is not functioning adequately.

What starts this chain of events?

The cascade can be triggered by several upstream sources. What determines whether a child's cascade progresses is not any single cause but whether they have sufficient metabolic headroom to absorb it:

- **Gut bacteria imbalance and disrupted digestion** — often following repeated antibiotic courses in early childhood — produces opioid-like molecules and inflammatory compounds that activate the immune system and drive the downstream cascade.
- **Environmental toxin exposure** — heavy metals, pesticides, and plasticizers can directly impair the methylation cycle and mitochondrial function, reducing the child's metabolic reserve.
- **Maternal immune activation during pregnancy** — inflammatory conditions in the mother can pre-load the inflammatory pathway in the developing fetus before birth.
- **Immune activation events in genetically susceptible individuals** — in children who already have reduced metabolic reserve due to genetic factors, any concentrated immune challenge during a critical developmental window may be sufficient to tip the cascade.

Whatever the triggering source, the downstream mechanism is the same. Chronic immune activation switches on an enzyme called **IDO1** (indoleamine 2,3-dioxygenase 1), which diverts the amino acid

tryptophan away from producing serotonin and redirects it toward producing toxic compounds called kynurenines. One of those — quinolinic acid — damages the traffic controller cells directly. At the same time, inflammatory signaling blocks the genetic switch that turns somatostatin production on. The traffic controller cells lose from both directions at once.

Framework note

The Biology of Autism cascade model — developed at Decoding Autism Now — maps this full chain of events from multiple upstream insults through the immune activation, IDO1, and SST-14 suppression sequence to the observable clinical phenotype. Every mechanistic step is independently grounded in published and reproducible science. The full cascade documentation is available at decodingautismnow.com.

SECTION 02

What happens when the traffic controllers stop working

When somatostatin production falls, several things happen simultaneously. Understanding all of them explains why autism presents the way it does.

The brain's chemical balance shifts toward excess stimulation

Dopamine and norepinephrine — brain chemicals responsible for alertness, attention, and stress response — build up because the regulatory braking system has been removed. This excess produces sensory overload, difficulty filtering social information, heightened anxiety, and behavioral patterns that reflect a nervous system running without adequate regulation.

Three important hormones stop working properly

These three hormones — VIP, oxytocin, and secretin — are responsible for some of the most recognizable features of autism, and they are all downstream of the traffic controller cells that have been blocked. Oxytocin receives all the cultural attention as the 'love hormone,' but VIP and secretin — which govern whether oxytocin functions at all — are almost entirely absent from the public conversation about autism.

VIP (vasoactive intestinal peptide)

Despite its name, VIP is the upstream conductor of the entire social neuropeptide architecture. It synchronizes the timing of brain activity that allows us to perceive social signals coherently — faces, voices, emotional cues. VIP also controls when and how much oxytocin is released from the hypothalamus, and it is the master signal of the brain's internal clock governing sleep-wake rhythms. Without VIP, social perception fragments, oxytocin release becomes unpredictable, and sleep degrades.

Oxytocin

The love hormone drives eye contact, bonding, the desire to engage with other people, and the emotional satisfaction of reciprocal relationships. Crucially, oxytocin must be released at the right moment, in the right amount, in response to social context — a timing function driven by VIP. This is why giving children exogenous oxytocin through a nasal spray has produced inconsistent results in trials: adding more oxytocin into a system whose release mechanism is broken does not reliably restore the function.

Secretin

A gut-brain hormone that coordinates digestion, pancreatic function, and the broader gut-brain communication axis. In 1998 physicians discovered that children with autism showed behavioral improvement after receiving secretin during a routine digestive procedure. Subsequent controlled trials produced inconsistent results and the treatment was largely abandoned. The IDA framework explains why: secretin is one part of a three-part coordinating system. Giving secretin without restoring VIP and oxytocin is like trying to balance a three-legged stool on one leg.

> *The one-legged stool principle: single-pathway interventions into a multi-pathway regulatory failure consistently produce partial, unstable, non-reproducible outcomes. This applied to secretin trials, to exogenous oxytocin — and, as we will explain, it applies to L1-79 alone.*

The gut runs too loud

The gut has its own version of somatostatin called SST-28. Unlike the brain version which is being blocked and producing too little, the gut version is being overdriven — running too high and not being properly turned off. Several mechanisms contribute simultaneously:

- Opioid-like molecules from incomplete casein and gluten digestion drive gut SST-28 cells into excess activity
- Chronic cortisol elevation from the constitutively active stress response maintains appetite suppression continuously
- Zinc deficiency removes the mineral-dependent regulatory moderator of SST-28 release
- Gut bacteria imbalance — particularly Clostridia overgrowth — directly stimulates SST-28 production

The result is chronic appetite suppression, restricted dietary tolerance, slowed gut motility, reduced gastric acid, and impaired digestive coordination. This is why so many children with autism are highly selective eaters — not by choice, but because multiple layers of gut chemistry are actively suppressing appetite simultaneously.

Sleep is disrupted

VIP is the master signal of the brain's internal clock — the suprachiasmatic nucleus, which controls circadian rhythm. When VIP is impaired, the clock loses its synchronization signal. Sleep disruption in autism is typically attributed to melatonin deficiency, but it is more accurately a VIP-driven circadian clock failure. Melatonin supplements help at the symptom level; VIP restoration is the mechanism.

SECTION 03

What L1-79 does — and why it helps

L1-79 is a drug developed by Yamo Pharmaceuticals. It works by slowing down an enzyme called **tyrosine hydroxylase (TH)** — the enzyme that controls the first step in producing dopamine and norepinephrine. By slowing this first step, L1-79 reduces the amount of dopamine and norepinephrine the body makes.

In a brain where the traffic controller cells have stopped working, dopamine and norepinephrine have been building up unchecked. L1-79 reduces that excess. The result is a brain with less chemical noise — a quieter, more regulated environment in which social signals can be perceived more clearly.

The clinical trial results are real and meaningful

In a rigorous 12-week study of 58 adolescents and young adults with autism, L1-79 produced a nearly 8-point improvement on a standardized social behavior scale called the Vineland-3 — described by the investigators as potentially the largest improvement ever measured with a medication on this scale. Clinicians rated participants as meaningfully improved, and caregivers reported real changes in their children's most bothersome symptoms.

The subdomain pattern tells an important story

Looking at which specific social skills improved tells us exactly what the drug is and is not doing:

Subdomain	Result	Why
Play and Leisure	Strong improvement	Dopamine-driven — the drug directly reduces excess
Interpersonal Relationships	Trend only	Oxytocin-dependent — requires VIP restoration
Coping Skills	No effect	Requires full neuropeptide cascade to be working

This pattern is exactly what you would expect from a drug that reduces chemical noise without restoring the underlying hormone system. The quieter brain can engage in play. But the deeper social connection that requires VIP and oxytocin to be working remains out of reach.

Regulatory note

L1-79 is an investigational drug and has not been approved by the FDA. Safety and efficacy have not been established. It will require a prescription upon approval and will need physician monitoring given the cardiovascular and hormonal considerations identified in the Phase 2 safety data.

SECTION 04

What L1-79 cannot fix

L1-79 is doing something real and helpful. But understanding what it cannot do is just as important for families as understanding what it can do.

It does not restore the traffic controller cells.

The SST-14 interneurons remain blocked. L1-79 reduces the demand those cells are under, and this may allow partial recovery during treatment. But the underlying blockade from the immune and methylation system remains. When the drug is stopped, the excess gradually returns.

It does not restore VIP, oxytocin, or secretin.

Because these three hormones all depend on the traffic controller cells to function, and because L1-79 does not restore those cells, the deeper social connection, bonding, and gut-brain coordination that families most want to see remains out of reach with L1-79 alone.

It does not address the immune system driver.

The original cause — the chronic immune activation and IDO1 reaction — continues operating during treatment. While behavioral presentation may appear improved, the underlying inflammatory process continues. Over time, the traffic controller cells may continue advancing toward more severe compromise while the drug is reducing visible symptoms.

It does not fix the methylation cycle.

The methylation cycle is the body's system for switching genes on and off. When it is impaired — particularly when a specific enzyme called AHCY is not working properly — the somatostatin gene cannot be fully activated even if all other problems are resolved. This constitutional methylation impairment is the single most important unresolved gap in current autism treatment.

The improvements may not last after stopping the drug.

Because L1-79 is managing a downstream consequence rather than fixing the upstream cause, benefits are largely dependent on continued treatment. When the drug is stopped, the immune system and IDO1 pathway that were never addressed continue driving the same chemical excess that the drug was suppressing.

SECTION 05

The three-part program — and why it offers more

The Biology of Autism framework proposes a hypothesis that three interventions working together could produce results that none of them achieves alone. Each addresses a different part of the problem:

L1-79

Reduces the chemical excess — the dopamine and norepinephrine overload — that the impaired traffic controller cells can no longer regulate. This quiets the brain enough that those cells, under less metabolic stress, may begin to partially recover.

The IMIG priming protocol

A carefully sequenced program of nutritional and supplement support — including luteolin, NMN, fisetin, hydroxocobalamin, sulforaphane, and forskolin — that works from below. Luteolin reduces the inflammatory blockade on the somatostatin gene. Forskolin directly activates the chemical pathway that drives somatostatin production, bypassing the blockade. NMN supports the energy supply of the cells themselves. Hydroxocobalamin supports the methylation cycle. Together these address multiple suppression mechanisms simultaneously.

IMIG (intramuscular immunoglobulin)

An immune-modulating therapy that addresses the original cause — the chronic immune activation driving the IDO1 enzyme reaction and the inflammatory blockade on the traffic controller cells. By modulating the immune system from above, IMIG removes the ceiling that has been preventing the traffic controller cells from recovering. The toxic quinolinic acid production slows. The inflammatory competition for the somatostatin gene resolves. The conditions for genuine recovery become possible.

> ***Why these three together produce something none achieves alone:*** *L1-79 reduces the demand on exhausted traffic controller cells. The priming protocol raises the metabolic supply available to those cells and drives their recovery from below. IMIG removes the immune ceiling blocking their recovery from above. When all three work together, the conditions for genuine SST-14 recovery — not just symptom reduction — become possible.*

When the traffic controller cells recover, the results go much further:

• VIP is restored — cortical brain rhythms synchronize, sensory processing becomes less overwhelming, social perception improves

• Oxytocin release is restored — properly timed, in response to real social context — enabling genuine affiliative bonding and reciprocal social engagement

• Secretin signaling is restored — gut-brain coordination improves, digestive function normalizes

• Sleep normalizes — not because melatonin was added, but because the VIP-driven brain clock synchronization is restored

• Appetite expands — because the gut SST-28 overactivity driven by multiple factors quiets down

• Social learning consolidates durably — because in a neurologically restored environment, social experiences are encoded and retained through synaptic strengthening

SECTION 06

The window of opportunity

There is an important concept in the Biology of Autism framework called the **window of reversibility.** The traffic controller cells in the brain can exist in three states.

State	Condition	Recovery potential
State 1	Blocked but structurally intact	Most direct path to recovery with the right intervention
State 2	Blocked and metabolically exhausted	Recovery possible with metabolic support; takes longer
State 3	Structural damage present	Recovery is possible but slower and more demanding — not a closed door. Recent research on brain plasticity proteins confirms that resolving neuroinflammation can reopen recovery even in significantly damaged brain.

A child treated early — while the traffic controller cells are still in State 1 or 2 — has the most direct path to meaningful, durable recovery. A child treated later faces a longer and more demanding recovery process — but not a closed door. The three-part program is most powerful when started early, but the brain's capacity for recovery, while reduced, is not eliminated at any stage.

This is also why using L1-79 alone over the long term carries an important risk: if the drug reduces visible symptoms while the underlying immune activation and IDO1 reaction continue to progress, the traffic controller cells may be advancing toward the more severely compromised third state while the child appears to be doing better. The window of opportunity may be narrowing behind the appearance of improvement. Addressing the immune system — through IMIG — is the intervention that keeps the window open.

SECTION 07

What remains unresolved — and where research is heading

The three-part program addresses the most important elements of the cascade. But there are gaps that current medicine has not yet closed, and families deserve to know about them honestly.

The methylation problem in some children is constitutional.

Some children carry genetic variants — particularly in a gene called AHCY — that impair the body's ability to clear a chemical byproduct called SAH. When SAH accumulates, it blocks the methylation cycle, which means the somatostatin gene cannot be fully activated even after all other interventions. The current three-part program reduces the amount of SAH being produced, but cannot fully resolve the clearance problem in children with this genetic variant. This remains the single most important unresolved gap in the program for genetically vulnerable individuals. Developing a targeted therapeutic to address this represents the next horizon for this population — a dedicated pharmaceutical development project that the IDA framework's patient characterization work is helping to define.

The gut entry point remains open.

Gut pH dysregulation, incomplete protein digestion, and the opioid-like molecules produced from casein and gluten remain active unless dietary intervention is also part of the program. Casein and gluten elimination reduces the opioid peptide load at source and is the most impactful single dietary intervention available.

The IDA subgroup has not yet been formally sized.

The Biology of Autism framework proposes that this specific immune and metabolic pattern is responsible for a significant portion of autism cases — possibly anywhere from a quarter to three quarters of all children meeting the diagnostic criteria. Determining the true proportion requires biomarker-based clinical research that has not yet been completed.

The research is advancing.

Decoding Autism Now is developing the scientific and clinical foundation for future investigation of the three-part program. The goal is to demonstrate that the combination produces outcomes — particularly in social bonding, communication depth, sleep, and appetite — that L1-79 alone cannot achieve. The full framework documentation is available at decodingautismnow.com.

SECTION 08

What this means for you as a parent right now

The science in this document is real, carefully documented, and built on published research. But it is important to be honest about what is established versus what is still being developed.

What is established:

L1-79 produces real and meaningful improvements in social behavior, particularly in play and activity engagement. Its safety profile is favorable compared to existing medications. The Biology of Autism framework provides a coherent mechanistic explanation of why those specific improvements occur and why deeper social bonding improvements require additional intervention.

What is a well-founded scientific hypothesis:

The three-part program combining L1-79, the priming protocol, and IMIG is predicted by the framework to produce deeper and more durable outcomes than L1-79 alone. The specific clinical predictions — improved interpersonal bonding, sleep normalization, appetite expansion, durable social learning — follow directly from the mechanism. But formal clinical trial validation of the combination program is not yet complete.

What you can do now:

Share this document with your child's physician. Ask whether your child has been evaluated for immune and metabolic contributors to their autism presentation — gut microbiome, inflammatory markers, and methylation cycle function. Ask about the priming protocol supplements, which are available now and address the metabolic and inflammatory layers of the cascade.

The most important thing:

Time matters. The window during which the traffic controller cells remain most recoverable is not indefinite. Early, comprehensive intervention — addressing the immune system, the methylation cycle, the gut, and the chemical excess — offers the best opportunity for meaningful, durable improvement. A drug that quiets the symptoms while leaving the underlying cause untreated is better than nothing. But it is not enough.

For more information on the Biology of Autism framework, the IDA cascade model, and the three-part intervention program, visit Decoding Autism Now at decodingautismnow.com