

# **A Systems-Based Hypothesis of Autism: Availability, Transport, and Cycle Completion**

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## **Author Note**

Jacob Boatman is a non-speaking individual with autism who communicates independently through typing on a standard QWERTY keyboard. Many of the concepts discussed in this paper were communicated directly by Jacob and subsequently explored, organized, and developed by the authors into the Systems-based hypothesis presented herein. The Concepts presented should be regarded as exploratory and intended to stimulate discussion and future investigation.

## **Abstract**

Traditional models of autism often focus on deficiencies, excesses, mutations, or isolated biochemical abnormalities. This hypothesis proposes an alternative framework: that a subset of individuals with autism may experience disruptions not primarily from deficiency, but from impaired availability and transport within interconnected biological systems.

In this model, the critical issue is not whether nutrients, signaling molecules, or metabolic resources exist, but whether they successfully reach their intended destinations and complete their functional cycles.

The result may be a state experienced subjectively as cognitive “noise,” internal “traffic,” impaired focus, and reduced access to thought.

## **Development of the Hypothesis**

This hypothesis emerged through years of observation, review of clinical data, genetics, metabolic pathways, and direct communication between the authors. A significant portion of the systems-level concepts described in this paper originated from discussions with Jacob Boatman, a non-speaking individual with autism who

communicates independently through typing. These communications frequently centered on themes of transport, availability, cycle completion, cognitive clarity, and internal “traffic,” which subsequently informed the development of the present hypothesis. The concepts presented herein should be regarded as exploratory and intended to stimulate discussion, investigation, and future research.

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## The Central Hypothesis

The human body functions as a series of interconnected transport networks.

Resources must:

1. Be produced or acquired.
2. Be transported.
3. Reach target cells.
4. Be utilized.
5. Complete their biological cycle.
6. Be recycled or renewed.

Failure at any point may create bottlenecks that produce downstream dysfunction despite apparently normal laboratory values.

The critical variable becomes availability rather than absolute quantity.

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## The Cycle Completion Model

According to observations and communication obtained from a non-speaking autistic individual, optimal cognitive function occurs when a biological cycle successfully completes.

Cycle completion is associated with:

- Increased mental clarity
- Improved focus
- Reduced internal noise
- Greater ability to remain on a single topic
- Improved access to thoughts

Cycle failure is associated with:

- Cognitive traffic

- Internal noise
  - Reduced focus
  - Difficulty maintaining thought continuity
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## **Serotonin as a Functional Marker**

Within this model, serotonin is not viewed solely as a neurotransmitter but as a marker of successful system flow.

When serotonin becomes “stuck,” the cycle cannot complete.

The resulting state is described as:

- Traffic
- Noise
- Reduced access to thought
- Reduced focus

The hypothesis suggests that successful serotonin utilization may reflect successful completion of a larger transport and availability network.

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## **The Role of B12**

B12 emerges as a recurring component within the proposed model.

Rather than functioning solely as a vitamin, B12 appears to act as a critical enabling resource required for cycle completion.

The hypothesis proposes:

- B12 must reach target cells.
- Cellular competition may occur when availability is limited.
- Availability may be more important than serum abundance.
- Failure of intracellular access may contribute to cycle disruption.

The question becomes:

“Is B12 available where it is needed?”

Versus

“Is B12 present?”

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## **SHMT1 and SHMT2**

The SHMT pathways are proposed as functional checkpoints within the cycle.

In this model:

- SHMT1 supports folate flow.
- SHMT2 supports downstream signaling and neurotransmitter availability.

Whether these represent literal biochemical pathways or systems-level representations remains unknown.

However, their repeated appearance suggests a potential connection between:

- Folate metabolism
  - One-carbon metabolism
  - B12 utilization
  - Neurotransmitter regulation
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## **Lipid Transport and Availability**

A recurring theme within this framework is that signaling systems may depend upon successful transport mechanisms.

This includes:

- Cellular membrane composition
- Cholesterol availability
- Lipid transport
- Receptor accessibility
- Neurotransmitter signaling efficiency

The hypothesis proposes that signaling dysfunction may arise from impaired transport and delivery rather than simple production deficits.

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# The Availability Principle

The central principle can be summarized as:

Presence does not equal availability.

A nutrient may exist but not arrive.

A signal may exist but not reach its target.

A system may be supplied but unable to complete its cycle.

In this framework, many observed abnormalities may represent downstream consequences of transport bottlenecks rather than primary deficiencies.

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## Future Directions

Future investigation should focus on:

- Intracellular nutrient availability
- Transport pathways
- Lipid trafficking systems
- B12 transport and utilization
- Folate cycle dynamics
- Neurotransmitter availability
- Mitochondrial support of transport networks
- Whole genome sequencing correlations

The ultimate goal is not simply to identify what is present, but to understand what successfully arrives.

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## Conclusion

This hypothesis proposes that a subset of autism may be understood through the lens of transport, availability, and cycle completion.

When biological resources successfully reach their intended destinations, signaling networks operate efficiently, internal noise decreases, and cognitive focus improves.

When transport fails, traffic develops, signals become unavailable, cycles remain incomplete, and cognitive performance declines.

Under this model, the fundamental question shifts from:

“What is missing?”

to

“What cannot get where it needs to go?”