

OPINION

How do we confirm a grand signaling theory?

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Our latest [Classic Paper Review](#) traces the evolution of the excitation/inhibition (E/I) imbalance hypothesis — a popular theory suggesting that a disruption in the delicate interplay between excitatory and inhibitory activity in the brain, especially during critical periods of development, may underlie autism.

The review garnered a number of comments, including a salty debate among some researchers.

[Read the full article here »](#)

Proponents of the E/I imbalance hypothesis propose that many of the different risk factors for autism feed into this one unifying pathway. Others caution that this thinking oversimplifies a complex process.

What do you think?

- **What specific evidence is needed to help confirm the E/I imbalance theory of autism? Would a theory like this remain valid and useful if it continues to be supported by neurological data, but not by genetics?**
- **Scientists have a fairly clear picture of how the abundance or paucity of certain proteins in the brain can cause abnormal communication between brain cells. But how does this abnormal activity lead to symptoms associated with autism and related disorders? How can we design experiments to illuminate the path from**

signaling dysfunction — such as E/I imbalance — to behavior?

Share your thoughts in the comments section below. Or, to dig deeper, continue the conversation in the moderated **SFARI Forum** for researchers. Not yet a member? Learn how to register **here**.

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