

## CASE ILLUSTRATIONS

### CASE ILLUSTRATION 4.1 — METABOLIC FRAGILITY FROM INSUFFICIENT GLU & GLN

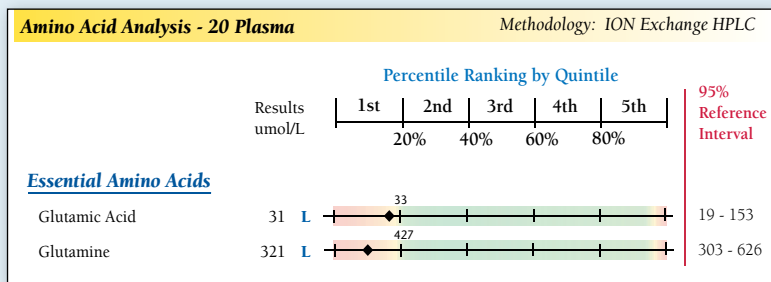
History: Four years prior to the testing date, this 30-year-old clinician had recurrent urinary tract infections for several months, treated with antibiotics. She says, “Then suddenly my body just broke down. My immune system just collapsed and I had infections everywhere: lungs, ears, nose, bladder, etc. I got allergic to everything I ate.”

Current diagnosis and symptoms: Interstitial cystitis, frequent upper respiratory infections, constipation and bloating.

The amino acid profile was measured on a plasma specimen. The results show concurrent low Glu and Gln. Although neither amino acid is below the 95th percentile ranges, the dependent nature of this pair makes the results quite unusual. The physiological state might be described as one of metabolic fragility because of difficulty in responding to systemic pH and ammonia production shifts.

She reports that her bladder and general body pain is much worse in the morning, a time when systemic pH is under stress from cortisol-stimulated organic acid and ammonia formation.

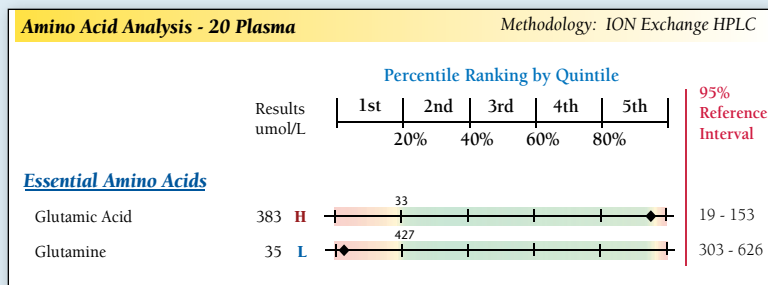
The most common amino acid-based treatment in such cases is to use customized free-form essential amino acid mixtures formulated as described in this chapter. Such low glutamic acid and glutamine patterns are very frequently accompanied by multiple imbalances in utilization of EAA. ❖



### CASE ILLUSTRATION 4.2 — HIGH GLU/GLN RATIO IN A PATIENT WITH AUTISM

A routine amino acid profile was ordered along with other metabolic testing on a severely autistic 3-year-old girl. Her plasma glutamate was very elevated, 383 μM (19–153), while glutamine was only 35 μM (303–626).

Extreme elevations of multiple organic acids were found in urine, consistent with acidemia and calcium was very high in erythrocytes, indicating difficulty maintaining the membrane calcium pumping system. Her plasma fatty acid profile showed an extremely high AA/EPA ratio that is associated with the pro-inflammatory state and her urine contained very high quinolinate and kynurenate levels, indicating interferon-γ-stimulated macrophage inflammatory response. Since her urinary p-hydroxybenzoate was very elevated, one area of suspected etiology is a severe overgrowth of bacteria or parasites producing an inflammatory response in the gut.



This case has multiple metabolic disturbances that can be affecting systemic pH and brain chemistry. Her situation is exacerbated by the loss of control over conversion of glutamate to glutamine. There is the possibility of genetic impairment of hepatic glutamine synthetase, but correction of potential dependent metabolic issues could be undertaken before attempting to confirm such diagnosis. ❖