

DEEPER THAN SYMPTOMS

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RESEARCH DECODED

# The Root Cause Methylation Guide

*What an MTHFR result really means — and what you can actually do about it.*

## Inside this guide

A fear-free explainer of MTHFR and methylation, what enzyme activity really means, the three tests that guide a real plan, folate-rich foods, and the one caveat most articles skip.

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A free resource from

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## What MTHFR Actually Means (Without the Fear)

An MTHFR mutation is one of the most common genetic variants in the US — about 1 in 3 Americans carry at least one copy. MTHFR is an *enzyme*, not a disease. Its job is to convert the folate you eat into its active, usable form (L-methylfolate), which powers **methylation** — the process your body uses to regulate genes, brain chemicals, hormones, and detox. A variant *slows* that conversion. It's a slowdown, not a shutdown.

### The bottom line, up front

Your gene shows a *tendency*. Your homocysteine shows the *reality*. Functional medicine reads them together to figure out what **you** actually need — not what a variant predicts for the population.

## The Two Variants — and What Enzyme Activity Really Means

Variant / status	Effect	What it means for you
C677T (one copy)	Enzyme runs at ~65% capacity	Usually well-covered by adequate dietary folate.
C677T (two copies)	Enzyme runs at ~30% capacity	Sounds dramatic, but 30% is still meaningful folate processing.
A1298C	Generally milder effect	Can still influence related pathways; context matters.

*For most people, the right nutrition and lifestyle largely compensate. Supplement decisions should never be made from a gene result alone.*

## The Testing That Actually Guides a Plan

In conventional medicine you might get a genetic panel — and then not much guidance after. A functional workup looks at what your genes are *doing in your body right now*.

- 1 MTHFR genotyping** — confirms whether you carry C677T and/or A1298C.
- 2 Homocysteine** — the functional readout of how well methylation is running (often left off standard panels).
- 3 Serum and active B12, plus folate** — the nutritional raw materials that feed the whole pathway.

### Where symptoms hide

A homocysteine of 11–12  $\mu\text{mol/L}$  might be flagged “within range” on a standard US report — but many functional practitioners aim closer to 7–9  $\mu\text{mol/L}$ . That gap is where symptoms live for a lot of people, even after being told everything looks fine.

## How to Support Methylation — Food First

The field of **nutrigenomics** studies how nutrients interact with your genes. Its core lesson: you can actively support these pathways. If your enzyme converts folate slowly, the workaround is to give your body folate that’s already in its active form — and food is the best starting point.

### Folate-rich foods

- Dark leafy greens: spinach, kale, romaine, arugula
- Legumes: lentils, black beans, chickpeas
- Avocado
- Asparagus and broccoli

### The wider network

- Methylation doesn’t run on folate alone — it also needs **B2, B6, B12, and magnesium**.
- Active forms some practitioners consider: **L-methylfolate (5-MTHF)** over synthetic folic acid, and **methylcobalamin** (active B12) — decided with a clinician, not from a gene result.

### A critical caveat

More is not always better. Some people — especially those sensitive to methyl donors — feel *worse* on high-dose methylfolate. This is exactly why supplement decisions belong with a practitioner who reads your genes, labs, and symptoms together.

### Your next step

Your gene is a tendency; your labs are the reality. Review both with a practitioner — and read the full methylation guide at [deeperthansymptoms.com](https://deeperthansymptoms.com).