

Homocysteine Methylation- A Nutrigenetic Perspective

Anusha Sunder*

Doctorate in Life Science/Human Nutrition, Accredited Certification in Nutrigenetics; Lead Scientist and Nutrigenetic Expert, Xcode Life Sciences, Pvt. Ltd. Chennai, India

***Corresponding author:** Anusha Sunder, Doctorate in Life Science/Human Nutrition, Accredited Certification in Nutrigenetics; Lead Scientist and Nutrigenetic Expert, Xcode Life Sciences, Pvt. Ltd. Chennai, India

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ABSTRACT

Ebola virus disease (EVD) continues to pose a significant challenge as an emerging infectious disease in Africa, presenting with difficulty in urgency, high fatality rates and transmission through health systems. Even though improvements have been introduced across various aspects in the medical field, such advancements have not been able to eliminate existing challenges. In 2026, the Democratic Republic of the Congo (DRC) and Uganda suffered outbreaks of the Bundibugyo virus, showing a certain level of chance for potential spread of the disease. The outbreak in the DRC, however, continued across provinces and resulted in the largest outbreak in the history of EVD noted in the country. The key reason of the widening of the outbreak was the absence of the vaccine against Bundibugyo virus as well as antiviral therapy. This paper assesses the development of the Ebola crisis in Africa, looking at epidemic transmission, clinical characteristics, problems with diagnosis, differences in the virus, and improvements in treatment and vaccination. Special emphasis has been placed on the Bundibugyo outbreak that took place in 2026. The paper contends that effective control of the Ebola epidemics requires an integrated system that combines quick diagnostics, genomic surveillance, and adaptable methods of medical treatment and engagement of the community.

Keywords: Ebola Virus Infection; Bundibugyo Virus; Continent of Africa; Newly Emerging Infectious Diseases; The Category of Viral Hemorrhagic Fever; Preparedness for Disease Outbreak; Vaccinations and use of Monoclonal Antibodies

Introduction

Our body cells (nearly a trillion) are just like us, they respire, digest food, assimilate nutrients, and, excrete waste/toxic products. Their normal functioning is essential for us to be in good health. As cells perform their routine functions, they release certain by-products like homocysteine, which should be promptly cleared or recycled with the help of efficient enzymes. These enzymes require nutrients, like folate, to efficiently recycle the toxic homocysteine into an essential amino acid (methionine), through a process called methylation. A natural process called methylation keeps our body's inflammation under control. A disturbance in this process can affect health, and relates with everyday pain-points like acne, hives, tiredness, diarrhea, stomach cramps, allergy, asthma, sleep disturbances and weight gain. The best way to identify its likelihood is through a painless genetic assessment. Right nutrients and suitable dietary recommendations can normalize this process and enhance health through inflammation control.

Health Impact of Disturbed Homocysteine Methylation

A delay in homocysteine clearance or recycling can trigger inflammation. And undue inflammation is linked with health disorders like obesity, high blood pressure, heart ailments, stroke, nerve problems, joint ache, accelerated skin ageing, and gut problems like irritable bowel syndrome.

What does a Genetic Report on MTHFR & Homocysteine Methylation Reveal?

The enzymes that recycle homocysteine are controlled by genes, and a genetic report tells the innate tendencies for –

- Efficiency of methylation pathway and promptness of homocysteine clearance
- Precise nutrient requirements for efficient methylation/recycling of homocysteine

Unfavorable genetic changes can result in delayed homocysteine clearance, implicating the probability of health disorders due to undue inflammation. Each unfavorable genetic change requires a precise nutrient for its management and this is tailor-made based on the individual's genetic results. Thus quickening homocysteine clearance and counterbalancing inflammation are achievable through gene-specific nutrients.

Science Behind Your Genetic Results

Inflammation control is achieved through optimal homocysteine methylation. And this relies on the expression and function of enzyme-encoding genes in the biological pathway. The following table (Table 1) explains the crucial biological pathways of homocysteine methylation, along with the core enzyme-coding genes and their precise nutrients [1-5].

Table 1: Nutrigenetics of homocysteine methylation.

Homocysteine methylation and inflammation control		
Biological pathway	Enzyme-encoding Gene	Gene-specific nutrient (Nutrigenetic management)
Homocysteine recycling (Folate and Methionine Cycles) Cycles): Identifies bottlenecks in methionine production, the body's primary methyl donor (S-Adenosylmethionine).	MTHFR	Folate/vitamin B9, Riboflavin/vitamin B2
	MTR	Cobalamin/ vitamin B12, Betaine
	MTRR	Cobalamin/ vitamin B12
	MTHFD1	Folate/vitamin B9
Homocysteine clearance (Transsulfuration Pathway & Nitric oxide levels) Assesses glutathione synthesis, and nitric oxide levels, which are crucial for detoxification and antioxidant defense against inflammation.	CBS	Pyridoxine /vitamin B6
	AHCY	Niacin/Vitamin B3
	CPS1	Cobalamin/ vitamin B12, Pyridoxine /vitamin B6, Glutamine/ glutamate, Nattokinase (from the Japanese fermented food Natto), Bergamot oil (from bergamot orange, a citrus fruit)
	BHMT	Choline/vitamin B4 and its derivative betaine
	SHMT	Pyridoxine /vitamin B6
	CTH	Pyridoxine /vitamin B6
	MAT1A	Methionine, cysteine, glycine, serine, glutathione, potassium, magnesium
	GNMT	Betaine
Inflammation control (Neurotransmitter Regulation) Analyzes gene variants that affect feel-good hormone levels (dopamine, serotonin, norepinephrine) and regulate inflammation	NOS3	Tetrahydrobiopterin/BH4, Vitamin C, Riboflavin/vitamin B2, omega-3 fatty acids, iron, magnesium, co-enzyme Q10/ ubiquinol, arginine, citrulline, Nattokinase (from the Japanese fermented food Natto)
	MAO-A	Probiotics/Prebiotics & omega-3 fatty acids
	COMT	Probiotics/Prebiotics, omega-3 fatty acids, Magnesium

Note: The main enzyme that recycles homocysteine is MTHFR (Methylene tetrahydro folate reductase), and it is ably supported by other enzymes including MTR (methionine synthase), MTRR (methionine synthase reductase), MTHFD1 (methylenetetrahydrofolate dehydrogenase 1), CBS (cystathionine beta-synthase), AHCY (adenosylhomocysteine hydrolase), CPS1 (carbomyl phosphate synthetase), BHMT (betaine-homocysteine S-methyltransferase), SHMT (serine hydroxymethyl transferase), CTH (cystathionine-gamma-lyase), MAT1A (methionine adenosyltransferase), GNMT (glycine N-methyltransferase), NOS3 (endothelial nitric oxide synthase/eNOS), MAO-A (monoamine oxidase A), COMT (catechol-o-methyltransferase).

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