NutriPATH
INTEGRATIVE PATHOLOGY SERVICES
P: 1300 688 522
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SAMPLE REPORT
09-May-1990 Female

16 HARKER STREET
BURWOOD VIC 3125

Dr.SAMPLE REPORT
TEST HEALTH CENTRE
123 TEST STREET
BURWOOD VIC 3125

LAB ID : 3814205
UR NO. :
Collection Date : 09-May-2022
Received Date: 09-May-2022

3814205

INTEGRATIVE MEDICINE

BLOOD - PLASMA | Result | Range | Units

Methylation Profile.

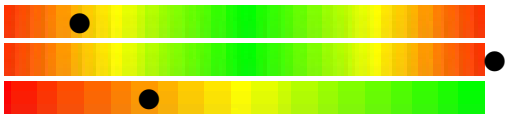
Methionine Metabolism Pathway

S-Adenosyl Methionine (SAMe) is the most active methyl group donor in the body. Endogenously, SAMe is formed in the Methionine Metabolism Pathway (Transmethylation). S-Adenosyl Methionine (SAMe) is formed through a reaction involving the amino acid methionine and ATP.
As SAMe releases methyl groups to the methylation process, it is converted to S-Adenosyl Homocysteine (SAH), which in turn is converted to homocysteine. Thereafter, re-methylation of homocysteine to form methionine is required to continue the Methionine Metabolism Pathway.
Methyl groups are formed through the Folate Metabolism Cycle and donated to homocysteine which is converted to Methionine, which re-enters the Methionine Metabolism Cycle to form SAMe.

Importantly, SAMe functions to promote the following;

synthesis of DNA and RNA	(for Gene Regulation)
synthesis of Glutathione	(for detoxification & metals removal),
synthesis of CoQ10, creatine, carnitine	(for energy and mitochondrial function).
inhibition of Histamine	(for anti-inflammatory effects)
crucial in neurotransmitter balance	(for conversion of Serotonin to Melatonin for promotion of sleep)

	Result	Range	Units
S-Adenosyl Methionine	*68.0* *L	86.0 - 145.0	nmol/L
S-Adenosyl Homocysteine	*39.0* *H	10.0 - 22.0	nmol/L
SAM/SAH Ratio	*1.7* *L	> 4.0	RATIO



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Folate Metabolism Pathway

The Folate Metabolism Pathway is required for the formation of Methyl groups that are utilised in the Methionine Metabolism Pathway for methylation purposes. It is also the regulator of the Methionine Metabolism Pathway.
Folates are naturally occurring vitamins and are found in numerous foods. In contrast Folic Acid, is a synthetic form of folate.

Methyl groups are acquired from either Trimethylglycine (TMG) or 5methyltetrahydrofolate (5MTHF). The former reaction however only occurs in the kidney and liver, whereas the latter reaction occurs in most cells of the body. Hence, the latter reaction is the preferential pathway.

5MTHF is the most abundant folate form in plasma and as such is the most important form for the methylation process. 5MTHF is converted to THF via the MTR enzyme and the MTHFR enzyme. In the process a Methyl group is donated to homocysteine to form methionine. The effectiveness of this process is influenced by the genetic polymorphism of the MTHFR enzyme. MTHFR mutations don't allow efficient processing of folic acid to a readily utiliseable form (5MTHF).

FOLINIC ACID (5-formyl THF), is an active and reduced form of folate. In the body, folinic acid may be converted into any of the other active forms of folate. Supplying the body with folinic acid bypasses many of the required metabolic steps, and it is rapidly converted to 5MTHF.

TETRAHYDROFOLATE (THF) is the basic, reduced form of folate from which other forms of reduced folate are made.

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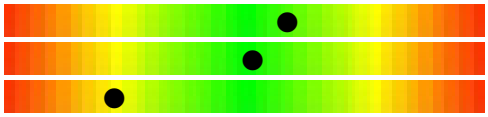
Tetrahydrofolate	4.2	0.6 - 6.8	nmol/L	
Folinic Acid	21.7	9.0 - 35.5	nmol/L	
5-Methyl Tetrahydrofolate	6.9	6.6 - 39.9	nmol/L	



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Methylation Comments

LOW S-ADENOSYL METHIONINE (SAMe) LEVEL:
SAMe level may be low due to the following;
Inadequate Methionine (the chief substrate for methylation) either through;

1. Inadequate dietary intake (Poor Diet, Vegetarian/Vegan Diet, GIT dysfunction, Hypochlorhydria)
   Improve dietary methionine intake (cheeses, dairy, poultry, meats, nuts) combined with magnesium,
   Vit B6, folate, Betaine (TMG) and Vit B12 support.
   Supplementation with Methionine (Must also include magnesium, Vit B6, folate, Betaine (TMG) and
   Vit B12 support).

2. Inadequate Homocysteine metabolism
   Possible causes: Use of Niacin (depletes methyl groups), Antacids (depletes Vit B12)
   Assess Active B12 and Red Cell Folate levels

3. Inadequate Magnesium (chief cofactor for SAMe synthesis)
4. Inhibition of enzymic activity or
5. genetic/chemical influences.

Consider SAMe supplementation - 200-400mg daily, taken on an empty stomach (capsules should be foil packed to retain potency). Also Methionine, Magnesium, B3 and increase protein intake.
PRECAUTION:
Prior to considering SAMe or Methionine supplementation, ensure concurrently SAH levels are not elevated.
If SAH is elevated, consider firstly improving SAH clearance/metabolism (via Homocysteine) towards Methionine (Methionine Synthesis pathway) or Cysteine/Glutathione (Transulphuration pathway).
Consider TMG (trimethylglycine) or Betaine to lower SAH.
Thereafter consider the use of SAMe or Methionine supplementation.

ELEVATED S-ADENOSYL HOMOCYSTEINE (SAH) LEVEL:
Elevated SAH levels suggest inadequate homocysteine metabolism to methionine. Check Homocysteine levels.

As SAH is a strong inhibitor of the methylation process, its levels need to be regulated.
May be due to NAD cofactor deficiency (B3) or commonly SNPs in AHCY.
Consider TMG (trimethylglycine) or Betaine to lower SAH.

LOW METHYLATION INDEX:
Balancing the SAMe/SAH ratio is important to facilitate optimal enzymic activites in the methylation process.
A reduction in this ratio, below the reference range, is reflective of a decrease in methylation activity.

Tests ordered: CFee,5101

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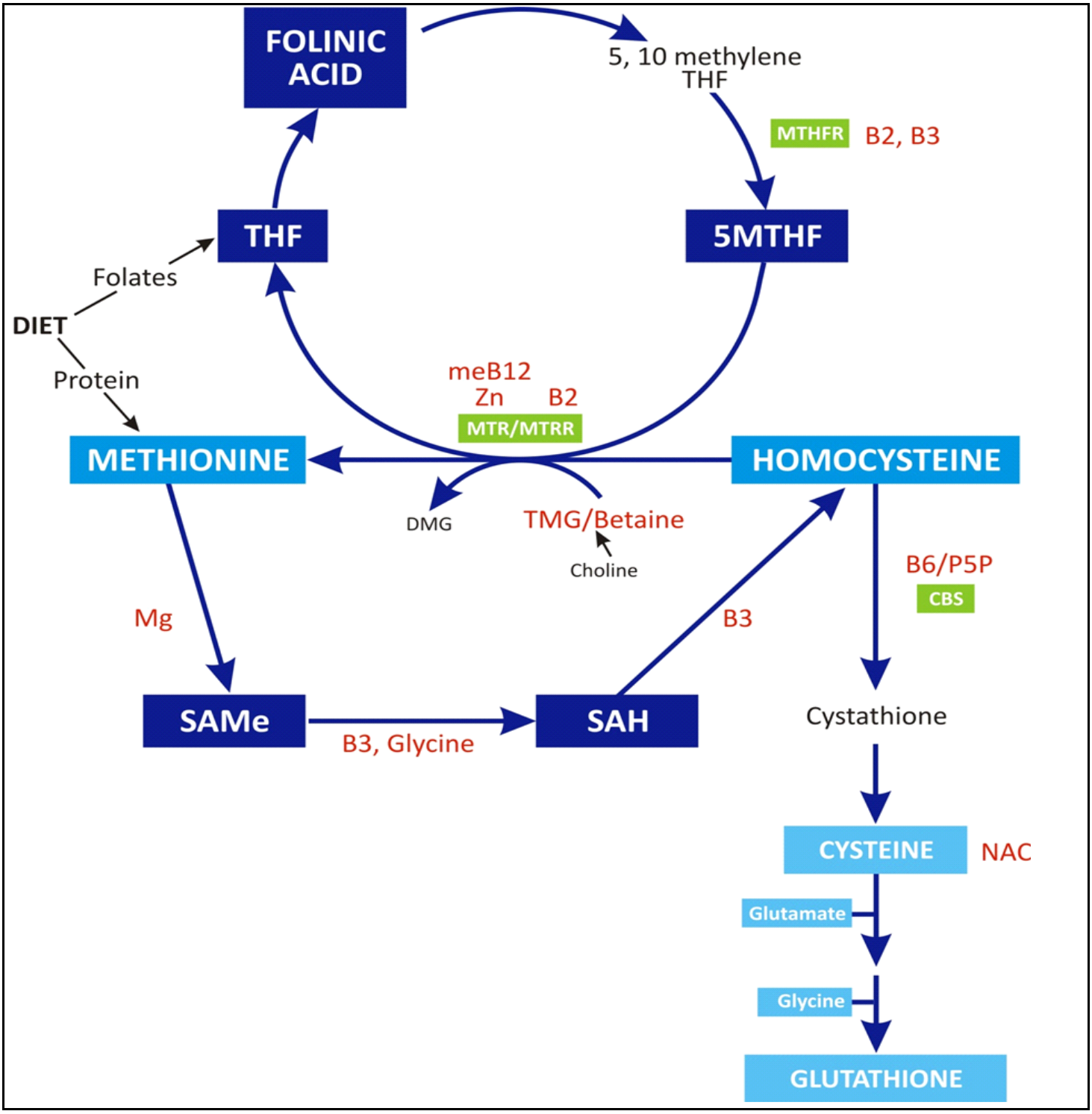

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