NutriPATH PATHOLOGY · PATIENT REPORT
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26116-0010

Dr Test Doctor (Test Doctor) Test Clinic. 123 Test Street, Test Suburb Victoria 3125

Lab ID
Patient ID P000060
Ext ID 26116-0010

Test Patient

Sex: Female • 56yrs • 01-Jan-70
123 Home Street, Test Suburb VIC 3125

RECEIVED
26-Apr-26

PFEIFFER PROFILE - COMPREHENSIVE

	Collected
Specimen type - Blood-Serum	15-Apr-26
Specimen type - Blood-K2EDTA	15-Apr-26
Specimen type - Blood-Trace Metal	15-Apr-26
Specimen type - Blood-Whole	15-Apr-26
Specimen type - Blood-EDTA	15-Apr-26

COPPER/ZINC BALANCE

TEST	RESULT	H/L	REFERENCE	UNITS
Copper	20.8		(13.0-22.0)	umol/L
Zinc	10.1		(9.0-19.0)	umol/L
Copper/Zinc Ratio	2.06	H	(0.54-1.68)	ratio
Caeruloplasmin	0.24		(0.20-0.60)	g/L
% Free Copper	45	H	(5-25)	%

BIOCHEMISTRIES

TEST	RESULT	H/L	REFERENCE	UNITS
Histamine	3.9	L	(5.0-60.0)	ng/mL
Vitamin D	45	L	(50-200)	nmol/L
Homocysteine	17.0	H	(5.0-15.0)	umol/L

METHYLATION FACTORS

TEST	RESULT	H/L	REFERENCE	UNITS
S-Adenosyl Methionine (SAMe)	81.0	L	(86.0-145.0)	nmol/L
S-Adenosyl Homocysteine (SAHe)	24.3	H	(10.0-22.0)	nmol/L
SAMe/SAHe Ratio	3.3	L	(>4.0)	ratio

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PFEIFFER PROFILE COMMENT

ELEVATED FREE COPPER:

Consider chelation therapy, Vit C, Zinc, Molybdenum, Glutamine, Histidine and threonine. Optimal free Copper of 5-25% is recommended by Dr B Walsh. Retesting Zinc and Copper levels after 3 months of treatment is recommended. Signs of Cu overload include hyperactivity, skin sensitivity to metals. Skin tags, estrogen dominance, emotional meltdowns, tinnitus, abnormal periods.

ELEVATED Cu/Zn RATIO:

The ratio of copper to zinc is clinically more important than the individual levels of each analyte. Elevated copper/Zinc ratios can be particularly serious for persons with low blood histamine (over methylation). This combination of imbalances has been associated with anxiety, panic disorders, paranoia and hallucinations.

LOW WB HISTAMINE LEVELS:

(If following Pfeiffer protocols Normal Whole Blood Histamine is considered 33 - 65 ng/ml).

Low Whole Blood Histamine is otherwise known as Histapenia. In assessing Histamine levels, Diamine Oxidase (DAO) should also be tested concurrently. Serum/plasma copper levels in histapenic patients are often abnormally high. As copper is a brain stimulant (and also destroys histamine), the elevated serum/plasma (and presumably brain) copper level probably accounts for many visible symptoms, including the low blood histamine level. Behavioral symptoms in high-copper histapenia include paranoia and hallucinations in younger patients. In older patients, depression may predominate. Some studies of schizophrenics have revealed high blood copper, as seen in histadelia, with low urinary copper (showing that copper is being retained) as well as low blood zinc.

TREATMENT SUGGESTIONS: Treatment protocol consists of the administration of Amino Acid Histidine, (converts to histamine), a diet high in protein, supplement with B6, Folic Acid & B12 injections as they increase histamine levels while lowering the degree of symptoms. Zinc and manganese with vitamin C remove copper from the tissues. Copper destroys histamine and therefore as copper levels decrease, histamine levels should return towards normal. With this treatment the high blood copper is slowly reduced and symptoms are slowly relieved in several months' time. Treating histadelia is not a quick process. Early noticeable results/improvements may not be seen until close to 6-10 weeks. Treatment can take more than 12 months to complete. To find out more about this see Mental Illness: The Nutrition Connection, a book by Carl Pfeiffer. The prognosis is seen to be good if the histadelic patient cooperates with treatment and works to give up detrimental addictions.

VITAMIN D COMMENT:

Vitamin D adequacy: ≥50 nmol/L at the end of winter (level may need to be 10–20 nmol/L higher at the end of summer, to allow for seasonal decrease).

Mild vitamin D deficiency: 30–49 nmol/L

Moderate vitamin deficiency: 12.5–29 nmol/L

Severe vitamin D deficiency: <12.5 nmol/L

Consider measuring vitamin D by LCMS if the result does not correlate clinically or does not change with vitamin D supplementation.

ELEVATED HOMOCYSTEINE:

Elevated homocysteine levels may result from low levels of folic acid, vitamin B6, or vitamin B12, some drugs and renal impairment, as well as several inherited defects. However, an elevated plasma homocysteine may also be due to high protein/Paleo diet.

The following genetic SNPs in CBS, MTHFR, MTR, MTRR may also impact levels of plasma homocysteine.

NOTE: Fasting overnight is required for this test. A falsely elevated result can occur if the blood has not been preserved appropriately and there is a delay in separation of the plasma from cells.

METHYLATION COMMENT

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LOW S-ADENOSYL METHIONINE (SAMe) LEVEL:

Description: S-adenosylmethionine (SAM) is the universal methyl donor in mammalian biology, synthesised from methionine and ATP by methionine adenosyltransferase (MAT1A/MAT2A). It donates its methyl group to over 200 substrates including DNA (via DNMT), histones (via HMT), phosphatidylcholine (via PEMT), catecholamines (via COMT), and creatine (via GAMT), generating S-adenosylhomocysteine (SAH) as the by-product.

Clinical Significance: Low plasma or erythrocyte SAM indicates impaired methylation capacity, which can arise from methionine deficiency, folate or B12 deficiency (impairing homocysteine remethylation back to methionine), MAT1A deficiency, or Elevated SAH (which competitively inhibits SAM-dependent methyltransferases). Low SAM is associated with hepatic steatosis, depression (SAM depletion impairs monoamine methylation), epigenetic dysregulation, impaired phospholipid synthesis (membrane integrity), and Elevated homocysteine. The SAM/SAH ratio is a more informative marker of functional methylation capacity than SAM alone.

Suggested Treatment: Address upstream deficiencies: ensure adequate methionine, folate (5-MTHF), B12 (methylcobalamin), B6 (P5P), riboflavin, and zinc. SAMe (S-adenosylmethionine) supplementation on an empty stomach is the direct intervention, with evidence for hepatic steatosis, depression, and osteoarthritis. Assess homocysteine, SAH, and the SAM/SAH ratio to determine the primary driver of SAM depletion.

S-ADENOSYL HOMOCYSTEINE (SAH) ELEVATED:

Description: SAH is a potent competitive product inhibitor of all SAM-dependent methyltransferases (Ki approximately 1-5 uM), with inhibitory potency often exceeding that implied by simple competitive kinetics due to tight binding to the methyltransferase active sites.

Clinical Significance: Elevated plasma SAH is the strongest independent predictor of impaired methylation capacity, and is a more sensitive indicator of methylation insufficiency than homocysteine alone. Elevated SAH inhibits DNA methyltransferases (DNMTs), histone methyltransferases, COMT (catecholamine metabolism), PNMT (adrenaline synthesis), GNMT, and PEMT (phosphatidylcholine synthesis), creating broad downstream methylation insufficiency. SAH accumulates when SAHH activity is impaired (folate or B12 deficiency slow homocysteine remethylation, causing SAH to accumulate through the reversible SAHH reaction) or in AHCY gene deficiency. Elevated SAH is independently associated with cardiovascular disease, cognitive decline, and epigenetic dysregulation.

Suggested Treatment: Address folate (5-MTHF) and B12 (methylcobalamin) deficiencies as the primary drivers of SAH accumulation through impaired homocysteine remethylation. Assess and supplement riboflavin (B2, MTHFR cofactor), B6 (P5P), and zinc. Evaluate homocysteine and the SAM/SAH ratio. If AHCY deficiency is suspected (markedly Elevated SAH from birth with myopathy and developmental delay), refer for specialist evaluation.

SAM/SAH RATIO LOW:

Description: The SAM/SAH ratio (methylation index) is the most clinically informative measure of cellular methylation capacity, as SAH is a competitive inhibitor of all SAM-dependent methyltransferases. A low ratio indicates that SAH accumulation is limiting methyltransferase activity relative to SAM availability, regardless of absolute SAM concentration.

Clinical Significance: A low SAM/SAH ratio (below 4.0) indicates functional hypomethylation capacity, impairing a broad range of methylation-dependent reactions including DNA methylation (epigenetic regulation), histone methylation (chromatin remodelling), phosphatidylcholine synthesis (membrane integrity and VLDL secretion), catecholamine metabolism (via COMT), adrenaline synthesis (via PNMT), and myelin maintenance (via PEMT-dependent phospholipid production). A low ratio is strongly associated with cardiovascular risk (Elevated homocysteine and impaired PEMT), hepatic steatosis (impaired VLDL assembly), depression, cognitive decline, and epigenetic dysregulation contributing to cancer risk. The low ratio is most commonly caused by Elevated SAH (from folate, B12, or riboflavin deficiency slowing homocysteine remethylation) rather than low SAM.

Suggested Treatment: Identify the primary driver - evaluate SAM, SAH, and homocysteine individually. If SAH is Elevated : supplement 5-methyltetrahydrofolate (5-MTHF), methylcobalamin (B12), P5P (B6), and riboflavin-5-phosphate (B2). If SAM is low: consider SAMe supplementation. Reassess the SAM/SAH ratio and homocysteine after 8-12 weeks of treatment.

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Methodology

Automated Chemistry/Immunochemistry, Inductively Coupled Plasma Mass Spectrometry (ICP-MS), Enzyme-Linked Immunosorbent Assay (ELISA), Liquid Chromatography-Mass Spectrometry (LC-MS/MS/MS)