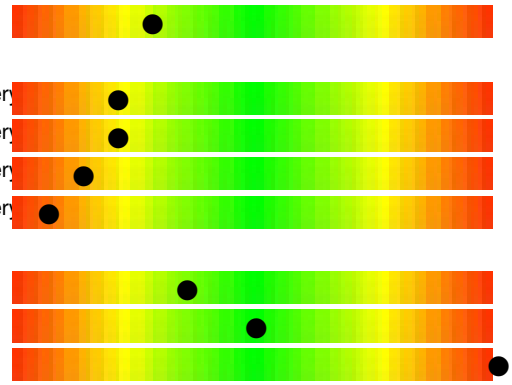
NutriPATH
Integrative Pathology Services
P: 1300 688 522
E: info@nutripath.com.au
A: PO Box 442 Ashburton VIC 3142

Date of Birth : 31-Oct-1973
Sex : M
Collected : 24-Aug-2016

INTEGRATIVE MEDICINE

URINE, SPOT	Result	Range	Units
DETOXIFICATION CAPACITY PROFILE			
PHASE I (OXIDATION)			
Caffeine Clearance	**0.7**	0.5 - 1.6	ml/min/Kg
PHASE II (CONJUGATION)			
Glutathionation	***5.2*** ***L**	5.6 - 11.4	% Recovery
Glycination	***27.9*** ***L**	30.0 - 53.0	% Recovery
Sulphation	***11.6*** ***L**	16.0 - 36.0	% Recovery
Glucuronidation	***9.0*** ***L**	27.0 - 56.0	% Recovery
RATIOS			
PHASE I / PHASE II - Sulphation	**6.0**	3.5 - 13.0	RATIO
PHASE I / PHASE II - Glycination	**2.5**	1.3 - 3.5	RATIO
PHASE I / PHASE II - Glucuronide	***7.8*** ***H**	1.9 - 4.2	RATIO



(*) Result outside normal reference range (H) Result is above upper limit of reference rang (L) Result is below lower limit of reference range

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Liver Detox. Profile Comments

The Detoxification Capacity Profile is a functional test to assess the ability of an individual to process caffeine, aspirin, and paracetamol by assessing certain metabolites in saliva and urine specimens measuring the different phases of liver detoxification.

Adequate Phase I (P450) liver enzyme detoxification activity. Within normal limits.
LOW GLUTATHIONE CONJUGATION
(acetaminophen mercapturate recovery):

GLUTATHIONATION is responsible for the conjugation of Paracetamol, Pesticides, Toxic Metals (Hg, Pb, Cd), Antibiotics (Penicillin, Tetracycline) Alcohol, Petroleum distillates.

Suspect:
a) Insufficient glutathione conjugation/Difficult removal of toxins from the body.

Possible causes:
a) Depletion of reduced glutathione,
b) Excess exposure to xenobiotics and/or free radical production,
c) Impairment of other Phase 2 pathways, resulting in compensation by GSH conjugation
d) Inadequate nutrient precursors and cofactors,
e) Induced P450 activity (more compounds to detoxify),
f) Genetic uniqueness,
g) Enhanced bile production (mercapturate elimination via the bile).
h) Chronic diseases (arthritis, CVD, diabetes)

Consider the following actions:
a) Identify and reduce sources of xenobiotic exposure and free radical generation,
b) Increase intake of GSH and its precursors,
c) Reduced glutathione, N-acetylcysteine, glycine, L-methionine, L-glutamine,
d) Address impairments in other Phase 2 pathways,
e) Address induced P450 system, to reduce burden on GSH conjugation,
f) Increase intake of nutrient cofactors: Zn, Cu, riboflavin, niacin, selenium, Mg, vitamin B6, B12, folic acid,
g) Increase intake of cruciferous vegetables (induces conjugating enzyme).
h) Increase alpha Lipoic acid, St Mary's thistle, Curcumin, Flavinoids

LOW GLYCINE CONJUGATION
(salicyluric acid recovery):

GLYCINATION is responsible for the conjugation of salicylic acids, benzoic aids, and phenylacetic acids (commonly found in nuts and cigarettes).

Suspect: Insufficient glycine conjugation/Difficult removal of toxins from the body.

Possible causes:
a) Insufficient glycine available,
b) Insufficient nutrient cofactors,
c) Underlying hepatic disease,
d) Genetic uniqueness.

Consider the following actions:
a) Increase intake of glycine,
b) Increase intake of nutrient cofactors,

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c) Cysteine, pantothenic acid, Mg (coenzyme A required for activation of the metabolite to be conjugated),
d) Increase alkaline ash foods to enhance glycination
e) Reduce salicylates intake

LOW SULPHATION
(acetaminophen sulphate recovery):

SULPHATION is responsible for the conjugation of steroid hormones (estrogens, progesterone, DHEA), phenols (histamine, Dopamine, gallic acid, coumarin), catecholamines (adrenalin, noradrenalin)

Suspect: Insufficient sulfation/Difficult removal of toxins from the body.

Possible causes:
a) Depletion of inorganic sulphate,
b) Excess exposure to xenobiotics or free radical production,
c) Inadequate dietary sulphate available,
d) Insufficient nutrient cofactors,
e)Induced P450 activity,
f) Impaired sulphoxidation ability (especially if high cysteine/sulphate ratio, with low plasma sulphate),
g) Molybdenum insufficiency, especially if low plasma sulphate,
h) Molybdenum or vitamin B6 excess (can inhibit sulfation),
i) Underlying hepatic disease,
j) Genetic uniqueness,
k) Sulphation may be low if a shared pathway (e.g. glucuronidation) is taking on more compound than usual.

Consider the following actions:
a) Rule out excess xenobiotic exposure, especially if elevated caffeine clearance,
b) Consider foods or supplements containing sulphate precursors, unless high cysteine/sulphate ratio with low plasma sulphate,
c) supplement with: L-methionine, L-cysteine, N-acetylcysteine, Reduced glutathione,
d) Increase intake of nutrient cofactors Zn, Cu, riboflavin, selenium, Mg, vitamin B6, B12, folic acid,
e) Consider inorganic sulphate (especially if high cysteine/sulphate ratio with low plasma sulphate),
f) Increase intake of molybdenum, if signs of insufficiency such as impaired sulphoxidation and/or sulphite toxicity.

GLUCURONIDATION is responsible for conjugation of Paracetamol, NSAIDS, Benzodiazepines, Sex hormones (especially estrogens).

If low glucuronidation (acetaminophen glucuronide recovery):

Suspect: Impaired glucuronidation detoxification/Difficult removal of toxins from the body

Possible causes:
a) Insufficient carbohydrate reserves (e.g. fasting or insulin insensitivity),
b) Possible free radical damage to mitochondria (ATP necessary for glucuronidation),
c) Iron deficiency,
d) Excess vitamin K,
e) Insufficient nutrient cofactors for glucuronidation,

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f) Hypothyroidism (delays maturation of conjugating enzyme),
g) Antibiotics chloramphenicol and novobiocin can interfere with the conjugating enzyme,
h) Genetic uniqueness (e.g. Gilbert's disease) Glucuronidation may be low if a shared pathway (e.g. sulfation) is taking on more compound than usual.

Consider the following actions:
a) Improve glucose utilization or insulin resistance, if relevant,
b) Supplement antioxidants if evidence of free radical damage,
c) Correct nutrient deficiencies,
d) Increase intake of nutrient cofactors,
e) L-glutamine, aspartic acid, niacin, vitamin B6, Fe, Mg,
f) Rule out hypothyroidism,
g) Support other Phase II pathways, especially sulfation and glycination, to reduce burden,
h) Increase intake of cruciferous vegetables (induces conjugating enzyme).

Phase I/Phase II Ratios

IF Low, Then
Toxin exposures tend to show higher accrual of tissue levels because clearance is limited by hepatic oxidation.

If High, Then
Risk of carcinogenesis is increased due to higher rates of accumulation of toxic intermediates.

Improve Phase I to Phase II levels accordingly, by upregulating or down regulating phase I or phase II levels.

Tests ordered: LIVER

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The Liver detoxification profile evaluates the ability of an individual to process caffeine, aspirin, and paracetamol by assessing certain metabolites in saliva and urine specimens measuring phases of liver detoxification.

Phase 1, also known as caffeine clearance, bioactivation occurs via oxidation, reduction and hydrolysis, predominantly by the cytochrome p450 enzyme family.

Phase	Causes	Treatment Considerations
High Phase 1 Increased exposure to toxins and production of free radicals.	• Exposure to P450 enzyme inducers - Drugs e.g. barbiturates, HRT, steroids, sulfonamides - Environmental pollutants e.g. exhaust fumes, paint fumes, dioxin & • pesticides - Gut-derived toxins from gut dysbiosis or leaky gut - Others: alcohol, cruciferous vegetables, charcoal-broiled foods, tobacco.	• Assess and remove exposure to any P450 inducing substances • Reduce exposure to environmental toxins • Assess and treat gut dysbiosis and/or intestinal permeability (IP) • Antioxidant supplementation- e.g. acai, selenium, vitamin C & E, zinc • Botanical liver support- e.g. ellagic acid, green tea, silymarin, grapefruit juice
Low Phase 1 Reduced activity of Cytochrome P450 from exposure to: Drugs - benzodiazepines, antihistamines, ketoconazole, H2blockers		• Green tea (catechins) • Turmeric • B group vitamins • Bioflavonoids • Amino acids - Glutathione, glycine, glutamine, cysteine

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Phase	Causes	Treatment Considerations
Low Glucoronidation Reduced acetaminophen glucuronide recovery.	• Increased exposure to drugs and xenobiotics requiring glucuronidation - e.g. steroid hormones, oxazepam, carbamates, phenols, aniline • Genetic enzyme defect - e.g. Gilbert's disease • Medications: - Antibiotics e.g. chloramphenicol, novobiocin Nutritional & Metabolic Causes: • Decreased energy production or reduced energy from dietary sources • Hypothyroidism • Insulin resistance • Vitamin K excess • Upregulation of other Phase II pathways.	• Discontinue medications which may affect glucuronidation • Reduce xenobiotic exposure • High quality protein source • Support mitochondrial function to help improve energy production - e.g. antioxidants, coQ10, magnesium - Aspartic acid, iron, L-glutamine, magnesium, niacin, vitamin B6 • Increase cruciferous vegetable intake e.g. watercress • Reduce enterohepatic recirculation of toxins e.g. calcium D-glucurate • Support other Phase II pathways.
Low Glycination • Reduced salicyluric acid recovery.	• Increased levels of drugs & xenobiotics requiring glycination - e.g. aspirin, benzoate, phenylacetic acid, aliphatic amines • Liver disease • Genetic enzyme defect.	• L-glycine supplementation • Supplement glycination cofactors- cysteine, magnesium, vitamin B5 • Reduce benzoate exposure - e.g. sodium benzoate preservative • Reduce xenobiotic exposure • Reduce salicylate exposure from cosmetics, drugs & diet.

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Phase	Causes	Treatment Considerations
Low Glutathionation: Reduced acetaminophen mercapturate recovery.	• Increased exposure to drugs & xenobiotics requiring glutathionation e.g. acetaminophen, penicillin, tetracycline, styrene, toxic metals, bacterial toxins • Increased reactive oxygen species • Impairment of other Phase II pathways • Genetic enzyme defects • Enhanced bile production (increases mercapturate elimination via the bile).	• Assess and remove exposure to xenobiotics • Glutathione and glutathioine precursor and cofactor supplementation - glutathione, L-glycine, L-glutamine, L-methionine, N-acetylcysteine, • B12, zinc • Botanical liver support supplementation e.g. silymarin, artichoke, watercress • Antioxidant supplementation e.g. vitamin C & E, zinc, selenium, acai • Support other Phase II pathways.
Low Sulfation: Reduced acetaminophen sulfate recovery.	• Increased exposure to drugs & xenobiotics requiring sulfation - e.g. minoxidil, terpines, amines, phenols • Increased reactive oxygen species • Impaired sulfoxidase activity • Molybdenum or vitamin B6 excess (can inhibit sulfation) • Liver disease • Genetic enzyme defects • Upregulation of other Phase II pathways.	• Assess and remove exposure to xenobiotics • Sulfate precursors and cofactor supplementation - glutathione, L-methionine, N-acetylcysteine, zinc • Supplement inorganic sulfate (MSM) and/or molybdenum if inadequate cysteine to sulfate conversion (sulfoxidase activity) is suspected • Reduce dietary phenols and amines.

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High Phase 2 pathways

Use adequate cofactor and nutrient support. This will ensure that these molecules do not become depleted and liver detoxification does not become impaired.

Phase 1: Sulphation

Demonstrates the relationship between Phase I and the sulphation pathway and demonstrates whether the biochemicalload from Phase I is too high.

Phase 1: Glycination

These two ratios reflect the relationship between Phase I and these two conjugation pathways and will demonstrate whether the biochemical load from Phase I is high or low.

Phase 1: Glucuronide

These two ratios reflect the relationship between Phase I and these two conjugation pathways and will demonstrate whether the biochemical load from Phase I is high or low.