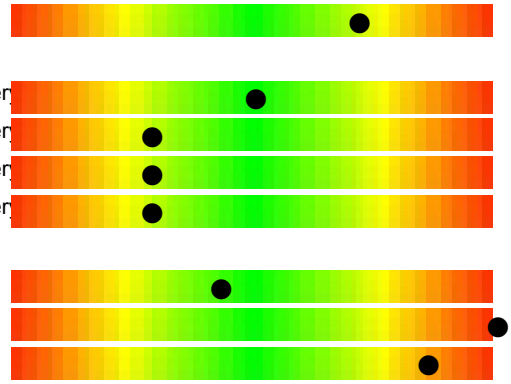
NutriPATH
Integrative Pathology Services
P: 1300 688 522
E: info@nutripath.com.au
A: PO Box 442 Ashburton VIC 3142

TEST PATIENT
Sample Test Name
Sex : F
Date Collected : 00-00-0000
111 TEST ROAD TEST SUBURB
LAB ID: 00000000 UR#:0000000

TEST PHYSICIAN
DR JOHN DOE
111 CLINIC STREET
CLINIC SUBURB VIC 3000

INTEGRATIVE MEDICINE

URINE, SPOT	Result	Range	Units
DETOXIFICATION CAPACITY PROFILE			
PHASE I (OXIDATION)			
Caffeine Clearance	**1.4**	0.5 - 1.6	ml/min/Kg
PHASE II (CONJUGATION)			
Glutathionation	**8.2**	5.6 - 11.4	% Recovery
Glycination	**33.7**	30.0 - 53.0	% Recovery
Sulphation	**18.5**	16.0 - 36.0	% Recovery
Glucuronidation	**32.1**	27.0 - 56.0	% Recovery
RATIOS			
PHASE I / PHASE II - Sulphation	**7.6**	3.5 - 13.0	RATIO
PHASE I / PHASE II - Glycination	***4.2* *H**	1.3 - 3.5	RATIO
PHASE I / PHASE II - Glucuronide	***4.4* *H**	1.9 - 4.2	RATIO



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. 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,IMPEI

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

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LIVER DETOX INTERPRETATION GUIDE

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
	<u>Nutritional & Metabolic Causes:</u>
	• 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.