"Integrated Cancer Care and the role of Mitochondria"

Dr Rajendra Sharma

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THE *CANCER ACT 1939*

- I am not an oncologist.
- I do not treat cancer. I work with/support people *with* cancer.

- **Complementary or natural approaches**
  in combination with
  conventional cancer treatments,
  can be referred to as
  Integrated or Integrative care

The cause of cancer

DNA v Mitochondrial Theory

Aka

Somatic Mutation Theory (SMT)
v
Metabolic Theory

…but mitochondria contain their own DNA
and
cancer cells exhibit both genetic and metabolic abnormalities.

So there is clearly overlap with both theories.

Somatic Mutation Theory

(Correct term for DNA theory)

- A somatic cell

(any cell of a living organism other than the reproductive cells) ***accumulates*** **genetic/DNA damage** when it

a). **multiplies** or

b). is damaged by **outside influences.**

c). DNA/Genetic changes are ***inherited*** and triggered by age and damage. This can be

Familial

or

- Possibly from human **early evolutionary days** having been of ancestral benefit (wound-healing and tissue regeneration)

Origin of DNA theory Boveri 1914. SMT; Fardon 1953

DNA gone wrong

At some point the DNA … departs from the cells healthy regulatory systems and "embarks on its own agenda".
(Rapid replication)

Paraphrased **Paul Davies** *Theoretical physicist; https://www.edge.org/response-detail/25380*

alternatively

- A cell loses *control* of correct replication
(Loss of replication cessation – **senescence**)

Cancer as a Metabolic Disease (Wiley 2012) by Thomas N. Seyfried, PhD

When DNA goes wrong

- Ignores apoptosis (natural cell death) instructions
- Loss of replication control
- Increases angiogenesis (the blood supply to itself),
- Develops Stem-like behaviour & metastasises (migrates) to other tissues
- Metabolizes (manufactures) growth factors
- ‘Tricks’ the immunity

Metabolic Theory

- A re-emerging area of interest surrounds mitochondria and their role in chronic disease.
- A theory suggesting cell metabolism goes wrong due to **failure of normal cell respiration** leading to DNA mutation.
- The key lethal flaw in cancer does not originate in the nucleus at all but in the **mitochondria.**

Mitochondrial actions - simplified

Acknowledgment & thanks to Gilian Crowther

- Making energy
- Recognition of nutritional sufficiency
- Synthesising metabolic components (inc. steroid hormones
- Promoting metabolic pathways (detoxification, cell structure, immune function, control of glucose levels)
- Cell to cell signalling and cell homeostasis
- Involved in apoptosis
- Recognition of intra-cellular infection and toxicity

Metabolic theory

- Cancer cells show defects in metabolism. Various changes of metabolic behaviour are known that can create

"Genome instability (DNA errors)."

Mitochondria theory

- Damaged mitochondria cause chronic inflammation and cellular respiratory insufficiency.

- Most cells die but others ‘thinking’ they are doing the best thing to survive, switch from Oxidative Phosphorylation to fermentation demanding an abundant supply of glucose or carbohydrates to make energy.

Mitochondria theory

Mitochondrial function is important in controlling

'stemness' (the ability to self-renew

and differentiate) and cell fate.

Semin Cancer Biol. 2017 Dec;47:18-28. Mitochondrial biology in cancer stem cells. Loureiro R et al

Empirical evidence…

- If you transplant a cancer cell's mutated nucleus into a normal cell (from which the nucleus has been removed - anucleated) cancer cells do not develop.

  (McKinnell 1969; Mintz 1975; Howell 1978; Harris 1988; Shay 1988; Li 2003; Hochedlinger 2004).

- But if you transplant a normal nucleus into an anucleated cancerous cell, the cell *can* form tumours.

  (Israel 1987; Israel 1988).

Mitochondria, if not the cause, may still be associated with cancer

- Fermentation of glucose in the presence of oxygen (aerobic glycolysis) is a symptom of cancer, not the cause

Origins : Otto Warburg (Warburg, On the origin of cancer cells. *Science. 1956 Feb 24; 123(3191):309-14.*

- Many tumours continue to utilize oxygen while also engaging in fermentation but this appears to be without any energy-generating capacity. Cancer cells reprogram their energy metabolism.

(Pedersen 2007; Hanahan 2011).

(One glucose molecule produces 36-38 ‘packets’ of energy called adenosine triphosphate (ATP). This occurs in the mitochondria through processes called glycolosis, the Krebs (Citric acid) cycle + a process called oxidative phosphorylation).

SMT probably not acting alone

- More than 10,000 (possibly 60,000) new, naturally occurring DNA damages arise, on average, per human cell, per day, due to endogenous cellular processes

So many chances yet comparatively few cancers.

- Many mutagens are not carcinogenic and many carcinogens are not mutagenic

Poor evidence of cause and effect.

"The SMT has not been rigorously tested, and several lines of evidence raise questions that are not addressed by this theory" (Soto 2011).

Metabolic Theory
Mitochondria not acting alone

- Cells with dysfunctional mitochondria do not necessarily become cancerous.
- Cells with poor respiration generally simply undergo apoptosis.

What Makes Mitochondria Dysfunctional

- 1. Toxic effects
- 2. Infection
- 3. Chronic Inflammation
- 4. DNA ‘adducts’ – stuff stuck on DNA
- 5. Mitochondrial adducts
- 6. Familial or predetermined genomic errors
- 7. Anti-cancer Immune Dysfunction
- 8. Nutritional deficiency

Useful Investigations

- 1. Mitochondrial function
- 2. Mitochondrial adducts
- 4. Anti-cancer Immune Function
- 5. Detoxification capability
- 6. DNA ‘adducts’ – stuff stuck on DNA
- 7. Infection
- 8. Nutritional deficiencies

Lab 4 More Mitopro

MVZ Labor Bavariahaus — Lab4more

Dr. Musterarst
Musterweg 21
80000 Hainstadt

Name Ms Musterlady
Date of Birth 16.06.1959
Adresse Probenweg 77
89999 Musterstadt

Patient No. 197762
Lab. Number 8232505
Requested 19.05.2016

Height 0 cm **Weight** 0 kg **Body Mass Index** 0,0 **Reported** 03.06.2016

Diagnose:
CFS

RESULTS SUMMARY

1. The percentage of T cells with a adequate ATP supply (mitochondrial function) is lowered. This would appear to indicate the existence of a functional disturbance in the mitochondrial respiration cycle (mitochondriopathy) as well as a possible immune function deficiency (T cell function disorder).
2. No functional deficiencies of vitamin B12 detectible, methylmalonic acid within range.

Further therapeutic control recommended.

THERAPEUTIC RECOMMENDATIONS:
Avoidance of mitochondrial stressors i.e medications (antibiotics, anti viral Agents), toxins, physical and mental stress, micronutrient deficiencies and/or support of the mitochondrial metabolism with Q10, vitamin B12, vitamin B2, B3, carnitine and lipoic acid.

Kind Regards
Dipl.Biol. B. Knaberschuh /Dipl.Biol.W.Mayor

BASIC CHECK UPS

Creatinine (U)	0,38	g/l	0,3 - 2,2	

OXIDATIVE STRESS

Mitochondrial Profile
ATP-Check Mitochondrial Function

ATP Energy Level	[illegible]	% T-Zellen	> 85	

ESSENTIAL NUTRIENTS

Pyruvate (Plasma)	[illegible]	mg/dl	0,34 - 0,72	
Lactate (Plasma)	6,00	mg/dl	< 22,0	
Lactate/Pyruvate-Quotient	4,35	Quotient	< 25	
Methylmalonic acid (U/Crea)	1,37	mg/g KREA	< 2,0	

Explanation

PYRUVATE
Pyruvate is the end product of glycolysis. It is normally further metabolised in the liver to Acetyl- CoA, but only if sufficient NADH is available, or it is used in gluconeogenesis and/or converted anaerobically (extramitochondrial) to lactate or amino acids (alanine). In the brain, muscles, intestinal mucosa, adrenal cortex and erythrocytes pyruvate is not further utilized. It is converted to lactate and the liver takes over the further metabolism of lactate to fatty acids or for gluconeogenesis. In cases of primary (metabolic disorders) or secondary mitochondrial defects (mitochondriopathy) deficiencies in pyruvate utilisation occur leading to increases in pyruvate and lactate. In severe circulatory, pulmonary, liver, renal, muscular and systemic disorders increases inn lactate/pyruvate occur, whereby excessive lactate is produced and the LP quotient increases.

LACTATE
Lactate is the end product of anaerobic glycolysis. It is primarily produced in the muscles, but is also produced in the erythrocytes, adrenal medulla, brain and intestine.Cardiac muscles can cover 60% to their energy requirements with lactate. The liver, brain and to a lesser degreee the kidneys user lactate for gluconeogenesis. In cases of increased production or reduced clearance increased lactate levels are found in blood. A hyper lactataemia with a moderately increased lactate (2-5 mmol/l) without acidosis is found after physical activity, carbohydrate infusion, high

Validated by: P.D. Dr. med. F.-W. Tiller, Dr. Rudolf Raßhofer

Acumen ATP

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Acumen 151756 Patient [redacted]

Date of birth [redacted]

Reported 26/06/2015 Doctor Dr Rajendra Sharma

ATP (adenosine triphosphate), studies on neutrophils

ATP is hydrolysed to ADP and phosphate as the major energy source in muscle and other tissues. It is regenerated by oxidative phosphorylation of ADP in the mitochondria. When aerobic metabolism provides insufficient energy, extra ATP is generated during the anaerobic breakdown of glucose to lactic acid. ATP reactions require magnesium. ATP to ADP conversion can be blocked by environmental contaminants as can the translocator [TL] in the mitochondrial membrane. [TL] efficiency is also sensitive to pH and other metabolite-pace changes. [TL] deficits may demand excessive ADP to AMP conversion (not re-converted to ATP or through to ATP). Defects in Sig-ATP, ADP → ATP conversion and enzyme or [TL] blocking can all result in **chronic fatigue – a factor in any disease where biochemical energy availability is reduced.**

ATP whole cells:

With excess Mg added (Standard method of measuring ATP)	**1.48**	nmol/10^7 cells	1.6 – 2.9
Endogenous Mg only (Measured ATP result is lowered during intracellular magnesium deficiency)	**0.84**	nmol/10^7 cells	0.9 – 2.7
Ratio ATP^{Mg}/ATP^{Mg}	**0.57**		> 0.65

ADP to ATP conversion efficiency (whole cells):

ATP^{Mg} (from above)	**1.48**	nmol/10^7 cells (1*)	1.6 – 2.9
ATP^{Mg} (inhibitor present)	**0.45**	nmol/10^7 cells (2*)	< 0.3
ATP^{Mg} (inhibitor removed)	**1.02**	nmol/10^7 cells (3*)	> 1.4
ADP to ATP efficiency [(3*- 2*)/(1*- 2*)] x 100 =	**51.4 %**		> 60
Blocking of active sites (2*/1*) x 100 =	**30.4 %**		up to 14

ADP-ATP TRANSLOCATOR [TL] (mitochondria, not whole cells):

	ATP (nmol/10^7 cells)	Ref. range	change %	ref. range
Start	**260**	280 – 700		
[TL] 'raw'	**335**	410 – 950	**28.8**	over 35% (Increase)
[TL] 'in'	**222**	140 – 330	**14.6**	55 to 75% (Decrease)

Comments

Low whole-cell ATP

30% blocking of active sites leading to:

Low mt-ATP and poor provision of 'new' mt-ATP

Quite-marked blocking of translocator function.

Low ATP-related magnesium

Poor ADP to ATP re-conversion

Dr John McLaren-Howard DSc FACN - Directors - Mrs Mirhane McLaren-Howard

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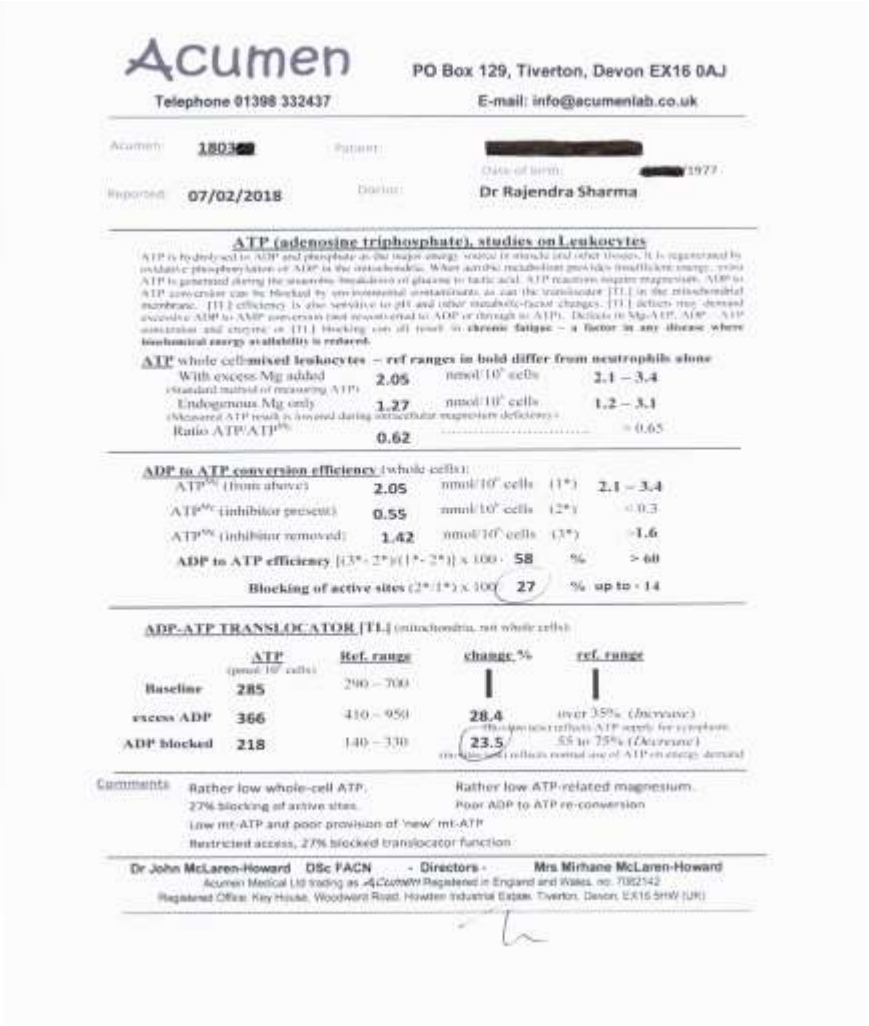
Active sites



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E-mail: info@acumenlab.co.uk

Acumen: 1803[redacted] Patient: [redacted]

Date of birth: [redacted]1977

Reported: 07/02/2018 Doctor: Dr Rajendra Sharma

ATP (adenosine triphosphate), studies on Leukocytes

ATP is hydrolysed to ADP and phosphate as the major energy source in muscle and other tissues. It is regenerated by oxidative phosphorylation of ADP in the mitochondria. When aerobic metabolism provides insufficient energy, extra ATP is generated during the anaerobic breakdown of glucose to lactic acid. ATP reactions require magnesium. ADP to ATP conversion can be blocked by environmental contaminants as can the translocator [TL] in the mitochondrial membrane. [TL] efficiency is also sensitive to pH and other metabolic-factor changes. [TL] deficits may demand excessive ADP to AMP conversion (not reconverted to ADP or through to ATP). Deficits in Mg-ATP, ADP, AMP conversion and enzyme or [TL] blocking can all result in **chronic fatigue – a factor in any disease where biochemical energy availability is reduced.**

ATP whole cell-**mixed leukocytes – ref ranges in bold differ from neutrophils alone**

With excess Mg added (Standard method of measuring ATP)	2.05	nmol/10^7 cells	**2.1 – 3.4**
Endogenous Mg only (Measured ATP result is lowered during extracellular magnesium deficiency)	1.27	nmol/10^7 cells	**1.2 – 3.1**
Ratio ATP/ATP^{My}	0.62	.....	**< 0.65**

ADP to ATP conversion efficiency (whole cells):

ATP^{My} (from above)	2.05	nmol/10^7 cells	(1*)	**2.1 – 3.4**
ATP^{My} (inhibitor present)	0.55	nmol/10^7 cells	(2*)	**< 0.3**
ATP^{My} (inhibitor removed)	1.42	nmol/10^7 cells	(3*)	**> 1.6**
ADP to ATP efficiency [(3*-2*)/(1*-2*)] x 100 =	58	%		**> 60**
Blocking of active sites (2*/1*) x 100 =	27	%		**up to < 14**

ADP-ATP TRANSLOCATOR [TL] (mitochondria, not whole cells):

	ATP (pmol/10^7 cells)	Ref. range	change %	ref. range
Baseline	285	290 – 700		
excess ADP	366	410 – 950	28.4	over 35% (*Increase*)
ADP blocked	218	140 – 330	23.5	55 to 75% (*Decrease*)

Comments: Rather low whole-cell ATP. Rather low ATP-related magnesium.
27% blocking of active sites. Poor ADP to ATP re-conversion
Low mt-ATP and poor provision of 'new' mt-ATP
Restricted access, 27% blocked translocator function

TLP blockage, Lipid Ox, Metals

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Acumen: 1803[redacted] Patient: [redacted]

Date of birth: [redacted]977

Reported: 07/02/2018 Doctor: Dr Rajendra Sharma

Mitochondrial membrane TL protein studies [TL]

[TL] scavenges ADP from cytoplasm and returns ATP from re-conversion plus new ATP from oxidative phosphorylation. [TL] can be blocked by xenobiotics and/or partial detoxification products. The site is also pH sensitive and can be affected by local or general acidosis including organic acid accumulation from anaerobic metabolism. [TL] efficiency is also compromised by increases in intracellular calcium. **The test sequence** examines the whole cell mitochondria using phase-contrast, dark field and polarising microscopy. We search for [TL] adducts using a micro-plate array of fluorescence probes. Positive indications are explored at the molecular level. Detailed fluorescence microscopy at extreme magnifications employs de-convolution and Helicon FocusPro™ software.

Initial examination:	Patient's result ↓	Fluorescent indicators or micro-electrode studies:			
Mitochondia numbers of:	Normal	Outer membrane pH	< 6.8 (low)	6.8 – 7.4 normal	> 7.4 (high)
Mitochondrial clumping	High #2	Patient's result: →		7.1	
Mt-membrane structure	Normal	Outer membrane Ca^{2+}	< 200 normal	200 – 300 borderline	> 300 nmol/l raised
Mt-DNA fluorescence	Trace	Patient's result: →		225	
Mt-membrane binding: →	Proteins	Esterases	Lipids	Diplein	Aldehydes
Patient's results: →	Normal	Normal	Normal	ND	High #1

(ND = not detected)

Chemical on TL sites	Amount	Comment
Malondialdehyde #1	High	#1 Lipid oxidation products.
Tissue breakdown product	Moderate	Peptide approx 2.2kDa.
		#2 Cu, Ni & Ca -actin binding.

Mt membrane K: Normal Mt membrane Mg: Low-norm Mt membrane Zn: Low-norm

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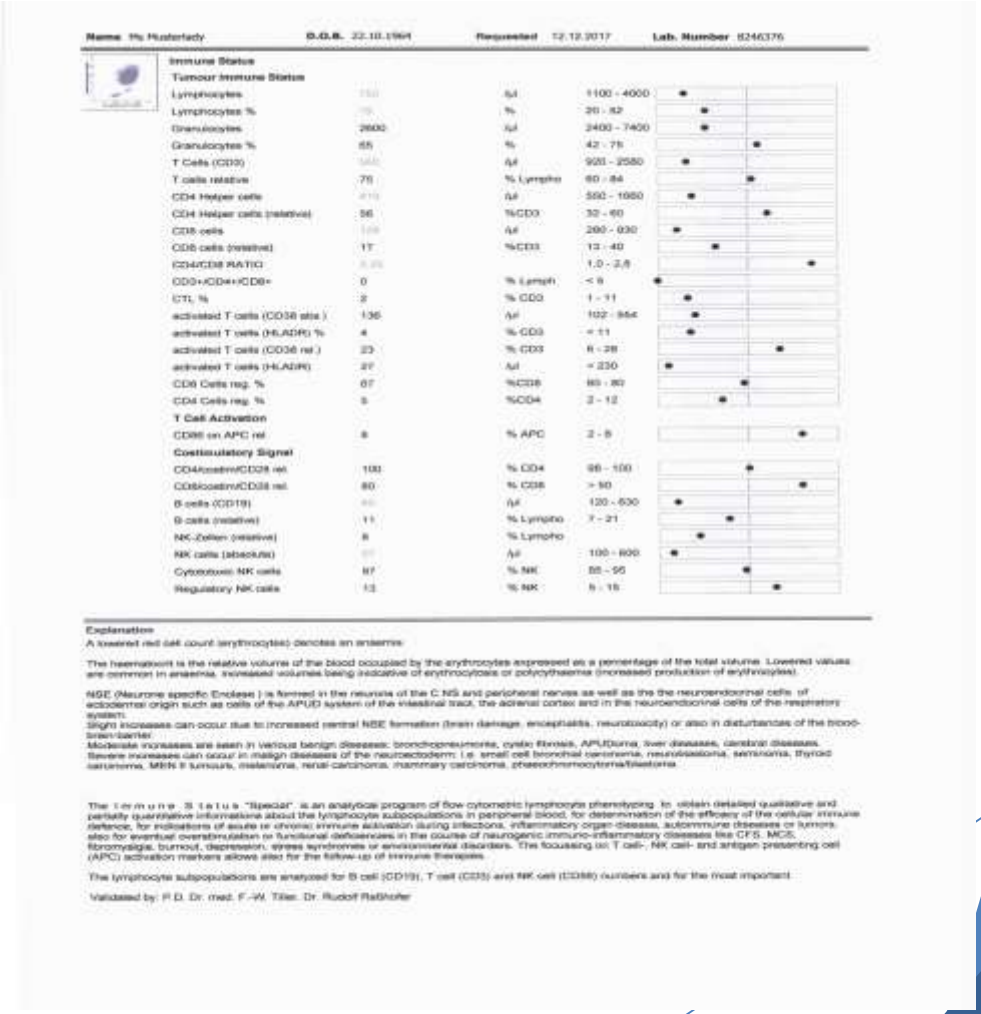
Tumour immunity



Name: Ms Hysterlady D.O.B. 22.10.1964 Requested 12.12.2017 Lab. Number 8246376

Immune Status

Tumour Immune Status

Lymphocytes	[illegible]	/µl	1100 - 4000	
Lymphocytes %	[illegible]	%	20 - 52	
Granulocytes	2600	/µl	2400 - 7400	
Granulocytes %	65	%	42 - 75	
T Cells (CD3)	[illegible]	/µl	920 - 2580	
T cells relative	76	% Lympho	60 - 84	
CD4 Helper cells	[illegible]	/µl	550 - 1950	
CD4 Helper cells (relative)	56	%CD3	32 - 60	
CD8 cells	[illegible]	/µl	260 - 930	
CD8 cells (relative)	17	%CD3	13 - 40	
CD4/CD8 RATIO	[illegible]		1,0 - 2,8	
CD3+/CD4+/CD8+	0	% Lymph	< 9	
CTL %	2	% CD3	1 - 11	
activated T cells (CD38 abs.)	136	/µl	102 - 954	
activated T cells (HLADR) %	4	% CD3	< 11	
activated T cells (CD38 rel.)	23	% CD3	8 - 28	
activated T cells (HLADR)	27	/µl	< 230	
CD8 Cells reg. %	67	%CD8	60 - 80	
CD4 Cells reg. %	5	%CD4	2 - 12	
T Cell Activation				
CD86 on APC rel	8	% APC	2 - 8	
Costimulatory Signal				
CD4/costim/CD28 rel.	100	% CD4	95 - 100	
CD8/costim/CD28 rel.	80	% CD8	> 50	
B cells (CD19)	[illegible]	/µl	120 - 630	
B cells (relative)	11	% Lympho	7 - 21	
NK-Zellen (relative)	8	% Lympho		
NK cells (absolute)	[illegible]	/µl	100 - 600	
Cytotoxic NK cells	87	% NK	85 - 95	
Regulatory NK cells	13	% NK	5 - 15	

Explanation

A lowered red cell count (erythrocytes) denotes an anaemia.

The haematocrit is the relative volume of the blood occupied by the erythrocytes expressed as a percentage of the total volume. Lowered values are common in anaemia, increased volumes being indicative of erythrocytosis or polycythaemia (increased production of erythrocytes).

NSE (Neurone specific Enolase) is formed in the neurons of the C.NS and peripheral nerves as well as the the neuroendocrinal cells of ectodermal origin such as cells of the APUD system of the intestinal tract, the adrenal cortex and in the neuroendocrinal cells of the respirator system.
Slight increases can occur due to increased central NSE formation (brain damage, encephalitis, neurotoxicity) or also in disturbances of the blood-brain-barrier.
Moderate increases are seen in various benign diseases: bronchopneumonia, cystic fibrosis, APUDoma, liver diseases, cerebral diseases. Severe increases can occur in malign diseases of the neuroectoderm: i.e. small cell bronchial carcinoma, neuroblastoma, seminoma, thyroid carcinoma, MEN II tumours, melanoma, renal carcinoma, mammary carcinoma, phaeochromocytoma/ileotoma.

The Immune Status "Special", is an analytical program of flow cytometric lymphocyte phenotyping. It obtain detailed qualitative and partially quantitative informations about the lymphocyte subpopulations in peripheral blood, for determination of the efficacy of the cellular immune defence, for indications of acute or chronic immune activation during infections, inflammatory organ diseases, autoimmune diseases or tumors, also for eventual overstimulation or functional deficiencies in the course of neurogenic immuno-inflammatory diseases like CFS, MCS, fibromyalgia, burnout, depression, stress syndromes or environmental disorders. The focussing on T cell-, NK cell- and antigen presenting cell (APC) activation markers allows also for the follow-up of immune therapies.

The lymphocyte subpopulations are analyzed for B cell (CD19), T cell (CD3) and NK cell (CD56) numbers and for the most important

Validated by: P.D. Dr. med. F.-W. Tiller, Dr. Rudolf Raßhofer

Hep detox

DOCTOR'S DATA

LAB #: U150402-2295-1
PATIENT: [redacted]
ID: [redacted]-D-00017
SEX: Female
AGE: 23

CLIENT #: 38598
DOCTOR:
Regenerus Laboratories Ltd
Aero 14, Redhill Aerodrome, Kings Mill Lane
Redhill, Surrey, RH1 5YP UNITED KINGDOM

Hepatic Detox Profile; Urine

TOXIC EXPOSURE MARKERS

	RESULT per creatinine		REFERENCE INTERVAL		PERCENTILE 2.5th 16th 50th 84th 97.5th
D-Glucaric Acid (Phase I)	320	nM/mg	40 - 400		
Mercapturic Acids (Phase II)	130	µM/mM	40 - 95		

URINE CREATININE

	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	83	35 - 225	

INFORMATION

The human body attempts to eliminate xenobiotics (foreign organic chemicals) through a concerted effort of enzymatic "functionalization" (phase I) and conjugation (phase II). Functionalization involves chemical modification of the xenobiotic by the cytochrome P-450 or the "mixed function oxidase" enzyme systems. Once functionalized, the altered xenobiotic can then be conjugated and excreted. Urinary D-glucaric acid, a hepatic byproduct of enzymatic response to chemical toxins (phase I), is a reliable indicator of exposure to xenobiotics. Mercapturic acids are direct, excretory end products of the functionalized xenobiotics that have been conjugated with glutathione prior to excretion. Together, the urinary levels of these metabolites provide valuable information about exposure to xenobiotics, liver disease, and quantitative assessment of the status of hepatic phase II detoxification.

D-GLUCARIC ACID MARGINALLY ELEVATED: The level of D-glucaric acid, a marker of exposure to hepatotoxic substances, is marginally elevated for age and gender in this patient's urine sample. This suggests possible mild exposure to xenobiotics with normal detoxification (check mercapturic acids level/phase II activity). Elevated urinary excretion of D-glucaric acid is an indication of induction of cytochrome P-450 enzymes (phase I) in the liver that may be the result of exposure to any of over 200 different xenobiotics (e.g. pesticides, herbicides, fungicides, petrochemicals, drugs, alcohol, toluene, xylene, formaldehyde, styrenes, ibuprofen etc.). Occupational and environmental exposure to toxic compounds causes induction of the glucuronic acid enzyme pathway and production of D-glucaric acid, thus D-glucaric acid excretion is considered an indirect by-product of detoxification reactions. Elevated levels of urinary D-glucaric acid have also been correlated with viral hepatitis and jaundice, and have also been found in patients receiving antirheumatic drugs independent of disease activity. With marginally elevated levels of D-glucaric acid, there may be an increased need for antioxidant protection because toxins that are processed through phase I generate free radicals that require quenching or neutralization. It is important to consider that phase I detoxification tends to become less active with aging.

MERCAPTURIC ACIDS ELEVATED: The levels of mercapturic acids in this patient's urine sample are abnormally high for age and gender, and consistent with exposure to xenobiotics and enhanced detoxification via glutathione conjugation (phase II). Mercapturic acids are final excretory products of detoxification and include a variety of functionalized xenobiotics that have been conjugated with cysteine, or glutathione. Ideally, urinary levels of mercapturic acids should be increased with exposure to xenobiotics and enhanced phase I detoxification; mercapturic acid levels will gradually return to basal levels commensurate with successful hepatic detoxification and removal of the patient from the source of exposure. Detoxification should be supported with supplemental vitamins C, E, and lipoic acid, selenium, Mg, K, rGSH, and sulfur containing amino acids. It should be noted that falsely elevated levels of mercapturic acids can occur in patients with cystinuria, or with the use of mono- and dithiol chelators (D- penicillamine, DMSA and DMPS), and some 'thio-capto' type medications (e.g. thioridazine, captodiamine).

SPECIMEN DATA

Comments:

Date Collected: 03/30/2015
Date Received: 04/02/2015
Date Completed: 04/09/2015

Methodology:
D-Glucaric: HPLC
Mercapturic: Enzymatic

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Detox Phase I genes

DNA

Acumen PO Box 129, Tiverton, Devon EX16 0AJ

Telephone 01398 332 457 E-mail: acumenlab@hotmail.co.uk

Acumen: 091821 Patient: Another Real Patient

Date of birth:

Reported: Doctor: Sample Report

DNA ADDUCTS (genomic DNA from leucocytes)

Genomic DNA from leucocytes is analysed by gas-liquid chromatography for the group-presence of organic chemicals and by atomic emission for the presence of toxic metals. We identify specific adducts using fluorescent-marker probes, Raman spectrophotometry, fluorimetry and gel electrophoresis. We try to selectively precipitate any abnormal proteins for further investigation by polarisation microscopy and immuno-assay procedures. Wherever possible, we identify the location of the adducted chemical on the DNA and say if it associated with a specific gene or control factor.

Total DNA (from 1ml whole blood) = 54 ug (reference 30 – 60)

Adduct found	ng/ml blood	Gene (if identified)
p-phenylene diamine	7	DNA-ligase gene (q13)
Methyl resorcinol	4	Mn-SODase gene (q6)

Comments: **DNA-associated Zinc = 31 ng/ml (reference 21 - 74)**

PPD is a dye-precursor mainly used in hair colourants. Methyl resorcinol is also used in colourants, shampoos and hair conditioners. DNA-ligase expression is essential for normal DNA-repair.

Please note:

The table above includes *all* adducts found. The following is a list of common adducts (included in the above table if found in this sample).

Chemical or group: Amines-general, Diamino compounds, Diazo compounds, Nitrosamines, Pentachlorophenol and other fungicide-type chlorinated phenols, Bactericide-type chlorinated phenols, para-Dichlorobenzene, Other halogenated benzene compounds, Vinyl halide, Acrylamide, Malondialdehyde, Other aldehydes, Aflatoxin/ochratoxins, Lindane (& other BHCs), Toxaphene, Other organochlorine compounds, Tetrachlorvinphos, Other organophosphates, Organophosphate metabolites, Phthalates, Abnormal proteins, Abnormal peptides.

Metals: Aluminium, Antimony, Arsenic, Barium, Cadmium, Chromium, Cobalt, Copper, Lead, Manganese, Mercury, Nickel, Platinum, Rhodium, Silver, Strontium, Thallium, Tin, Titanium.

Dr John McLaren-Howard DSc FACN - Directors - Mrs Mirhane McLaren-Howard

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Registered Office: Kay House, Woodward Road, Howden Industrial Estate, Tiverton, Devon, EX16 8HW (UK)

Detecting the presence of cancer

Others

- 7. Infection
- 8. Nutritional deficiencies

- But…….


HERMAN
by Jim Unger
DID YOU SEE THE DOCTOR?
HE GAVE ME THREE MONTHS.
HOW ARE YOU GONNA PAY £487 IN THREE MONTHS?

Anecdotal Observations

- Abnormal ATP production – 75%
- Mitochondrial adducts – 80%
- Mito associated Lipid oxidation - 50 %
- DNA adducts – 90+%
- Detoxification issues – close to 100%
- Subfunctional Tumour Immunity – 75%

Therapeutic options

1. Establishing and removing the cause of cancer
2. The use of orthodox techniques and medical care
3. Nutritional assessment and dietetic therapy
4. Complementary Medicine - supporting cancer therapy
5. Assessment and activation of the detoxification systems
6. Assessment and activation of anti-cancer immunity
7. Psychological and healing techniques
8. Tests and Investigations

Diet

- Other than being proven to reduce the occurrence rate of cancer, anything but aggressive diet and life style alteration appears not have a profound effect on the progression of cancer.

  http://www.wcrf-uk.org/cancer_prevention/recommendations.php?gclid=CPHYu9WVjq0CFYQLfAodnWyeoQ

- Simple restrictions of are not proven to be curative

Nevertheless – don't provide a banquette for cancer.

Avoiding these foods reduces rates of cancer so logic would suggest it is advisable to avoid them

- dairy produce – please review the work of Prof Jane Plant http://www.cancersupportinternational.com/janeplant.com/index.asp
- refined sugar & natural sugars above that found in 2 pieces of fruit a day - https://www.oncologynutrition.org/erfc/healthy-nutrition-now/sugar-and-cancer/
- fried foods,
- alcohol
- and limit caffeine to 1 'hit' daily

- American Cancer Society Guidelines on Nutrition and Physical Activity for Cancer Prevention: Reducing the Risk of Cancer with Healthy Food Choices and Physical Activity Tim Byers et al., http://onlinelibrary.wiley.com/doi/10.3322/canjclin.52.2.92/pdf

Get Expert Advise To Start With

- Mitochondrial savvy nutritionists
- The few Integrated Doctors working in this area
- Dr Sarah Myhill's web site and book

"Diagnosis and Treatment of Chronic Fatigue Syndrome and Myalgic Encephalitis" Hammersmith Press

- Cell Symbiosis Therapy ® by Dr. Heinrich Kremer

https://www.heal24.com/therapies/cell-symbiosis-therapy--by-dr-heinrich-kremer-2038/#micro-and-macro-nutrients-and-vital-substances-lack

Mitochondrial Therapy

- Pre-emptive: Can I reduce my risk of cancer through mitochondrial therapy?
- Therapeutic: Mitochondrial normalisation might be of therapeutic benefit

Mitochondrial Therapy
According to the principles of
Dr. med.
Heinrich Kremer

- Binding and neutralizing of free radicals
- Provision of building blocks of defence cells and therefore reinforcement of the immune system
- Provision of building blocks of collagen
- Improvement of oxygen transfer in the respiratory chain (improved blood circulation)
- Improvement of iron utilization
- Reduction of allergic diseases and histamine release
- Improvement of cellular respiration by enhancement of mitochondrial function Regulation of proliferation
- Regulation of cell detoxification

Bowel flora restoration

- Gut bacteria influence mitochondrial dynamics
- Gut microbiota manage mitochondrial related ROS production, pro-inflammatory signals and metabolic limits

Detoxification

- Cellular
- Tissue and organ
- Liver up-regulation of Phase 1 & 2
- Bowel function – Phase 3

Removal of infection

- Viral – EBV, HS, Lyme co-Infections
- Bacterial - Borrelia, Mycoplasma, Chlamydia
- Fungal/yeast ?

Energy Medicine

- Mitochondria react with/to photons

More research needed on

- LASER
- Far/infrared

Multi-nutrient Stimulation

- Drs Myhill, Booth, McLaren-Howard

Chronic fatigue syndrome and mitochondrial dysfunction Int J Clin Exp Med (2009) 2, 1-16

Mitochondrial dysfunction and the pathophysiology of ME/CFSInt J Clin Exp Med 2012;5(3):208-220

- Dr Heinrich Kremer

The Silent Revolution in Cancer and AIDS Medicine

Supplements Increasing Mitochondrial Activity

- Magnesium
- Zinc
- L-carnitine,
- lipoic acid,
- B complex
- D-Ribose
- Coenzyme Q10
- PQQ (*pyrroloquinoline quinone*
- Others

Herbal Extracts – effects on Mitochondria

- Medicinal plants contain a range of phytochemicals both beneficial and detrimental to mitochondrial function
- Positive effects – direct, anti-oxidant and unknown
- Curcumin
- Quercetin
- Resveratrol
- Salvestrols
- Others

Cell Symbiosis Therapy

- Pro Basan complete – Prebiotic + Probiotic + Nutrient

https://drive.google.com/file/d/0B8ToBiT4VsbiS0pwMTRxTkJZN28/view

- Pro Curmin Complete - Phytochemicals

https://drive.google.com/file/d/0B8ToBiT4VsbiMWZvZVZLZ0FMcHc/view

- Pro Dial Vit 44 – Mitochondrial supportive multi-nutrient

https://drive.google.com/file/d/0B8ToBiT4VsbiR3N3Y05FQk5KUmc/view

- Pro Sirtusan- antioxidant, anti-inflammatory, mito.biogenic

https://drive.google.com/file/d/0B8ToBiT4VsbiY1BnbkllndmgwcDg/view

CST Protokoll Injekt N
Intravenous infusions – adjunct to oral therapy

Amino acids

Taurine L-Carnitine L-Lysine Acetylcysteine L-Arginine 200 mg L-Carnosine 200 mg

Vitamins

Ascorbic Acid (Vitamin C) Nicotinamide (Vitamin B3) Dexpanthenol (Vitamin B5) Pyridoxin Hydrochloride (Vitamin B6) Riboflavin-5-phosphate-mononatrium (Vitamin B2) 10 mg

Hydroxocobalamin (Vitamin B12)

Minerals

Zinc Gluconate Calcium Chloride Magnesium Chloride Potassium Chloride Na-Selenite Folic Acid

Major Antioxidant

Glutathione (test levels in cancer treatment)

Dr Rajendra Sharma

MB BCh BAO LRCP&S (Ire) MFHom
GMC No: 2919995

Dr Sharma has no affiliation with the manufacturers or distributors of any products or tests mentioned in this presentation.

Dr Sharma's Award Winning Book

'Live Longer Live Younger'

http://www.amazon.co.uk/Live-Longer-Younger-Rajendra-Sharma/dp/1780285108

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