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Dictated on 24 March 2009

Dr John Woodward  
The Red Horse Surgery  
The Old School  
Market Square  
Kineton  
Warwickshire  
CV35 0LP

Our ref: sm/nw

Dear Dr Woodward,

**Re: John Hoyte, Harefield House, High Street, Fenny Compton, Southam, Warwickshire**  
**CV47 2YG DOB: 17.10.55**

John's health continues to improve – indeed he now feels he is almost back to how he felt in 1989 which, of course, is excellent news. This improvement is evident in the enclosed test results because he decided to retest his mitochondrial function and antioxidant status again as well as checking DNA adducts and doing a fat biopsy for levels of toxins still remaining. Well, there is no bad news. As you can see from the enclosed results his ATP profiles have completely normalised, cell free DNA shows just a mild increase in cell degradation, niacin status is now well within the normal range and both SODase and GSH-PX studies are essentially normal, the exception being slightly low levels of glutathione at 1.67mmol/l (1.7 – 2.6) which means John is still using up glutathione in the detoxification process. So he needs to keep supplementing with glutathione 250mg daily to assist here.

In his last DNA adducts test there was evidence of high levels of malondialdehyde – this was symptomatic of poor antioxidant status - it also showed very high levels of antimony. As you can see, there are no longer any adducts found on John's DNA – well done John!

As for the fat biopsy, there are still VOCs and pesticides present, however the levels are really very small and I am sure that before long these too will have disappeared.

So it is simply a case of more of the same for John which should continue to bring about further clinical improvements following these very encouraging biochemical results.

Yours sincerely,



Dr Sarah Myhill

Encs to GP: Test results.

Encs to patient: Test results, Cc.

Acumen: **090457**Patient: **John Hoyte**

Date of birth: 17/10/1955

Reported: **13/03/2009**Doctor: **Dr Sarah Myhill****Screen for PESTICIDES AND RELATED SUBSTANCES in FAT CELLS****Note: these tests are performed for medical purposes only. We cannot enter into legal disputes.**

PLOA is the Provisional Limit of Acceptability based on reference data from 57 controls.

<u>NAME or GROUP</u>	<u>= mg/kg *</u>	<u>PLOA (Control range)</u>	<u>COMMENTS</u>
<b>Organochlorine group</b> Inc: aldrin, dieldrin, toxaphene, DDT, DDE, DDD, HCB	<b>0.35</b>	<b>1.0</b> (0.1 – 1.1)	<b>Background</b>
<b>Lindane (not included in above)</b> and other isomers of BHC	<b>0.08</b>	<b>0.05</b> ND – 0.05	<b>Above 'average'</b>
<b>Chlorinated phenols group</b> Penta- and tri-chlorophenols with other chlorophenols if found	<b>0.07</b>	<b>0.2</b> ND - 0.28	<b>Background</b>
<b>Chlorinated benzenes</b> Para-dichlorobenzene is the most widely used	<b>0.47</b>	<b>0.5</b> Trace – 0.58	<b>Background</b>
<b>Polychlorinated byphenyls (PCBs) Banned chemicals</b> Very persistent in the body	<b>0.16</b>	<b>0.3</b> 0.02 – 0.41	<b>Background</b>
<b>Polybrominated byphenyls (PBBs) Flame retardants/plastics</b>	<b>0.12</b>	<b>0.05</b> 0.01 – 0.08	<b>Raised level</b>
<b>Carbamates group</b> Inc: bendiocarb & carbaryl		<b>0.25</b> Trace – 0.3	<b>Not detected</b>
<b>Naphthols group</b> Inc: alpha-naphthol (carbaryl metabolite)		<b>0.03</b> ND – 0.04	<b>Not detected</b>
<b>Organophosphate group</b>		<b>0.1</b> ND – 0.19	<b>Not detected</b>
<b>Docofol (Kelthane)</b>			<b>Just detectable</b>

\*These should be viewed as semi-quantitative, especially where chemicals have been grouped together.  
ND = not detected. Mg/kg is 1000 times the ug/l units used in expressing serum levels.

**Other comments:** Lindane is above 'average' levels and PBBs are raised. In all other respects, these are normal background-exposure findings.

Dr John McLaren-Howard

Mrs Mirhane McLaren-Howard

For and on behalf of Acumen



# Acumen

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Acumen: **090457**

Patient: **John Hoyte**

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## VOLATILE ORGANIC COMPOUNDS in Fat cells

Tests performed for medical purposes only. We cannot enter into legal disputes.

← Whole blood → ← Fat cells →

PA = population average. DL = detection limit. Empty cell = not detected.

<u>Chemical</u>	<u>ng/ml</u>	<u>PA</u>	<u>DL</u>	<u>Chemical</u>	<u>mg/kg</u>	<u>PA</u>	<u>DL*</u>
Benzene		0.8	0.2	Benzene	0.3	0.4	0.1
Toluene		0.5	0.2	Toluene	0.18	0.15	0.1
Xylene		1.3	0.2	Xylene		0.2	0.1
Styrene		<0.2	0.2	Styrene		<0.1	0.1
Trimethylbenzene(s)		0.6	0.2	Trimethylbenzene(s)		0.1	0.1
Dichloromethane		<0.2	0.2	Dichloromethane		<0.1	0.1
Chloroform		0.5	0.2	Chloroform	0.15	0.12	0.05
Trichloroethane		0.2	0.2	Trichloroethane		0.05	0.05
Tetrachloroethylene		<0.2	0.2	Tetrachloroethylene		<0.1	0.05
Dichlorobenzene(s)		0.6	0.2	Dichlorobenzene(s)	0.47	0.5	0.1
Carbon tetrachlor		0.2	0.2	Carbon tetrachlor		0.1	0.05
<u>Aliphatic:</u>							
N-Pentane		<1.2	1.2	N-Pentane		<0.4	0.4
Cyclopentane		<0.5	0.5	Cyclopentane		0.1	0.05
2-Methylpentane		2.2	0.4	2-Methylpentane	0.15	0.3	0.1
3-Methylpentane		5.7	0.5	3-Methylpentane	0.5	1.1	0.1
N-Hexane		10.3	0.4	N-Hexane	0.7	1.6	0.2
N-Heptane		<0.9	0.9	N-Heptane		<0.2	0.2

Empty cell indicates below the detection limit.

\*Average but will depend on sample size.

(Fat cell results are calculated on the basis of the measured total fat content of sample received)

Methods: Blood and fat cell samples. The head-space volatiles displaced during programmed heating are separated and analysed by GLC (similar to US EPA method 624). Blood only: a second GLC run is made on a methanol extract (similar to US EPA method 524.2). Fat cell samples only: a pre-column methyl-ester-derivatised aliquot is analysed for fat content (C12-24) The results are used in calculating the concentration of each of the volatiles in the fat cells. The instrumentation is a Technochem 277 twin-channel GLC with Yokagawa LR4110 4-channel combined integrator, recorder and printer. The capillary-column carrier gas is helium.

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## 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. Whenever 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) = 51 ug (reference 30 – 60)**

<u>Adduct found</u>	<u>ng/ml blood</u>	<u>Gene (if identified)</u>
None found		

Comments: **DNA-associated Zinc = 27 ng/ml (reference 21 - 74)**

**Normal findings.**

### 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/mitotoxins, 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.

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### 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. 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] defects may demand excessive ADP to AMP conversion (not re-converted to ADP or through to ATP). Defects in Mg-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)	<b>1.89</b>	nmol/10 <sup>6</sup> cells	1.6 - 2.9
Endogenous Mg only (Measured ATP result is lowered during intracellular magnesium deficiency)	<b>1.27</b>	nmol/10 <sup>6</sup> cells	0.9 - 2.7
Ratio ATP/ATP <sup>Mg</sup>	<b>0.67</b>	.....	> 0.65

#### ADP to ATP conversion efficiency (whole cells):

ATP <sup>Mg</sup> (from above)	<b>1.89</b>	nmol/10 <sup>6</sup> cells (1*)	1.6 - 2.9
ATP <sup>Mg</sup> (inhibitor present)	<b>0.13</b>	nmol/10 <sup>6</sup> cells (2*)	< 0.3
ATP <sup>Mg</sup> (inhibitor removed)	<b>1.37</b>	nmol/10 <sup>6</sup> cells (3*)	> 1.4
ADP to ATP efficiency [(3* - 2*)/(1* - 2*)] x 100 =	<b>70.5</b>	%	> 60
Blocking of active sites (2*/1*) x 100 =	<b>6.9</b>	%	up to 14

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

	<u>ATP</u> (pmol/10 <sup>6</sup> cells)	<u>Ref. range</u>	<u>change %</u>	<u>ref. range</u>
Start	<b>396</b>	290 - 700		
[TL] 'out'	<b>572</b>	410 - 950	<b>44.4</b>	over 35% ( <i>Increase</i> ) (in-vitro test) reflects ATP supply for cytoplasm
[TL] 'in'	<b>187</b>	140 - 330	<b>52.8</b>	55 to 75% ( <i>Decrease</i> ) (in-vitro test) reflects normal use of ATP on energy demand

#### Comments

Normal whole-cell ATP  
 No significant blocking of active sites.  
 Normal mitochondrial function.  
 Excellent progress.

ATP-related Mg seems to be normal.  
 ADT-ATP is normal but slightly slow.

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## Cell-free DNA in blood plasma

**Background.** Most of the cell-free DNA present in blood plasma is associated with cell degradation. Very low levels are present in healthy people and increases are associated with serious illnesses such as malignancy, stroke, auto-immune diseases, severe infections and *Chronic Fatigue Syndrome*.

### Patient's result:

### Reference range

Cell-free DNA      **10.6 ug DNA per litre plasma**      up to 9.5

### Comments:

Mild increase      = 9.6 to 12.4

Some increase     = 12.5 to 14.9

Definite increase = 15.0 to 20.0

Highly significant = over 20.0

**Method summary\*** Plasma is incubated with EDTA, a detergent and a proteinase prior to precipitation of the proteins. DNA is then precipitated with alcohol and re-dissolved in a Tris-acetate-EDTA Buffer. The DNA is measured in a Pharmacia GeneQuant<sup>TM</sup> or Jenway Genova analyser using a micro-cuvette.

\*Schmidt B, Weickmann S, Witt C, Fleischhacker M. Improved Method for Isolating Cell-Free DNA. *Clin Chem* 2005; 51(8); 1561-2

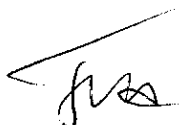
**Cell-free DNA in chronic fatigue syndrome (CFS)** In initial studies on 87 CFS patients, positive results were found in 93% of those with a disease duration of four months to five years (n = 75). In those with a disease duration of five to 14 years (n = 12), 75% had positive results.

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## **SUPEROXIDE DISMUTASE and GLUTATHIONE PEROXIDASE**

A functional test looks at the in-vitro efficiency of the patient's red cell superoxide dismutase (SOD) when their neutrophil superoxide production is maximally stimulated. The activity of the individual forms of SOD are explored. General cell protection from damage by superoxide is provided by intracellular zinc:copper-SOD (Zn/Cu-SOD). Mitochondria are protected by manganese-dependent SOD (Mn-SOD). Extracellular SOD (EC-SOD – another Zn/Cu SODase) protects the nitric oxide pathways that relax vascular smooth muscle.

For each form of SODase, genetic variations are known, mutations can occur during excessive oxidative stress on DNA and polymorphisms may be present. DNA adducts can chemically block these genes.

Glutathione peroxidase (GSH-PX) activity is measured in red blood cells. It is a selenium-dependent enzyme and selenium deficiency is the commonest cause of poor enzyme activity. As poor glutathione (GSH) availability is easily overlooked as an additional reason for poor GSH-PX activity, we also measure total GSH in red cells.

### **Blood test results:**

<b>Test</b>	<b>Result</b>	<b>Units</b>	<b>Reference range</b>
<b>Functional test</b>	<b>43</b>	<b>%</b>	<b>Over 40 (mostly 41 – 47)</b>
<b>Zn/Cu-SOD</b>	<b>315</b>	<b>Enzyme activity (u)</b>	<b>240 – 410</b>
<b>Mn-SOD</b>	<b>148</b>	<b>Enzyme activity (u)</b>	<b>125 – 208</b>
<b>EC-SOD</b>	<b>26</b>	<b>Enzyme activity (u)</b>	<b>28 – 70</b>

### **Gene studies:**

<b>Sod form</b>	<b>Gene(s)</b>	<b>Comments</b>
<b>Zn/Cu-SOD chromosome 21</b>	<b>Normal</b>	<b>Normal enzyme activity</b>
<b>Mn-SOD chromosome 6</b>	<b>Normal</b>	<b>Normal enzyme activity</b>
<b>EC-SOD chromosome 4</b>	<b>Normal</b>	<b>Rather low enzyme activity</b>

### **Glutathione peroxidase (GSH-PX)**

<b>Red cell</b>	<b>Glutathione peroxidase (GSH-PX)</b>	<b>72</b>	<b>U/gHb</b>	<b>67 – 90</b>
<b>Red cell</b>	<b>Glutathione (GSH)</b>	<b>1.67</b>	<b>mmol/l</b>	<b>1.7 – 2.6</b>

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## NIACIN STATUS (vitamin B3)

Red cell nicotinamide adenine dinucleotide (NAD) is a good indicator of B3 status.

**Red cell nicotinamide adenine dinucleotide = 19.2 ug/ml      14.0 – 30.0**

Interpretation of result:	Reference range	(14.0 – 30.0)
	Mild B3 deficiency	(12.5 – 13.9)
	Moderate B3 deficiency	(11.0 – 12.4)
	Fairly marked B3 deficiency	(10.0 – 10.9)
	Marked B3 deficiency	(8.5 – 9.9)
	Severe B3 deficiency	(less than 8.5)

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### References:

- 1) Fu CS, Swendseid ME, Jacob RA, McKee RW. Biochemical markers for assessment of niacin status in young men: levels of erythrocyte coenzymes and plasma tryptophan. *J Nutr* 1989; 1949 – 1955.
- 2) Critical review. Assessment of niacin status in humans. *Nutrition Reviews* 1990; 48: 318 – 320

### Note:

The amino acid tryptophan is a precursor of niacin. However, protein synthesis has a higher metabolic priority than the conversion of tryptophan to niacin coenzyme and adequate niacin levels cannot always be obtained from tryptophan.

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