

PE1463/HH

John Charville Letter of 24 January 2014

I have the Right of Rebuttal in respect of the British Thyroid Foundation's submission to the Scottish Parliamentary Petitions Committee guaranteed by Article 169 of the Treaty on the Functioning of the European Union and the 2nd & 3rd paragraphs of Article 4.3 from the Treaty on European Union. The UK cannot depend upon domestic rules as to the division of Power & Responsibility between constitutional authorities. [[Paragraphs 33 & 34 of European Court of Justice Joined Cases C-46/93 and C-48/93 ~ Brasserie du Pêcheur SA and Federal Republic of Germany and between The Queen and Secretary of State for Transport ex parte: Factortame Ltd and Others](#)]

I have been watching the Scottish Thyroid Petition PE 01463 with some interest. I know that the Petition was badly framed from the Outset, being defined as: -

"Effective thyroid and adrenal testing, diagnosis and treatment"

When the Petitioners were really concerned about the failure of the UK medical profession to recognise Free Triiodothyronine (FT3) Deficiency at the cellular level and dysfunction of the Adrenal Glands.

Based upon the title of Petition PE 01463 The Scottish Parliamentary Petition Committee would have no option but to uphold the UK & Worldwide medical profession's definition of hypothyroidism, which is perhaps best expressed on the [Wikipedia Website](#) as follows: -

Hypothyroidism is an endocrine disorder in which the thyroid gland produces inadequate quantities of the thyroid hormones thyroxine (T4) and triiodothyronine (T3).

I repeat that the Scottish Parliamentary Petition Committee would under normal circumstances have no option but to uphold the definition of hypothyroidism applied by the UK medical profession, where I am sure that most sensible people will agree the only treatment for Primary Hypothyroidism is L-T4 Levothyroxine monotherapy.

However, if The Scottish Parliamentary Petition Committee were to apply that definition and use it as a tool to dispense with the Petitioners' application the Petition Committee would be in breach of: -

1. [The 2nd & 3rd paragraphs of Article 4.3 of the Treaty on European Union](#)

The Member States shall take any appropriate measure, general or particular, to ensure fulfilment of the obligations arising out of the Treaties or resulting from the acts of the institutions of the Union.

The Member States shall facilitate the achievement of the Union's tasks and refrain from any measure which could jeopardise the attainment of the Union's objectives.

2. And specifically [Article 168 of the Treaty on the Functioning of the European Union](#). (Also see Articles 20 & 169).

1. A high level of human health protection shall be ensured in the definition and implementation of all Union policies and activities.

Union action, which shall complement national policies, shall be directed towards improving public health, preventing physical and mental illness and diseases, and obviating sources of danger to physical and mental health. Such action shall cover the fight against the major health scourges, by promoting research into their causes, their transmission and their prevention, as well as health information and education, and monitoring, early warning of and combating serious cross-border threats to health.

The Union shall complement the Member States' action in reducing drugs-related health damage, including information and prevention.

The Title of the Petition may be incorrect, but the body of the Petition clearly asks that FT3 Deficiency be recognised and treated. And this is a matter that does not fall under the definition of Hypothyroidism as defined by the UK medical Profession. It is a matter of failure by the bodily deiodinase system to convert available inactive pro-hormone Free Thyroxine (FT4) to the active form Free Triiodothyronine (FT3) and has nothing to do with Hypothyroidism as defined by the UK medical profession.

The thyroid gland can be making more than adequate quantities of the inactive pro-hormone Thyroxine (T4) but if the bodily system cannot convert it to the active real hormone Triiodothyronine (T3) essential to health then the only remedy is to prescribe L-T3 Liothyronine Therapy to supplement bodily T3 levels.

The FREE parts of T3 & T4 are those parts which are available for use by the body and not made unavailable by being bound to proteins. Hence FT3 & FT4.

I would now address the British Thyroid Foundation response to the Scottish Parliamentary Petitions Committee. Dr Mark Vanderpump is a Trustee of the BTF. Dr Mark Vanderpump is also a party to the [European Thyroid Association's 2012 Guidelines: The Use Of L-T4 + L-T3 In The Treatment Of Hypothyroidism](#), which the BTF Response invoked.

At the top of the left hand column of page 2 from the ETA Guidelines Hypothyroidism is defined as follows: -

"Hypothyroidism is a condition characterized by the clinical and biochemical manifestations of thyroid hormone deficiency in the target tissues of thyroid hormone."

The above definition of hypothyroidism clearly includes FT3 Deficiency at the cellular level, and relates to the level of Thyroid Hormones available for use in the body as opposed to solely dysfunction of the hypothalamus, pituitary and thyroid glands, as defined by the UK & Worldwide medical profession.

The interesting aspect of the ETA 2012 Guidelines is that its stated intention was to examine why patients claim they still require L-T3 therapy despite their TSH & FT4 being Normalised. Yet the study clearly excluded from consideration any other matter than Primary Hypothyroidism.

Despite such unreasonable exclusion of relevant matters and information, The ETA Guidelines provide clear evidence of FT3 Deficiency: -

It is clearly stated that Primary Hypothyroidism is mostly caused by Autoimmune Disease (Hashimoto's Thyroiditis). It is a fact that the autoimmune attack on thyroid gland tissues will produce Cytokines (interleukin 6) and C-Reactive Proteins, which will induce metabolic slowdown (Also See Inter-Alia: "Low T3 Syndrome", "Non-Thyroidal Illness Syndrome" & "Euthyroid Sickness Syndrome"): -

Metabolic Slowdown involves activation of bodily Deiodinase Enzyme D3 and conversion of available FT4 to REVERSE T3 (rT3), which blocks the cellular FT3 receptor sites. Whilst the action of bodily Deiodinase Enzyme D1, which should convert FT4 to FT3 in the body is inhibited.

However, in the brain which is separated from the body by a Blood-Brain-Barrier, Deiodinase Enzyme D2, which is 1,000 times more efficient than Deiodinase D1, locally converts FT4 to FT3 so the brain is protected from Metabolic Slowdown.

Furthermore the Pituitary which has little or no Deiodinase Enzymes D1 or D3, but is supplied with Deiodinase Enzyme D2 locally converts FT4 to FT3 which the hypothalamus sees as bodily FT3 and therefore reduces Thyrotropin (TSH) levels suppressing Normal production of T4 (80%) & T3 (20%) by the Thyroid Gland.

The ETA Guidelines clearly identify that in healthy subjects their FT4:FT3 ratio is approximately 3:1. (This is an important number).

It also identifies that FT4:FT3 ratios of between 4.0-->5.5 are identified in the study cohorts. Indeed it clearly states that as the administration of L-T4 Levothyroxine increases, the FT4:FT3 ratio also increases.

Any intelligent person will recognise that this is exactly the response one expects when Deiodinase Enzyme D3 is active and Deiodinase Enzyme D1 is inhibited.

Indeed when Metabolic Slowdown accompanies Primary Hypothyroidism, because the effect of pituitary Deiodinase Enzyme D2 is to lower TSH levels, it is obvious that Metabolic Slowdown will mask any Primary Hypothyroidism and make the patient's TSH & FT4 blood assays appear Normal.

The Medical Profession and undoubtedly the British Thyroid Foundation will claim that FT3 Deficiency is RARE. Yet as demonstrated by the ETA 2012 Guidelines it almost always accompanies Primary Hypothyroidism.

FT3 Deficiency is also the Primary cause of "Low T3 Syndrome", "Non-Thyroidal Illness Syndrome" and "Euthyroid Sick Syndrome": It is not RARE and probably occurs with more frequency than Primary Hypothyroidism itself.

Indeed FT3 Deficiency has multiple causes which can be individually present or co-existing.

For example: -

- Low Cortisol levels;
- Vitamin and Mineral Deficiencies;
- Low Co-Enzymes (e.g. CoQ10)
- Low Transport Proteins;
- Any Inflammatory condition which produces Cytokines (Interleukins 1 & 6) or C-Reactive Proteins;
- Diseases such as inter-alia: Cancer, Diabetes, Asthma, Chronic Obstructive Pulmonary Disease, Chronic Kidney Disease: It must be stressed that the causality is BI-DIRECTIONAL, which I shall address in a moment; in terms of Dilated Cardiomyopathy; and of course
- The failure by the Thyroid Gland to supply sufficient Thyroxine (T4) for conversion to Triiodothyronine (T3).

DILATED CARDIOMYOPATHY.

This is particularly relevant as it is mentioned in Petition PE 01463: -

Vitamin D3 (Now considered to be a hormone because the body can manufacture it) is necessary for the conversion of FT4 to FT3 and the usage of FT3 at the cellular level, thus Vitamin D3 deficiency may induce FT3 Deficiency.

I now refer to [3,5,3'-Triiodothyronine deprivation affects phenotype and intracellular \[Ca²⁺\] of human cardiomyocytes in culture](#) which clearly identifies that Low T3 causes inhibition of the calcium handling properties of cardiomyocytes and mutation of cardiomyocytes initially causing low diastolic & systolic blood pressure. Here FT3 Deficiency is an obvious causal factor not the result of Heart Failure as the medical profession will propose.

I also cite [Relation between free triiodothyronine/free thyroxine ratio, echocardiographic parameters and mortality in dilated cardiomyopathy](#) which clearly identifies the blood serum concentrations of FT3 at which Dilated Cardiomyopathy is induced. I must stress that this research also identifies that normal healthy people have an FT4:FT3 ratio of approximately 3:1 (As above this is a very important figure).

The research also asserts that Dilated Cardiomyopathy may be identified by an FT4:FT3 ratio of approximately 4.4:1 (which presumes that the patient's Thyroid Gland is not dysfunctional and is capable of producing the necessary levels of FT4 to produce such an FT4:FT3 ratio) accompanied by FT3 blood serum concentrations of 2.9-->5.1 pmol/l with a mean value of 4.0 pmol/l.

This research is useful because it also sets-aside the unsupported assertions by the UK medical profession that T3 treatment will cause heart problems: -

"Treatment of low T3 levels in congestive heart failure patients had been investigated in some short-term clinical studies and in acute conditions, such as after cardiopulmonary bypass surgery or cardiogenic shock [23,24]. Although most of these studies showed beneficial effects, data about effects of long-term thyroid hormone supplementation is not yet available. The relation between heart failure and thyroid hormone changes resembles a vicious circle where impairment in heart function leads to lower T3 levels and lower T3 in turn causes decreased contractility and diastolic dysfunction.

Therefore, in advanced congestive heart failure, thyroid hormone administration may help to break this vicious circle and improve the patients' clinical status."

In fact as a sufferer of FT3 Deficiency, who has been forced to self-treat, with L-T3 Liothyronine because of the flawed "Evidence-Based Medicine" thrust on the General Public by the UK medical profession, I can report that my own symptoms of heart failure are gradually diminishing. I am now able to walk more than 200 metres without becoming physically exhausted, mentally fatigued and suffering severe pain in my legs.

Given that Scotland has a much higher incidence of heart failure than the rest of the UK I submit that Petition PE 01463 has very clearly identified a health scourge that the Scottish Parliamentary Petitions Committee cannot simply disregard because the Petition was wrongly framed in its Title.

The British Thyroid Foundation is very obviously not impartial as proven by both its submission to the Scottish Parliamentary Petitions Committee and as demonstrated by the [European Thyroid Association's 2012 Guidelines: The Use Of L-T4 + L-T3 In The Treatment Of Hypothyroidism](#), to which it was a major contributor in the person of Dr Mark Vanderpump.

Hypothyroidism is only one possible cause of FT3 Deficiency which can readily be identified by an FT4:FT3 ratio higher than 3:1, a 'Low Free Triiodothyronine (FT3)' serum concentration and 'Reverse T3 (rT3)', often accompanied by Cytokines, C-Reactive Proteins, and Thyroid Peroxidase Antibodies (TPOAb).

The above is only a small part of my challenge to the Flawed Science relied upon by the UK medical profession, which will be identified more fully in my forthcoming Application for Judicial Review by the Administrative Court at the Royal Courts of Justice, Strand, London.

Yours Sincerely

John Charville