



## TPA's REBUTTAL to PRESCQIPP – BULLETIN 121- LIOTHYRONINE

<https://www.prescqipp.info/drop-list/send/52-drop-list/2047-bulletin-117-drop-list>

### INTRODUCTION:

My name is Sheila Turner. I am founder/chair of the Charity Thyroid Patient Advocacy (TPA). Registered No. 1138608. I am writing to formally request that the thyroid hormone liothyronine (L-T3) be removed from 'The PrescQIPP DROP-List 2015 (Bulletin 121)'.

The length of this response is necessary because the majority of information compiled by PrescQIPP CIC about triiodothyronine (T3) and liothyronine (L-T3) is fundamentally flawed and contains substantial errors of both a scientific and a general nature that need correcting.

The use of the acronym 'DROP' is misleading and gives the impression that there is an official instruction that liothyronine should be removed from the prescribing list.

Although PrescQIPP CIC has cited 4 references in an attempt to back up its statements concerning liothyronine (L-T3), only one link, (number 7) works. That links to the original 2011 Royal College of Physicians' policy statement on the Diagnosis and Management of Primary Hypothyroidism. That policy statement has not been valid since November 2015. Links No.8 and No.9 do not work and link No.10 is also invalid as it relates to Armour Thyroid, a branded natural desiccated porcine thyroid extract (NDT) and not to liothyronine. Therefore there is no evidence to support any of the statements.

In November 2015, the RCP asked whether the British Thyroid Association (BTA) would take ownership of their 2011 statement as "*their communications department were expressing concern regarding the volume of calls regarding this issue*". It is interesting to note that the BTA, in their July 2015 Summer Newsletter[91] admitted to receiving recurring criticism from the public that they were out of step with thyroidology in the rest of the world. Regarding the reason why the BTA has, by their own admission, received so much criticism, TPA takes the view that the RCP et al., original 2011 policy statement, of which they were a co-author, did not bear close scrutiny.

Before PrescQIPP starts to provide "... *explanations as to why L-T3 has been included in the DROP-List*" or attempts to explain "... *in what circumstances prescribing might be reasonable*", or suggests "... *prescribing alternatives to liothyronine (L-T3)*", it is imperative that PrescQIPP CIC understands precisely how the thyroid system functions and what thyroid hormones do.[1]

Despite studies bringing into question the safety and effectiveness of L-T4 monotherapy for a large minority of patients suffering continuing symptoms of hypothyroidism,[2-7] and confirmed harm,[8,9] the British Thyroid Association (BTA) encourages L-T4 as the only preferable approach for all who have been diagnosed with hypothyroidism. This is an incorrect analysis. There is mounting evidence which suggests that conventional [allopathic] endocrinologists, drug companies and the BTA profit financially from their mutual support thus depriving some thyroid patients of good health and well-being. This suspicion will require further investigation should the BTA (a) refuse to acknowledge those studies, (b) refuse to acknowledge evidence showing T3 replacement is required for those unable to regain optimal health on L-T4-monotherapy,[10-84] and (c) refuse to acknowledge research produced by others experienced in the field of endocrinology. Doctors are also showing signs of dissatisfaction with L-T4 monotherapy for all sufferers.[85-88]

The above statement is reinforced by the BTA's standard method of enforcing the practice of L-T4 monotherapy among doctors: dictatorship ignoring scientific argument and debate. Such suspicion is further evidenced by close examination of the relationship of the 7 original co-authors of the now redundant 2011 RCP policy statement.[89] These authors seem to collude to the detriment of patients receiving optimal treatment.[90]

The BTA's view that L-T4 is the 'treatment of choice' for all sufferers of the symptoms of hypothyroidism brought to mind the article: '*The Tomato Effect: Rejection of highly efficacious therapies*' (JAMA 1984).[92] "*Pharmaceutical companies influence medical decisions in a way that encourages the use of newer patentable therapies at the expense of older (but perhaps equally effective and less expensive) therapies begins on the first day of medical school and lasts through to retirement... it starts slowly and insidiously, like an addiction, and can end up influencing the very nature of medical decision-making and practice. . . attempts to influence the judgment of doctors by commercial interests serving the medical industrial complex are nothing if not thorough*". An explanation of how '*The Tomato Effect*' could influence the treatment of hypothyroidism is given in a Lancet editorial.[93]

In "*SIGN 50: A Guideline Developer's Handbook*",[94] Dr. P. Abernethy, states (a)...the case of Hunter v. Hanley establishes the customary standard of care for evaluating medical negligence and (b)... the case of Bolitho v. City and Hackney H.A. found that if the customary standard of care is not logical, the judge is entitled to determine that the defendant's expert's opinion was not reasonable or responsible.[95]

The evidence gathered by TPA indicates that the BTA's continued endorsement of L-T4-monotherapy for all with continuing symptoms of hypothyroidism is without basis, and can be harmful to hundreds of thousands of sufferers in the UK.[96]

It should be noted here that in the RCP 2011 policy statement [89] it was stated: "*Overwhelming evidence supports the use of thyroxine (T4 or tetra-iodothyronine) alone in the treatment of hypothyroidism, with this usually being prescribed as levothyroxine*". TPA requested that the President of the RCP cite references to such "overwhelming evidence". The RCP did not respond. Invoking Freedom of Information (FOI) a further request was made which also failed to attract a response. The 'Ask for Evidence' website, run in association with 'Sense About Science' was contacted requesting evidence on the safety and efficacy of L-T4 as a treatment for hypothyroidism. This was forwarded to the RCP who eventually responded stating "*The RCPs guidance is based on the opinion of an expert panel which was temporarily formed for this purpose. The evidence they used to form their individual opinions has not been collated and therefore the RCP cannot provide any evidence list*". This stance places the RCP in danger of accusations of possible medical negligence and/or negligent misstatement.

Studies have demonstrated the positive effect of T3 therapy, including psychiatric researchers, pointing out that T3 is generally well-tolerated.[97] Moreover, psychiatrists report that dosages of T3 higher than replacement dosages augment the depression-relieving effects of antidepressant.[4,98-101] Case reports and open systematic clinical trials also showed improvement in euthyroid fibromyalgia patients treated with T3.[102-105]

Another notable exception about T3 that the BTA have neglected to consider in the past is that some critically ill cardiac patients can also benefit from T3 therapy. Studies show that T3 improves these patients' heart function in a variety of ways. T3 decreases the severity and incidence of their cardiac abnormalities, and increases their survival rate. The BTA might not recommend T3 for these heart patients, but some cardiologists and cardiac surgeons do. Rather than the BTA denouncing T3 therapy, they would better serve their patients' welfare by reading the relevant studies and then rectifying their judgment of T3 to reflect its safety and the potential benefits for selected patients.[106-117]

Considering the effects of T3 on the heart, a large study of the health status of patients using T4-only was conducted in the UK. Compared to matched control patients, hypothyroid patients on "adequate dosages of T4 had a higher reported incidence of four diseases: depression, hypertension, diabetes, and heart disease".[118] Hypothyroid patients on inadequate T4 replacement (where TSH levels were elevated) also had a higher incidence of strokes.[119]

One interesting case is reported by Kaplan et al in 1981 of a patient who needed 500 mcgs of L-T3 daily to be free of hypothyroid symptoms. The patients' metabolism was normal and she had no tissue over-stimulation.[120] Another study demonstrated T3-induced recovery from fibromyalgia by a hypothyroid patient resistant to L-T4 and also to desiccated thyroid.[121] As with Kaplan's patient, many others have remained healthy for years on these supraphysiological dosages. None experienced adverse health affects, and none showed evidence of tissue over-stimulation upon serum and urine

biochemical testing, electrocardiography, or bone densitometry. Most of these patients who recovered with T3 therapy had previously failed to benefit from the use of T4-only replacement.

About 13% of patients in a large UK study prescribed L-T4-monotherapy continued to suffer symptoms that might be related to their L-T4 therapy.[122] Another study showed around 300,000 UK citizens do not benefit from L-T4-monotherapy.[119] Numerous assumptions are attached to medical practice guidelines concerning the ultimate use of thyroid hormones, particularly the active hormone T3.[124-131] These assumptions have led to unacceptable standards of care. They contain vague, imprecise and misleading language that focuses on the thyroid gland only and disregards the functions of the hypothalamus, the pituitary, peripheral metabolism, cellular thyroid hormone reception, up-regulation of cellular respiratory cycle and the necessary chemical infrastructure for all of these to function. These assumptions have contributed to false conclusions about what constitutes proper diagnosis and treatment of hypothyroidism.[131]

Whilst TPA is not saying that these inadequate protocols are in any way dishonourable, they do not reflect well on the competence of the UK's endocrinology establishment. The protocols are overstated and cover more physiology than supported scientifically. [131]]

The BTA have also disregarded the results of a hypothyroid survey undertaken in 2006 by TPA, in which 1500 participants undergoing T4-monotherapy, were asked, “*Do you feel that you have fully regained your optimal state of health?*” **1176 (78.4%)** participants answered “**NO**”.[132]

TPA, having the experience that accords with the test protocol Challenge/De-challenge/Re-challenge (CDR)[133] collated a ‘Register of Counterexamples to T4 Monotherapy’.[134] This register recorded 2080 patients who continued to suffer symptoms on L-T4-monotherapy, yet found their symptoms were mitigated or disappeared when they started a T3 hormone therapy. This register shows a greater number of patients who did decidedly better on L-T3 monotherapy, T4/T3 combination therapy (synthetic or natural thyroid extract) than the number of participants (900) in the studies showing that T4/T3 combination therapy worked no better than L-T4 monotherapy. ***In our understanding of scientific rigour, that simple fact nullifies those L-T4-only studies.***

Although the BTA amended statement[135] is about the management of *Primary* hypothyroidism, restrictions go beyond this. There are tests for the symptoms of ‘*Primary*’ hypothyroidism, but the issue is not so much about diseases of the thyroid gland, as the total lack of attention to the other glands and functions that can adversely affect the impact of the thyroid gland at the cellular level.[136-160] The Differential Diagnosing Protocol (DDP) requires examination of *all* physical issues and potential causes of symptoms,[161-164] but little attention, in the past, has been paid to the importance of this protocol. However, the BTA amended statement does attempt to address some of these, and medical practitioners must take account of these if their patients continue to suffer symptoms on L-T4 monotherapy.

PrescQIPP CIC, by (a) failing to take account of readily available evidence and research; (b) putting a slanted interpretation on explanations given in the BTA amended statement; and (c) exhibiting an unquestioning reliance on that statement, has failed to present a balanced argument in Bulletin 121

TPA has, therefore, provided herewith, literature and research evidence not considered by PrescQIPP CIC that may help correct the general and scientific errors made in Bulletin 121.

1. Out of the 4 links provided in Bulletin 121 to references concerning liothyronine (L-T3), only one (7) works. Links (8) and (9) do not work. Link (10) is invalid, as it relates to Armour Thyroid, a branded natural desiccated porcine thyroid extract (NDT) and not liothyronine.
2. As stated in Bulletin 121, PrescQIPP CIC's document has not alluded to any circumstance where liothyronine might be appropriate as treatment.
3. There is **no** validated, published research showing L-T4 monotherapy is safe and effective for **all** patients suffering symptoms of hypothyroidism, therefore, the proposition that L-T4 works for all cannot, and must not be relied upon.
4. PrescQIPP CIC's statements have given no indication regarding what the savings would be to the NHS if liothyronine is dropped from prescription.

5. PrescQIPP CIC's claim that "*some of the products available are not licensed medicines in the UK*" refers to a branded natural thyroid extract (Armour Thyroid), and not to liothyronine [165]
6. The claims that "*there is no robust evidence to support the use of liothyronine in routine practice*", and that "*no circumstances can be found where prescribing liothyronine might be reasonable*" is misleading, false and as evidenced in this document, contradicted by medical research.[10-84] PrescQIPP CIC's bias towards the RCP, BTA, BTF, NHS UKMi and local CCG's stance shows a complete disregard of any scientific evidence from outside of these organisations and a complete disregard of liothyronine as a successful therapy. [215]
7. PrescQIPP CIC's claim that the 1.5 to 2 days half-life of T3 is a reason to stop prescribing it is reprehensible. All pharmacists should know the basic facts regarding the biological half life of medicines.[166] L-T4 is a better choice of therapy for primary hypothyroidism because of its long half life of around 7 days, but because T4 has to convert to T3, it doesn't work for those patients who have impaired peripheral conversion of T4 to T3.[2-9]
8. Her claim that the variation in hormonal content in liothyronine *may* lead to increased serum levels of T3 and subsequent thyrotoxic symptoms, such as palpitations and tremor is incorrect. Levothyroxine, liothyronine and natural desiccated porcine thyroid extract are all standardised to the specifications laid down by the US Pharmacopoeia (USP). If there was any variation in the hormonal content of the above thyroid products, L-T4 and L-T3 would not have been licensed as a medication and the MHRA would not allow them to be prescribed by NHS doctors.
9. It is true that the British National Formulary (BNF) quote 25 mcg liothyronine as equivalent to 100 mcg of levothyroxine. T3 is 4 to 5 times as potent as T4 in the blood. However, when taken in tablet form, liothyronine is 3 to 4 times as potent as levothyroxine. PrescQIPP CIC is acting negligently and irresponsibly by publishing information that fails to take account of clinical evidence and research studies.[167]
10. The RCP claim the only evidence in related medical science is contained in 11 randomised clinical trials that compare a T4 monotherapy with combination T4/T3, a claim which TPA would dispute. The authors of the T3-T4 meta-analysis[168] found 501 papers, but rejected 490. An important factor in the design of studies is the choice of subjects, and the comparison. **Since these studies are not representative of those with deficient peripheral metabolisms or increased peripheral hormone receptor resistance, they are defective.**

The focus on the thyroid gland suggests, in the literature, a 14 to 1 exchange because that is the ratio of T4 to T3 in the thyroid gland. Some studies used other ratios, such as 10:1 or 5:1. However, according to Celi,[169] the relative therapeutic value is 3:1, indicating that those on the T4/T3 combination were under-treated. Furthermore, the T3 dosage levels were generally below the starting dose for adults. Moreover, the statistical averaging makes any improvement appear negligible. **These studies should now be acknowledged as woefully deficient and discounted.** Thus the Royal Pharmaceutical Society's (RPS) recommendation that L-T4 can be used as an alternative to L-T3 will put patients at serious risk of harm if they are migrated from liothyronine to levothyroxine on the basis of replacing 25 mcg of L-T3 with 100 mcg of L-T4.

11. Liothyronine is not of "*low priority*" for those citizens suffering with inadequate cellular energy.[134,170]
12. PrescQIPP CIC, as a pharmacist, should know that all medicines have "*safety concerns*". The European Thyroid Association (ETA) Executive Committee developed guidelines specifically for the treatment of hypothyroidism with combination of L-T4 and L-T3.[171]
13. PrescQIPP CIC's claims that "*levothyroxine is an effective treatment for all*" and the RPS claim that "*levothyroxine is an alternative for liothyronine*" are without substance. The number of people in the UK who do not respond to the prescribed treatment using L-T4 monotherapy is estimated to be higher than around quarter of a million, 1.7 million people in the United States, and millions more internationally - 90 percent of whom are women. For deficiencies within the endocrine system L-T4 is good, however for post-thyroid deficiencies in the "peripheral tissue," T3 is generally needed .[172,173]

14. There is no “safer alternative” to liothyronine.
15. Liothyronine is not “*poor value for money*”. The limited value of medicines is determined by statistics that do not tell the whole story. Patients who suffer with inadequate cellular energy need T3 rather than T4.[174]
16. Liothyronine is not of “*limited clinical value*” as claimed by .PrescQIPP CIC L-T4-monotherapy is not effective for *all* patients and available evidence should be evaluated regarding the use of the liothyronine (either alone or in combination with T4) for those unable to regain optimal health on T4 monotherapy. [10-84]
17. Liothyronine has been licensed for long term (chronic) use, since Rosalind Pitt-Rivers first discovered triiodothyronine (T3) in 1952. T3 “*may be useful when absorption of levothyroxine is questionable, when impairment of peripheral conversion of thyroxine to triiodothyronine is suspected, or in patients allergic to natural thyroid hormone*”.[175]
18. PrescQIPP CIC’s claim that “*the prescribing of additional liothyronine is not recommended in any presently available formulation for Primary hypothyroidism, as it is inconsistent with normal physiology, has not been unequivocally proven to be of any benefit to patients, and may be harmful*”, has not been substantiated with scientific evidence. No long term studies have shown that T3 is harmful, yet despite such lack of evidence, the BTA warn of *potential* harm! This has generated an irrational fear of T3 among many doctors. According to Doctors Lowe and Honeyman-Lowe, many patients asking their doctors for a trial of T3 are told, “*If you take T3, you’re going to have a heart attack and die!*”[176]
19. PrescQIPP CIC’s claim that “*the variation in hormonal content may lead to increased serum levels of T3 and subsequent thyrotoxic symptoms, such as palpitations and tremor*” are contradicted by researchers with extensive clinical experience with T3. Conversely, there is much evidence to suggest that patients on T3/T4 combination do not usually feel any discomfort caused by higher T3 peaks provided by the T3/T4 treatment. Rather, there is evidence to show that they feel better.[177-185] In addition, studies comparing the effectiveness of L-T4 and L-T3 combination replacement did not report palpitations as an effect of the treatment.[118,186-191]
20. The Royal Pharmaceutical Society’s (RPS) recommendation that the prohormone levothyroxine (L-T4) can be used as an alternative to L-T3, is a **monumental scientific error**. Studies show L-T4-monotherapy does not work for all suffering with symptoms of hypothyroidism.[2-9]
21. The RPS cannot achieve “*optimal patient outcomes*” from prescribing L-T4 monotherapy for patients who need the active L-T3. Optimal outcome is defined by chemical euthyroidism. Optimal patient outcomes are achieved through randomised clinical trials evaluated with meta-analyses. However, evidence based medicine (EBM) demands neither accuracy, nor validity as required in scientific papers. [134] Endocrinology examines what is within the endocrine system only, as defined in 'The Greater Thyroid System Table'.[1] No account is being taken of what happens to the hormones after they leave the system for the peripheral tissues, which can be diminished by impaired peripheral conversion of T4 to T3.[190] cellular hormone reception, [139] and actual use within the cell.[22,24]
22. The NHS does NOT necessarily “*achieve greater value for money invested in medicines*”. It spends more money fixing problems created through inadequate cellular energy, which is produced by T3 up-regulating the cellular respiratory cycle via up-regulating the production of pyruvate from glucose. Cellular energy is needed for the proper operation of all but one of the body's 200 cell types.[134]
23. The NHS does NOT necessarily “*achieve greater value for money invested in medicines*”. It spends more money fixing problems created through inadequate cellular energy, which is produced by T3 up-regulating the cellular respiratory cycle via up-regulating the production of pyruvate from glucose. Cellular energy is needed for the proper operation of all but one of the body's 200 cell types.[134]
24. The RPC’s claim that patients are “*more engaged, understand more about their medicines and are able to make choices...*” is untrue. Whilst doctors use the narrow, albeit proper definition of 'hypothyroidism', as reflected in the thyroid function tests, patients however, identify with

'hypothyroidism' by its symptoms. This confuses patients. Further, patients do not understand what is happening, because doctors tell them their symptoms are "*non-specific*", or that they are imagining them. Patients are being deceived by the lack of proper differential diagnosis based on medical science.[161-164]

25. The RPS's claim that "*Incidents of avoidable harm from medicines are reduced*" is untrue. Failure to give patients the medicines they need when they are suffering from inadequate cellular energy is harming them, thanks to endocrinology's erroneous belief that peripheral conversion, cellular hormone reception of thyroid hormones, and the production of adenosine triphosphate works for everybody, all of the time. Endocrinology fails to consider any other possible causes for those suffering with residual symptoms on L-T4 monotherapy.[39,140-158] This is contrary to allopathic medicine, which demands proof. However, such proof does NOT exist because there are at least, 2,080 patient counterexamples to L-T4 monotherapy, whose continuing symptoms went away, or were mitigated ONLY when they started a T3 therapy.[134,170]

Evidence further supports T3-monotherapy as enabling many sufferers to become free of symptoms, possibly due to the increased potency of T3 over T4,[15,16] or because the effectiveness of T4 may be reduced in conditions that reduce the conversion of T4 to T3 such as aging, obesity, disease, stress, exercise, malnutrition; where toxic substances such as phenols, cadmium, mercury, etc. or other medicines (e.g. propranolol, amiodarone) interfere by stimulating or inhibiting the T4 to T3 conversion; where hormone or trace element deficiencies, or excess (e.g. of T3, GH, insulin, melatonin, zinc, copper, selenium, glucocorticoids, ACTH, oestrogens etc.), may inhibit the conversion of T4 to T3.[24-48] Additionally, the absorption of oral T4 can be variable (50% to 73%), versus T3 which is more constant and efficient with an absorption rate of 95%.[49,50]

26. The BTA's amended statement is supposed to address Primary hypothyroidism, and evidence should therefore relate to Primary hypothyroidism only. Unfortunately, the ambiguous wording in that statement gives the impression that it relates to ALL forms and causes of hypothyroid symptoms and signs. This is causing confusion for doctors and distress for patients.[124]
27. PrescQIPP shows bias by 'sign-posting' those seeking further information about hypothyroidism and treatment to the British Thyroid Foundation (BTF) only. The BTF is the BTA's sister organisation. No approach, reference or mention has been made to other UK thyroid support organisations such as Thyroid Patient Advocacy, to seek their views and preferences.
28. PrescQIPP's claim that they "*ensure treatments provided to patients are safe, effective and good value for money by looking at the most recent evidence on a range of conditions and treatments, and providing recommendations for them to consider*" is without substance. Such a claim can be dangerous when it involves discussion about the possible removal of a vital life-saving hormone from the NHS Prescription List that has taken no account of any of the readily available evidence and scientific research.
29. It is not difficult to dose L-T3 correctly as claimed in PrescQIPP CIC's document. Interpreting blood tests is more difficult but patient response is better, more stable and more predictable with L-T3. High-dose levothyroxine can disrupt deiodinase mechanisms.[192]
30. The cost savings claimed in Bulletin 121 are grossly inaccurate. The BTA statement addresses Primary hypothyroidism, which accounts for just a small proportion of patients being prescribed L-T3. Around 85% of sufferers of Primary hypothyroidism appear to do well on L-T4 monotherapy so cost savings will be correspondingly small.[122]

PrescQIPP has a duty of care [210] and should be aware of ethics to their deliberations. Under the Law of Tort, a duty of care is a legal obligation which is imposed on an individual or organisation, requiring adherence to a standard of reasonable care while performing any acts that could foreseeably cause harm. It is the first element that **must** be established to proceed with an action in negligence. The Law of Tort requires *inter alia* that individuals, or organisations "**should know or should have known**" of available evidence and current scientific knowledge in order to avoid decision making leading to any act resulting in harm.[193]

PrescQIPP, BTA, BTF, NICE, NHS UK Medicines Information (UKMi), all local CCGs plus others involved in such decision making should know, or should have known of:

- the warnings about the failure of T4 monotherapy (Kirk & Kvorning, 1947),<sup>[194]</sup> (Means, 1954) *Means JH*, <sup>[195]</sup> (Baisier, Hertoghe, and Eeckhaut, Circa 2001),<sup>[195]</sup> and the greater activity of T3 over T4 (Gross & Pitt-Rivers, 1952),<sup>[15]</sup>
- the potential for euthyroid hypometabolism (Goldberg, 1960) showing that intracellular chemistry depends upon T3, and not T4,<sup>[139,140]</sup>
- the discovery of the physiology that connects the thyroid gland to the peripheral, symptom-producing cells. (Refetoff, 1957),<sup>[196]</sup>
- the dissatisfaction of many people using L-T4 monotherapy. (Saravanan, Dayan),<sup>[120,123]</sup>
- the existence of numerous subsequent studies on the characteristics of peripheral conversion or metabolism of T4 to T3, and peripheral cellular hormone reception functions,<sup>[10-84]</sup>
- patient counterexamples to T4-monotherapy whose symptoms were mitigated, or went away completely after treatment with T3 was initiated,<sup>[134]</sup>
- the demands of Differential Diagnostic Protocols,<sup>[165-168]</sup>
- the linguistic and logical standards of care,<sup>[197-204]</sup>
- the very common syndrome of thyroid and adrenal deficiency, and the science showing that the above syndrome has global effects, with imbalance of other hormones; the likely presence of systemic candida and dysbiosis, malabsorption and food allergy, all playing a probable role,<sup>[164]</sup>
- the need to use observation and medicine practised as an art as the primary diagnostic method, with the laboratory playing a secondary role,<sup>[163]</sup>
- their Duty of Care,<sup>[210]</sup>
- their vicarious liability, through the actions of medical practitioners who, following their guidance, are failing to bring patients back to optimal health.

Before PrescQIPP provide “... *explanations as to why L-T3 has been included in the DROP-List*”, it might have been better had they contacted Amdipharm Mercury Ltd. (AMCo) first, asking them to supply an analysis of the reason for the huge price increase, giving a breakdown in the constituent costs involved.

Liothyronine (L-T3) is a long-established, safe and effective thyroid hormone replacement for those who continue suffering symptoms whilst on L-T4-monotherapy. Liothyronine is of simple construction, and is cheap to both develop and manufacture.

Branded medicines are subject to price regulation. Up until 2007, the price of the brand *Tertroxin* therefore remained stable at £15.92 for 100 tablets. However, it does appear that the brand name *Tertroxin* may have been removed by the present owner Amdipharm Mercury Ltd., (AMCo) in order to by-pass price regulation. The product is now marketed as generic liothyronine. In March 2016, the cost of liothyronine had risen to a staggering £922.44 (based on 28 tablets at £256.20).

Generic drugs are, in theory, subject to competition. The UK liothyronine is therefore no longer subject to price regulation. Amdipharm Mercury Ltd. (AMCo) have written to Thyroid Patient Advocacy, stating that their UK liothyronine formula is “*unique and not interchangeable with other products in the market*”. However, there is NO competition. The Department of Health appear to have handed the monopoly of manufacturing and selling liothyronine in the UK to Amdipharm Mercury Ltd. (AMCo). Resultantly, there is thus NO competition. Incidentally, President Obama became involved in a similar case in the United States. <sup>[205]</sup>

Doctors can no longer write the brand name *Tertroxin* on a prescription form. They have to write instead liothyronine. As a result, the NHS local CCG's are writing to doctors advising that they should no longer prescribe generic T3, because of the huge cost to the NHS, and are recommending that they prescribe levothyroxine as an alternative. The consequences of this can be devastating to those approximately quarter of a million patients in the UK who need T3, as can be ascertained from the evidence given above.

Given the seriousness of the matter, and also considering PrescQIPP's 'OBJRCTS', an investigation into the reasons for why the cost to the NHS has been allowed to continue rising, should be the first priority. Investigation into this matter should also delve into why there was no action taken by the Department of Health, and/or the MHRA, despite them having been approached by various MP's asking for an explanation. The potential for devastating effects on patient's lives should also be recognised, and a case for medical negligence should be given consideration.

It is of great concern that AMCo have countered that their 'generic' UK liothyronine is "in fact" "unique and not interchangeable with other products in the market". The MHRA,[206] and British Generics,[207] inform us that generics that are NOT interchangeable MUST HAVE A BRAND NAME. A previously mentioned, the brand Tertroxin was removed some time in 2007, resulting in the product being marketed as generic liothyronine instead.

*"A generic medicine contains the same active ingredient as the equivalent original branded drug, and is marketed once the originator's patent protection has expired. Generics are authorised to the same standards of safety, quality and efficacy as original branded drugs, and have to demonstrate in clinical studies that they are bioequivalent to the original product: i.e., they deliver equal medical benefits to the patient.*

*Generic medicines are therefore normally interchangeable with the equivalent branded drug. On the rare occasions where this is not the case, the MHRA (Medicines and Healthcare products Regulatory Agency) requires generic medicines to have a brand name so that patients may be maintained on a single manufacturer's product."*

It is of great concern that in Europe, the cost of L-T3 is a mere fraction of what it costs in the UK.

Sweden -	100 tablets of L-T3 = £21.10
Finland -	100 tablets of L-T3 = £15.81
Norway -	100 tablets of L-T3 20mcgs – NOK 254.50 = 27.51€ = £21.65
Denmark -	100 tablets of L-T3 20mcgs – Thybon 20 Henning = DKK 190 = 25.53€ = £20.09
Germany -	100 tablets of L-T3 20mcgs -30.15€
Turkey -	100 tablets of L-T3 – Tiromel = £1.25 (info. from private correspondence)
UK -	100 tablets of T3 - Liothyronine = <b>£922.44</b> (based on 28 tablets at £256.20)

In PrescQIPP's Reg. Company statement, it is stated in their OBJECTS: "***The objects of the Company are to carry on activities which benefit the community and in particular (without limitation) to help NHS organisations to improve medicines-related care to patients, through the delivery of robust, accessible and evidence-based resources.***"

PrescQIPP's statement regarding liothyronine (L-T3) has failed to adduce any such evidence.

**References:** <http://www.tpauk.com/main/article/references-for-tpa-rebuttal-to-prescqipp/>