**Record of Determinations – Medical Practitioners Tribunal**

**PUBLIC RECORD**

**Dates:** 20 November to 8 December 2017  
22 to 24 October 2018  
29 October to 9 November 2018  
24 June to 5 July 2019  
25 to 27 September 2019

**Medical Practitioner’s name:** Dr Georges MOUTON

**GMC reference number:** 4689715

**Primary medical qualification:** MD 1983 Universite de l'Etat a Liege

**Type of case**  
New - Misconduct  
Outcome on impairment

**Summary of outcome**  
Suspension, 9 months  
Review hearing directed  
Immediate order imposed

**Tribunal:**

<table>
<thead>
<tr>
<th>Legally Qualified Chair:</th>
<th>Mr Charles Thomas</th>
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</table>
| Medical Tribunal Members: | Dr Hazel Busby-Earle  
Dr Tushar Vince |

| Tribunal Clerk: | 20 November to 8 December 2017: Ms Dee Montgomery  
22 to 24 October 2018: Mr Matthew O'Reilly  
29 October to 9 November 2018: Ms Jeanette Close |

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24 June to 5 July 2019:
Mr Matthew O’Reilly

25 to 27 September 2019:
Ms Esther Morton

Attendance and Representation:

<table>
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<tr>
<th>Medical Practitioner:</th>
<th>Present and represented</th>
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<tr>
<td>Medical Practitioner’s Representative:</td>
<td>Mr Selva Ramasamy, Counsel, instructed by Eastwoods Solicitors</td>
</tr>
<tr>
<td>GMC Representative:</td>
<td>20 November to 8 December 2017: Mr Craig Sephton QC, Counsel, instructed by GMC Legal</td>
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<tr>
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<td>All other dates: Mr Peter Atherton, Counsel, instructed by GMC Legal</td>
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Attendance of Press / Public
In accordance with Rule 41 of the General Medical Council (Fitness to Practise) Rules 2004 the hearing was held in public.

Overarching Objective
Throughout the decision-making process the Tribunal has borne in mind the statutory overarching objective as set out in s1 Medical Act 1983 (the 1983 Act) to protect, promote and maintain the health, safety and well-being of the public, to promote and maintain public confidence in the medical profession, and to promote and maintain proper professional standards and conduct for members of that profession.
DETERMINATION ON FACTS - 25/06/2019

Background

1. Dr Mouton qualified in 1983 from Liege University Medical School, Belgium. He has been registered with the GMC since 2000. Prior to and during the time of the events that are the subject of this hearing, Dr Mouton was practising privately in the UK and abroad as a General Practitioner, specialising in Functional Medicine. Functional Medicine is a systems, biology based approach to diagnosis and care, developed and taught by the Institute of Functional Medicine in the USA, established in 1991. Functional Medicine is not a recognised specialty in the UK. In London Dr Mouton practised at the Hale Clinic, Park Crescent, W1.

2. The Allegation that has led to Dr Mouton’s hearing can be summarised as concerns arising from his care of eight patients in the following areas:

   - Assessment
   - Diagnosis
   - Investigation
   - Prescribing
   - Communication
   - Record keeping
   - Treatment.

3. The GMC allege that Dr Mouton’s practice breached some of the tenets of Good Medical Practice and other guidelines.

4. Concerns were raised by a number of general practitioners with the GMC, initially on 17 February 2015 by Dr I, GP Partner at the practice at which Patients A and B were registered.

Patient A

5. On various dates, Dr Mouton consulted with Patient A. Dr Mouton first saw Patient A at Hale Clinic on 10 April 2013. At the time of the first consultation Patient A was 21 years of age. Patient A had been experiencing extreme fatigue and low mood. Patient A’s father had contacted Dr Mouton by email prior to the appointment indicating Patient A was going through depression, compounded by Patient A’s sleeping pattern, weight loss, appetite, fatigue, aches and pains, problems with concentration, and possibly poor circulation.

6. Patient A’s father also indicated that Patient A admitted to having experimented with “unhealthy stuff” at university and that his son was also “going
through a phase of being man of few words”, which was the principal reason for sending the confidential email ahead of the appointment.

7. It is alleged that Dr Mouton failed to elicit from Patient A adequate information about possible drug misuse, decline in academic performance, depressed mood, social withdrawal, paranoid symptoms and failed adequately to assess Patient A’s mental state. It is also alleged that Dr Mouton attributed Patient’s A’s symptoms to hypothyroidism, nutritional issues, obsessive compulsive disorder, and leaky gut syndrome. It is further alleged that Dr Mouton failed to make a differential diagnosis of a psychiatric disorder in light of Patient A’s presenting symptoms.

8. After Patient A consulted with Dr Mouton, Dr Mouton undertook a series of investigations. Based on the results, Dr Mouton diagnosed Patient A with autoimmune thyroiditis and prescribed multiple treatments including dietary supplements and Thyroxine (T4).

9. Dr Mouton saw Patient A again in July 2014. He took further blood tests and prescribed further dietary supplements and added Tri-iodothyronine (T3) to the existing T4 treatment.

10. On 20 November 2014 Patient A attended his local police station to report allegations of harassment. The police report described him as very paranoid and extremely agitated.

11. Dr Mouton had a further consultation with Patient A on 25 November 2014. He took further blood tests. Dr Mouton updated his prescription on 12 December 2014 with a number of dietary supplements and treatments including thyroxine, iodine and Tirform. It is alleged that the dietary supplements and treatments were not clinically indicated or supported by scientific evidence.

12. During the course of his management of Patient A, Dr Mouton undertook investigations of Patient A’s urinary thyroid hormones, steroid hormones, insulin and DIO2 genotype. It is alleged that these investigations were not clinically indicated.

13. Patient A’s General Practitioner sent a fax to Dr Mouton on 15 December 2014 dealing with Patient A’s attendance at the police station and his mental health status. Patient A’s General Practitioner requested information about the thyroid tests and treatment. Dr Mouton responded on 19 December 2014. Patient A’s General Practitioner responded seeking further clarification in January 2015 and Dr Mouton responded again on 21 January 2015.

14. Patient A’s father also emailed Dr Mouton on 15 December 2014 seeking advice as to whether Dr Mouton would recommend that Patient A should be referred to a Psychiatrist. Dr Mouton responded by email on the same date.
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15. It is alleged that Dr Mouton failed to communicate with Patient A’s General Practitioner regarding Patient A’s diagnosis of autoimmune thyroid disease with hypothyroidism, prescriptions for thyroxine and T3 therapy and failed to communicate clearly with Patient A’s father, in that Dr Mouton provided contradictory advice regarding referring Patient A to a psychiatrist.

Patient B

16. Patient B registered with a new General Practitioner on 21 October 2014. At his first consultation with his GP on 21 January 2015, Patient B informed his GP that he was receiving thyroid hormone treatment prescribed by Dr Mouton.

17. Dr Mouton consulted with Patient B between September 2014 and September 2015. Patient B was a 51 year old male when he first had a consultation with Dr Mouton. Patient B’s main complaint was of feeling tired and lethargic. Dr Mouton undertook blood and urine tests and based on the results, diagnosed Patient B as suffering from an autoimmune thyroiditis. It is alleged that Dr Mouton attributed Patient B’s symptoms of acute lethargy to hypothyroidism when there was no sound clinical basis for doing so.

18. Dr Mouton referred Patient B for CTLA-4 genotype testing and VDR genotype testing. It is alleged that these investigations were not clinically indicated and were conducted without obtaining informed consent from Patient B.

19. It is further alleged that Dr Mouton referred Patient B for investigation of: urinary thyroid hormones, steroid hormones, and insulin when they were not clinically indicated.

20. It is also alleged that Dr Mouton prescribed 16 treatments including Novothyral (combination T3 and T4), Iodine, Selenium and Pregnenolone when they were not clinically indicated, supported by scientific guidelines or evidence based.

21. Patient B’s General Practitioner wrote to Dr Mouton on 30 January 2015 requesting details of his treatment of Patient B. Dr Mouton responded on 16 February 2015 stating that Patient B was receiving thyroid hormone treatment. It is alleged that Dr Mouton failed to communicate adequately with Patient B’s General Practitioner with regards to his diagnoses and treatment.

Patient C

22. Dr Mouton consulted with Patient C, a 33 year old lady, between 15 December 2013 and 22 September 2015. Patient C had been diagnosed with ulcerative colitis two years previously and her main concern regarded continuing symptoms. Patient C had been under follow-up care with a number of gastroenterologists.
23. Patient C saw Dr Mouton for investigations and follow up consultations including a number of nutritional, genotype blood tests and miscellaneous thyroid tests. It is alleged that Dr Mouton referred Patient C for investigation of urinary thyroid hormones and thyroid ultrasound scan which were not clinically indicated.

24. Consequently Dr Mouton prescribed 21 treatments including Iodine, Pregnenolone and Armour Thyroid.

25. Patient C was referred by her General Practitioner to a Consultant Endocrinologist for an opinion regarding her hair loss. Following a consultation on the 23 April 2015, the Consultant Endocrinologist noted that Dr Mouton was using thyroxine for this patient. He noted he was not sure whether this was an effective treatment for her hair loss and that he did not think Patient C had underlying thyroid disease.

26. As part of Dr Mouton’s management of Patient C he referred her for seven different genotype tests. It is alleged that these tests were not clinically indicated, conducted without obtaining informed consent or without appropriate counselling of Patient C. It is further alleged that Dr Mouton failed to document any clear diagnosis or any differential diagnosis.

27. Dr Mouton sent a letter to Patient C’s General Practitioner, dated 12 July 2015, in which he stated he had issued prescriptions and dietary recommendations for the next 6 months and would see Patient C again in 5 months. It is alleged that Dr Mouton failed to communicate adequately with Patient C regarding her therapeutic treatment plan, Patient C’s General Practitioner regarding the tests referred to in Schedule 2 and their implications and Patient C’s treating Gastroenterologist regarding the intestinal and dietary treatments.

**Patient D**

28. Dr Mouton consulted with Patient D between 14 January 2014 and 9 July 2015. Patient D was a 45 year old lady who had been generally unwell and suffered a series of non-specific symptoms including high cholesterol, fatigue, anxiety, constipation, low libido, feeling cold, chilblains and dry skin.

29. Over the course of subsequent visits Dr Mouton prescribed a number of investigations including genotype testing, urinary thyroid tests and ultrasound scans. It is alleged that these investigations were not clinically indicated. It is also alleged that the genotype testing was conducted without obtaining informed consent and without appropriate counselling of Patient D.

30. On the basis of these tests Dr Mouton prescribed 19 treatments including Pregnenolone and Novothyral (combination T3/T4 treatment). It is alleged that
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these treatments were not clinically indicated, supported by scientific guidelines or evidence based.

31. Dr Mouton informed Patient D’s General Practitioner, in a letter dated 31 January 2015, that he had diagnosed Patient D with hypothyroidism. He stated his treatment plan was with T4 and T3, in increasing doses.

32. Patient D’s General Practitioner referred her to an NHS Endocrinologist who considered that T4 and T3 treatment was not indicated. Patient D’s General Practitioner subsequently referred the case to the GMC stating that Dr Mouton prescribed thyroid hormone despite normal thyroid function tests.

Patient E

33. Patient E consulted with Dr Mouton between 3 September 2014 and 20 August 2015. Patient E was a 31 year old lady who suffered from inflammatory bowel disease, various food allergies and was already taking dietary supplements prior to her first consultation with Dr Mouton.

34. During this time period Dr Mouton undertook a wide range of blood tests and investigations including genotype testing. Thereafter Dr Mouton prescribed multiple treatments including Iodine, Pregnenolone and Novothyral. Patient E saw Dr Mouton on 14 June 2015. It is recorded that the treatments seemed to be helping, and Patient E seemed “generally okay, if a bit tired”.

35. Dr Mouton’s document of 12 July 2015 lists 17 prescribed medications and Dr Mouton’s comment that “despite low TSH level, the patient needs more thyroid help as shown by a) symptoms, b) low hormone level (especially active t3 in urine), c) high cholesterol, d) anaemic tendency!”


37. It is alleged that the investigations of urinary thyroid hormones and the thyroid ultrasound scan were not clinically indicated. It is further alleged that Dr Mouton referred Patient E for genotype testing which was not clinically indicated, conducted without obtaining informed consent from Patient E and conducted without appropriate counselling of Patient E.
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38. It is also alleged that Dr Mouton failed to adequately interpret or act upon the findings that Patient E was biochemically euthyroid and wrongly diagnosed Patient E with adrenal insufficiency. Further it is alleged that Dr Mouton prescribed treatments which were not clinically indicated, supported by scientific guidelines or evidence based and that he failed to communicate adequately with Patient E’s General Practitioner regarding tests undertaken and their implications and a Gastroenterologist regarding the intestinal treatments he intended to prescribe.

Patient F

39. Patient F consulted with Dr Mouton between 17 January 2014 and 16 December 2015. At the time of the initial consultation Patient F was 12 years and 8 months of age and suffered a series of non-specific symptoms including an itchy rash, abdominal pain/wind/colic, poor concentration, restless legs and fatigue.

40. Dr Mouton organised genotype testing. It is alleged that these genotype investigations were not clinically indicated, conducted without obtaining informed consent from Patient F and/or from his parent or guardian, and conducted without appropriate counselling of Patient F and/or his parent or guardian.

41. Following a series of blood and urine tests, Dr Mouton made a diagnosis of Intestinal Dysbiosis. In a treatment proforma dated 5 February 2014, 14 specific treatments are prescribed for Patient F. Dr Mouton recorded his interpretation to include gluten allergy and “sluggish” thyroid function.

42. On 6 June 2014 Dr Mouton saw Patient F and recorded improved appetite and energy, reduced bowel noise, and improved skin.

43. In a further consultation, dated 19 December 2014, Dr Mouton recorded that Patient F was losing weight. Following further investigations Dr Mouton prescribed Armour Thyroid (combination T3/T4).

44. It is alleged that Dr Mouton failed to adequately interpret or act upon the findings that Patient F was biochemically euthyroid. It is also alleged that Dr Mouton prescribed thyroid hormone treatment, which was not clinically indicated, supported by scientific guidelines or evidence based.

45. It is alleged that Dr Mouton failed to communicate appropriately with Patient F’s General Practitioner in that he did not include information in his letters regarding his previous and/or new diagnoses, key results, investigations or treatment plans. Further, that Dr Mouton failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing namely: why the tests were carried out, the degree of explanation given to Patient F’s parent and/or Patient F prior to taking the test, and any implications that the General Practitioner should be aware of arising from the results of the genetic testing. It is also alleged
that Dr Mouton suggested that the results of the genetic testing should not be entered into Patient F’s medical records.

**Patient G**

46. Patient G consulted with Dr Mouton between 17 January 2014 and 16 December 2015. Dr Mouton first saw Patient G with his brother Patient F. At this time, Patient G was 15 years and 8 months of age and suffered a series of symptoms including fatigue, poor concentration and memory, a struggle to gain weight, hypoglycaemia, tinnitus, low temperature and feeling the cold. A range of tests was undertaken, including genotype testing and a diagnosis of Intestinal Dysbiosis was made. A prescription dated 14 January 2015, lists 18 different treatments including Armour Thyroid (combination T3/T4 treatment). The doses of thyroid hormone were amended over the course of subsequent visits.

47. It is alleged that Dr Mouton referred Patient G for genotype testing which was not clinically indicated, conducted without obtaining informed consent from Patient G and/or from his parent or guardian, conducted without appropriate counselling of Patient G and/or his parent or guardian.

48. It is further alleged that Dr Mouton failed to adequately interpret or act upon the findings that Patient G was biochemically euthyroid. It is also alleged that Dr Mouton prescribed Armour Thyroid treatment to Patient G, a treatment that was not clinically indicated, supported by scientific guidelines or evidence based.

49. It is alleged that Dr Mouton failed to communicate appropriately with Patient G’s General Practitioner in not including information in his letters regarding his previous and/or new diagnoses, key results, investigations or treatment plans. Further, that Dr Mouton failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing namely: why the tests were carried out, the degree of explanation given to Patient G’s parent and/or Patient G prior to taking the test, and any implications that the General Practitioner should be aware of arising from the results of the genetic testing. It is also alleged that Dr Mouton suggested that the results of the genetic testing should not be entered into Patient G’s medical records.

**Patient H**

50. Patient H consulted with Dr Mouton between 18 February 2016 and 22 March 2016. Patient H was a 67 year old lady and referred herself to Dr Mouton with a chronic dry cough and recurrent chest infections.

51. In the first consultation Patient H also reported non-specific systemic symptoms including poor sleep, poor concentration and memory, dry skin and hair, brittle nails, poor teeth, receding and bleeding gums, dry and gritty eyes, joint pains,
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cold hands and feet, bloating, heartburn and abdominal cramps. At this time Patient H was already taking fatty acid supplements, vitamin D and fish oil, but no regular prescribed medication.

52. Dr Mouton arranged a wide variety of blood and urine tests including genotype testing. Dr Mouton subsequently prescribed dietary treatments, thyroid extracts and pregnenolone.

53. It is alleged that the genotype testing was not clinically indicated, conducted without obtaining informed consent from Patient H and conducted without appropriate counselling of Patient H.

54. It is further alleged the Dr Mouton failed to adequately interpret or act upon the findings that Patient H was biochemically euthyroid and on Patient H’s steroid blood results. It is further alleged that Dr Mouton prescribed treatments which were not clinically indicated, supported by scientific guidelines or evidence based.

55. It is alleged that Dr Mouton failed to make any diagnosis or differential diagnosis that explained Patient H’s presenting complaint, namely, a chronic cough. It is also alleged that Dr Mouton failed to adequately or properly communicate to Patient H or her General Practitioner why he had referred Patient H for the genetic tests; the implications of the results of the genetic tests, Dr Mouton’s diagnosis and why he had prescribed the treatments referred to in Schedule 6.

The Outcome of Application(s) made during the Facts Stage

56. The Tribunal refused the GMC’s application, made pursuant to Rule 16A(2)(b) of the General Medical Council (Fitness to Practise) Rules 2004, as amended (the Rules), that the Tribunal refuse to admit the expert report of Dr J due to its late disclosure. The Tribunal determined that it would be unfair to Dr Mouton to refuse to admit Dr J’s report and that fairness to the GMC could be ensured by allowing the GMC any necessary time it required to consider the report with its own experts and respond accordingly.

The Allegation and the Doctor’s Response

57. The Allegation made against Dr Mouton is as follows:

**Patient A**

1. On various dates between 10 April 2013 and 18 November 2014, you consulted with Patient A and you:
   
a. failed to elicit from Patient A adequate information about:
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i. possible drug misuse;
   To be determined

ii. decline in academic performance;
    To be determined

iii. depressed mood;
     To be determined

iv. social withdrawal;
    To be determined

v. paranoid symptoms;
   To be determined

b. failed adequately to assess Patient A’s mental state;
   To be determined

c. attributed Patient’s A’s symptoms to:

i. hypothyroidism;
   Admitted and found proved

ii. nutritional issues;
    Admitted and found proved

iii. obsessive compulsive disorder;
     To be determined

iv. leaky gut syndrome;
    Admitted and found proved

d. failed to make a differential diagnosis of a psychiatric disorder in light of Patient A’s presenting symptoms.
   To be determined

2. You referred Patient A for the investigation of:

a. urinary thyroid hormones;
   Admitted and found proved

b. steroid hormones;
   Admitted and found proved
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c. insulin;  
   **Admitted and found proved**

d. DIO2 genotype testing.  
   **Admitted and found proved**

3. The investigations referred to in paragraph 2 were not clinically indicated. **To be determined**

4. You prescribed dietary treatments for Patient A which were not:
   
a. clinically indicated;  
      **To be determined**

   b. supported by scientific guidelines;  
      **To be determined**

   c. supported by sound scientific evidence ("not evidence-based").  
      **To be determined**

5. You failed to communicate with Patient A’s General Practitioner regarding Patient A’s:
   
a. diagnosis of autoimmune thyroid disease with hypothyroidism;  
      **To be determined**

   b. prescriptions for thyroxine and T3 therapy.  
      **To be determined**

6. In an email to Patient A’s father dated 15 December 2014, you:
   
a. advised him that psychiatrists ‘**too often react with a very heavy chemical load which I find dangerous and sometimes definitely destructive**’;  
      **Admitted and found proved**

   b. failed to communicate clearly with Patient A’s father, in that you provided contradictory advice regarding referring Patient A to a psychiatrist.  
      **To be determined**

**Patient B**

7. Between 1 September 2014 and 1 September 2015 you consulted with Patient B and you referred Patient B for the following investigations:
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a. CTLA-4 genotype testing;
   Admitted and found proved

b. VDR genotype testing.
   Admitted and found proved

8. The investigations referred to in paragraph 7 were:
   a. not clinically indicated;
      To be determined
   b. conducted without obtaining informed consent from Patient B;
      To be determined
   c. conducted without appropriate counselling of Patient B.
      To be determined

9. You referred Patient B for investigation of:
   a. urinary thyroid hormones;
      Admitted and found proved
   b. steroid hormones;
      Admitted and found proved
   c. insulin.
      Admitted and found proved

10. The investigations referred to in paragraph 9 were not clinically indicated.
    To be determined

11. You attributed Patient B’s symptoms of acute lethargy to hypothyroidism when there was no sound clinical basis for doing so.
    To be determined

12. You prescribed Patient B the treatments set out in Schedule 1, which were not:
   a. clinically indicated;
      To be determined
   b. supported by scientific guidelines;
      To be determined
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c. evidence based.
   To be determined

13. You failed to communicate adequately with Patient B’s General Practitioner regarding:

   a. Patient B’s diagnoses, including:
      
      i. hypothyroidism;
         To be determined
      
      ii. iodine deficiency;
         To be determined
      
      iii. hypogonadism;
         To be determined

   b. Patient B’s prescriptions as set out in Schedule 1.
      To be determined

Patient C

14. Between 15 December 2013 and 22 September 2015, you consulted with Patient C and you failed to record:

   a. any clear diagnosis;
      To be determined

   b. any differential diagnoses.
      To be determined

15. You referred Patient C for the investigations set out in Schedule 2.
    Admitted and found proved

16. The investigations referred to in Schedule 2 were:

   a. not clinically indicated;
      To be determined

   b. conducted without obtaining informed consent from Patient C;
      To be determined

   c. conducted without appropriate counselling of Patient C.
      To be determined
17. You referred Patient C for:
   a. investigation of urinary thyroid hormones;  
      Admitted and found proved
   b. thyroid ultrasound scan.  
      Admitted and found proved

18. The investigations referred to in paragraph 17 were not clinically indicated.  
    To be determined

19. You prescribed Patient C the treatments set out in Schedule 3, which were not:
   a. clinically indicated;  
      To be determined
   b. supported by scientific guidelines;  
      To be determined
   c. evidence based.  
      To be determined

20. You failed to communicate adequately with:
   a. Patient C regarding her therapeutic treatment plan;  
      To be determined
   b. Patient C’s General Practitioner regarding the tests referred to in Schedule 2 and the implications of them;  
      To be determined
   c. Patient C’s treating Gastroenterologist regarding the intestinal and dietary treatments referred to in Schedule 3.  
      To be determined

**Patient D**

21. Between 14 January 2014 and 9 July 2015, you consulted with Patient D and you referred Patient D for the following investigations:
   a. investigation of urinary thyroid hormones;  
      Admitted and found proved
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b. thyroid ultrasound scan.  
   Admitted and found proved

22. The investigations referred to in paragraph 21 were not clinically indicated.  
   To be determined

23. You referred Patient D for the investigation of DIO2 genotype testing and such investigation was:

   a. not clinically indicated;  
      To be determined

   b. conducted without obtaining informed consent from Patient D;  
      To be determined

   c. conducted without appropriate counselling of Patient D.  
      To be determined

24. You failed to adequately interpret or act upon the findings that Patient D was biochemically euthyroid.  
   To be determined

25. You prescribed the treatments set out in Schedule 4 which were not:

   a. clinically indicated;  
      To be determined

   b. supported by scientific guidelines;  
      To be determined

   c. evidence based.  
      To be determined

Patient E

26. Between 3 September 2014 and 20 August 2015, you consulted with Patient E and you referred Patient E for the following investigations:

   a. investigation of urinary thyroid hormones;  
      Admitted and found proved

   b. thyroid ultrasound scan.  
      Admitted and found proved
27. The investigations referred to in paragraph 26 were not clinically indicated. **To be determined**

28. You referred Patient E for the following investigations:
   a. DIO2 genotype testing; **Admitted and found proved**
   b. Apo E genotype testing; **Admitted and found proved**
   c. CTLA-4 genotype testing. **Admitted and found proved**

29. The investigations referred to in paragraph 28 were:
   a. not clinically indicated; **To be determined**
   b. conducted without obtaining informed consent from Patient E; **To be determined**
   c. conducted without appropriate counselling of Patient E. **To be determined**

30. You failed to adequately interpret or act upon the findings that Patient E was biochemically euthyroid. **To be determined**

31. You wrongly diagnosed Patient E with adrenal insufficiency. **To be determined**

32. You prescribed the treatments referred to in Schedule 5 which were not:
   a. clinically indicated; **To be determined**
   b. supported by scientific guidelines; **To be determined**
   c. evidence based. **To be determined**

33. You failed to communicate adequately with:
a. Patient E’s General Practitioner regarding the tests referred to in paragraph 28 and the implications of them;  
   To be determined
b. a Gastroenterologist regarding the intestinal treatments you intended to prescribe.  
   To be determined

Patient F

34. Between 17 November 2014 and 16 December 2015 you consulted with Patient F and you referred him for the following investigations:
   a. Apo E genotype testing;  
      Admitted and found proved
   b. DIO2 genotype testing;  
      Admitted and found proved
   c. FUT2 genotype testing.  
      Admitted and found proved

35. The investigations referred to in paragraph were:
   a. not clinically indicated;  
      To be determined
   b. conducted without obtaining informed consent from Patient F and/or from his parent or guardian;  
      To be determined
   c. conducted without appropriate counselling of Patient F and/or his parent or guardian.  
      To be determined

36. You failed to adequately interpret or act upon the findings that Patient F was biochemically euthyroid.  
    To be determined

37. You prescribed thyroid hormone treatment, Armour thyroid, to Patient F which treatment was not:
   a. clinically indicated;  
      To be determined
b. supported by scientific guidelines;
   **To be determined**

c. evidence based.
   **To be determined**

38. You failed to communicate appropriately with Patient F’s General Practitioner in that you:

a. did not include information in your letters to Patient F’s General Practitioner regarding his:

   i. previous and/or new diagnoses;
      **To be determined**

   ii. key results;
      **To be determined**

   iii. investigations;
      **To be determined**

   iv. treatment plans;
      **To be determined**

b. failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing to which you referred Patient F, namely:

   i. why the tests were carried out;
      **To be determined**

   ii. the degree of explanation given to Patient F’s parent and/or Patient F prior to taking the test;
      **To be determined**

   iii. any implications that the General Practitioner should be aware of arising from the results of the genetic testing;
      **To be determined**

c. suggested that the results of the genetic testing that you sent to the General Practitioner should not be entered into the medical notes of Patient F. **To be determined**

**Patient G**
39. Between 17 November 2014 and 16 December 2015 you consulted with Patient G and you referred him for the following investigations:

   a. Apo E genotype testing;  
      **Admitted and found proved**

   b. DIO2 genotype testing;  
      **Admitted and found proved**

   c. FUT2 genotype testing.  
      **To be determined**

40. The investigations referred to in paragraph 39 were:

   a. not clinically indicated;  
      **To be determined**

   b. conducted without obtaining informed consent from Patient G and/or from his parent or guardian;  
      **To be determined**

   c. conducted without appropriate counselling of Patient G and/or his parent or guardian.  
      **To be determined**

41. You failed to adequately interpret or act upon the findings that Patient G was biochemically euthyroid.  
    **To be determined**

42. You prescribed thyroid hormone treatment, Armour thyroid, to Patient G which treatment was not:

   a. clinically indicated;  
      **To be determined**

   b. supported by scientific guidelines;  
      **To be determined**

   c. evidence based.  
      **To be determined**

43. You failed to communicate appropriately with Patient G’s General Practitioner in that you:
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a. did not include information in your letters to Patient G’s General Practitioner regarding his:

i. previous and/or new diagnoses;
   To be determined

ii. key results;
   To be determined

iii. investigations;
   To be determined

iv. treatment plans;
   To be determined

b. failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing to which you referred Patient G, namely:

i. why the tests were carried out;
   To be determined

ii. the degree of explanation given to Patient G’s parent and/or Patient G prior to taking the test;
   To be determined

iii. any implications that the General Practitioner should be aware of arising from the results of the genetic testing;
   To be determined

c. suggested that the results of the genetic testing that you sent to the General Practitioner should not be entered into the medical notes of Patient G. To be determined

Patient H

44. Between 18 February 2016 and 22 March 2016, you consulted with Patient H and you referred Patient H for the following investigations:

a. APOE genotype testing;
   Admitted and found proved

b. DIO2 genotype testing.
   Admitted and found proved
45. The investigations referred to in paragraph 44 were:
   a. not clinically indicated;  
      To be determined
   b. conducted without obtaining informed consent from Patient H;  
      To be determined
   c. conducted without appropriate counselling of Patient H.  
      To be determined

46. You failed to adequately interpret or act upon:
   a. the findings that Patient H was biochemically euthyroid;  
      To be determined
   b. Patient H’s steroid blood results.  
      To be determined

47. You prescribed the treatments referred to in Schedule 6 which were not:
   a. clinically indicated;  
      To be determined
   b. supported by scientific guidelines;  
      To be determined
   c. evidence based.  
      To be determined

48. You failed to make any diagnosis or differential diagnosis that explained Patient H’s presenting complaint, namely, a chronic cough.  
    To be determined

49. You failed to adequately or properly communicate to Patient H or her General Practitioner:
   a. why you had referred Patient H for the genetic tests referred to in paragraph 44;  
      Admitted and found proved
   b. the implications of the results of the genetic tests referred to in paragraph 44;  
      Admitted and found proved
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c. your diagnosis;
   To be determined

d. why you had prescribed the treatments referred to in Schedule
   6. To be determined

The Admitted Facts

58. At the outset of these proceedings, through his counsel, Mr Ramasamy, Dr Mouton made admissions to some paragraphs and sub-paragraphs of the Allegation, as set out above, in accordance with Rule 17(2)(d) of the Rules. In accordance with Rule 17(2)(e) of the Rules, the Tribunal announced these paragraphs and sub-paragraphs of the Allegation as admitted and found proved.

Factual Witness Evidence

59. The Tribunal received evidence on behalf of the GMC from the following witness:

   • Dr K, General Practitioner (GP) to Patients A & B, via video-link.

60. The Tribunal also received evidence on behalf of the GMC in the form of witness statements from the following witnesses who were not called to give oral evidence:

   • Dr L, GP to Patient E;
   • Dr M, GP to Patients F & G;
   • Dr N, Consultant Psychiatrist in General Adult Psychiatry;
   • Dr I, GP to Patients A, B & C.

61. Dr Mouton provided his own witness statement(s), dated 21 November 2017, and also gave oral evidence at the hearing. In addition, the Tribunal received evidence from the following witness on Dr Mouton’s behalf:


Expert Witness Evidence

62. The Tribunal received evidence from six expert witnesses as follows:
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- Professor P, Consultant Clinical Geneticist, on behalf of the GMC;
- Professor Q, Consultant Physician specialising in Endocrinology, Diabetes and
  General Internal Medicine, on behalf of the GMC;
- Professor R, Microbiologist, on behalf of Dr Mouton;
- Dr S, specialist in Ecological Medicine, on behalf of Dr Mouton;
- Dr T, specialising in medical genotype testing, on behalf of Dr Mouton;
- Dr J, specialist in Hormone Therapy, on behalf of Dr Mouton.

63. Professor Q provided expert reports, dated 25 May 2015, 23 September 2015,
23 October 2015, 7 July 2016, 17 August 2016, 30 September 2016 and 12 July
2017, in respect of Patients A - H, and gave oral evidence at the hearing. Professor
Q’s evidence related to the care provided to Patients A - H by Dr Mouton and
whether it met the standards expected of a reasonably competent prescribing

64. Professor P provided an expert report, dated 5 July 2017, in respect of
Patients B, C, D, E, F, G and H. Professor P’s evidence related to Dr Mouton’s
undertaking of genotype testing and whether the overall standard of care provided
by Dr Mouton met the standard expected of a reasonably competent non-consultant
doctor. Professor P provided an addendum report, dated 19 October 2018.

65. Professor R provided an expert report, dated 1 November 2017, in respect of
Patients A – H. Professor R’s evidence was limited to the field of intestinal
microbiology.

66. Dr S provided an expert report, date 12 November 2017, in respect of
Patients A – H. Dr S’s evidence dealt with the care offered by Dr Mouton in the field
of functional medicine and the endocrine aspects of that care. He provided an expert
report and then addendum reports dated 6 December 2017, 16 October 2018 and
22 October 2018.

67. Dr T provided an expert report, dated 28 October 2017, in respect of Patients
A – H. Dr T was asked to provide a professional opinion on the genotype testing
undertaken by Dr Mouton.

68. Dr J provided an expert report, dated 17 November 2017, in respect of
Patients A - H. Dr J’s evidence was limited to the field of hormone therapy.

69. The Tribunal was also provided with copies of joint reports as follows:
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- Professor P and Dr T, dated 21 November 2017;
- Professor P and Dr J, dated 23 November 2017;
- Dr S and Professor Q, dated 24 November 2017;
- Professor Q and Dr J, dated 24 November 2017;
- Professor P and Dr S, November 2017.

Documentary Evidence

Submissions

70. The Tribunal bore in mind the submissions made by Mr Atherton, who replaced Mr Sephton QC, on behalf of the GMC at the submissions stage of the proceedings, and of Mr Ramasamy QC, on behalf of Dr Mouton.

71. The Tribunal had regard to the documentary evidence provided by the parties. This evidence included, but was not limited to the following:

- Dr Mouton’s medical records for Patients A – H;
- NHS records, where relevant, for Patients A - H;
- Guidelines:
  Royal College of Physicians (2011) The Diagnosis of Management of Primary Hypothyroidism
  European Thyroid Association (2013) 2013 ETA Guideline: Management of Subclinical Hypothyroidism
  ATA/AACE (2012) Clinical Practice Guidelines for Hypothyroidism in Adults: Co-sponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association (referred to as the ATA/AACE guidelines);
  Panicker (2011) Genetics of Thyroid Function and Disease;
  British Thyroid Association (2006) UK Guidelines for the use of Thyroid Function Tests;
  Okosime et al (2015) Management of Primary Hypothyroidism; statement by the British Thyroid Association Executive Committee (referred to as the Okosime paper);
  Dayan and Panicker (2018) Management of Hypothyroidism with Combination Thyroxine (T4) and Triiodothyronine (T3) hormone replacement in clinical practice: A review of suggested guidance;
The International Hormone Society’s Consensus Group of Experts on Hormone Therapies: Thyroid Hormone Therapy of Hypothyroidism (Sept 2005);

- A bundle of consent forms in respect of the genotype tests that were carried out in this case;
- A large number of scientific papers provided by Dr Mouton and the experts who gave evidence.

The Tribunal’s Approach

72. In reaching its decision on facts, the Tribunal has borne in mind that the burden of proof rests on the GMC and it is for the GMC to prove the Allegation. Dr Mouton does not need to prove anything. The standard of proof is that applicable to civil proceedings, namely the balance of probabilities, i.e. whether it is more likely than not that the events occurred.

73. The Tribunal has considered each outstanding paragraph of the Allegation separately and has evaluated the evidence in order to make its findings on the facts.

The Tribunal’s Overall Determination on the Facts

74. The Tribunal has determined the facts as follows:

**Patient A**

1. On various dates between 10 April 2013 and 18 November 2014, you consulted with Patient A and you:

   a. failed to elicit from Patient A adequate information about:

      i. possible drug misuse;

      **Found proved**

75. The Tribunal considered the email from Patient A’s father, undated, which stated:

   “With reference to [X] forthcoming appointment, just wanted to give some information ahead of time.

   He seems to be going through depression and its compounded by his sleeping pattern, also loss of weight and appetite, fatigue, aches and pains, problems with concentration, possibly poor circulation also. Perhaps some of these things are physical but having a negative effect on his psychological well-
being too. He recently admitted to having experimented with unhealthy stuff at University, not been able to ascertain quite to what extent, we decided not to assume anything and wait for confirmation through tests, before deciding the course of action to help him through this difficult phase...

76. The Tribunal also considered Dr Mouton’s response to this email dated, 8 April 2013, which states:

“I will of course take your e-mail into account without disclosing it to the patient. I never test patients for drugs without them being aware, as it is neither deontological nor legal (at least for patients older than 18, but I anyhow refuse doing that on kids as well). However, better that I know about the issue as I will try and discuss this frankly, still depending on how the consultation goes on. Thank you for informing me about your concerns.”

77. The Tribunal also considered Dr Mouton’s oral evidence:

“Some of them, where it was possible without disclosing the email and, for instance, it was easy for me to assess all his mental nervous system situation, which is the two central boxes there that we have gone through before. Of course, it was easy for me to ask him if he felt anxious, stressed, depressed, and that delivered a number of answers that were not in fact the same as what the father suggested. This is not uncommon. If you compare what a parent says about a teenager or a young adult and vice versa, what the young person says, it is typically very different, so I could not go further I felt when this young adult tells me that even though he had insomnia when he was at the university now it is okay. He acknowledged he was depressed when attending the university but now he feels he is not any more. Then, with this big gap between the two positions I felt I knew about the possible drug misuse but it was still a sort of a rumour. I had this information so I did elicit this information indirectly from Patient A, but I have not gone further because I was not in a position to suddenly out of the blue say, “Well, what about these drugs you have been taking?” I just feel that is not establishing a good relationship with the patient. He needs to have trust and not that I have been informed in secret by his father. That is how it happened.”

78. Given the email from Patient A’s father and the reference to “having experimented with unhealthy stuff at university”, and given that Dr Mouton was being consulted in the role of a General Practitioner, albeit one specialising in Functional Medicine, there was a clear duty to explore with Patient A his possible drug misuse. This is a routine enquiry often made by medical practitioners in appropriate circumstances. The Tribunal do not accept Dr Mouton’s explanation that it was inconsistent with establishing a good relationship with Patient A. The question could easily have been asked without revealing the concerns that had been raised by Patient A’s father.
79. The Tribunal therefore finds paragraph 1a(i) of the Allegation proved.

   ii. decline in academic performance;  
       **Found not proved**

80. The Tribunal considered Dr Mouton’s oral evidence in which he stated:

   “In this case it was reassuring because I knew from the father’s email that there was this concern, but I then thought that the father might have kept this worry because of what happened at uni but that the student back home was happy not to go to the uni anymore and just wanted to look at other pathways in his future life than studies, which you can accept from a young adult I feel, so he had depression, that was confirming what the father suspected but he felt he was on a different path now and this was not suggestive of depression because depression you would look into the mood; you would look into the sleep. I felt this was not sounding to me, from what the patient expressed, as depression.”

81. On review of Dr Mouton’s medical notes from his consultation with Patient A dated 10 April 2014, he identified that Patient A ‘had insomnia’, ‘can be distracted easily’ and ‘law student – has finished’. The Tribunal also noted that the email from Patient A’s father did not mention that Patient A had dropped out of university.

82. As set out at paragraphs 77 and 78 above, Dr Mouton did ascertain from Patient A that he had been at university but no longer was. In that respect he obtained from Patient A a history of what he had been doing academically and what the current position was. In the circumstances, the Tribunal concluded that Dr Mouton did not fail to elicit information about Patient A’s decline in academic performance.

83. The Tribunal therefore finds paragraph 1a(ii) of the Allegation not proved.

   iii. depressed mood;  
       **Found not proved**

84. There was a duty on Dr Mouton to elicit adequate information with regards to Patient A’s depressive mood. The Tribunal considered the email from Patient A’s father in which he stated Patient A ‘appears to be going through depression’ provided sufficient stimulus to require Dr Mouton to elicit further adequate information from Patient A in that regard.

85. In Dr Mouton’s consultation with Patient A on 10 April 2013, he documented:

   “Sleep: …had insomnia at uni’
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Memory: could be better +++
Concentration: ok (can be distracted)
Stress Control: easily”

86. The Tribunal went on to consider Dr Mouton’s medical notes regarding his consultation with Patient A, dated 18 November 2014, which state:

“sleeps lightly from 8am – mid afternoon
Depressed
Appetite halved...
But lethargic
Hard to socialise
Emotionally very flat at the moment”

87. The Tribunal therefore determined that Dr Mouton’s consultation notes from 10 April 2013 and those from 18 November 2014 demonstrate that Dr Mouton did address the issue of Patient A’s depressed mood.

88. The Tribunal therefore finds paragraph 1a(iii) of the Allegation not proved.

iv. social withdrawal;
Found not proved

89. In the consultation notes, dated 10 April 2013, Dr Mouton has recorded two asterisks next to ‘Motivation’, which he has circled. There was no reference to Patient A suffering social withdrawal. However, in the consultation notes, dated 18 November 2014, Dr Mouton does make a specific note stating ‘hard to socialise’. Furthermore, Dr Mouton has recorded that Patient A ‘plays lots of team sports/martial arts coach’. The Tribunal determined that Dr Mouton could not have elicited this information had he not adequately enquired as to Patient A’s social circumstances and social activities.

90. The Tribunal therefore finds paragraph 1a(iv) of the Allegation not proved.

v. paranoid symptoms;
Found not proved

91. The Tribunal noted the evidence that Patient A suffered a psychotic episode on 20 November 2014. However, the Tribunal further noted that Ms O, a psychologist, who Patient A had been seeing regularly, had seen no evidence of psychotic symptoms. The Tribunal considers that there is no evidence to suggest that Patient A would have exhibited such acute symptoms in any of the consultations he had with Dr Mouton and that none of the previous information available to Dr Mouton would have put him under a duty to make specific enquiries of Patient A in that regard.
92. The Tribunal therefore finds paragraph 1a(v) of the Allegation not proved.

   b. failed adequately to assess Patient A’s mental state;
      **Found proved**

93. The Tribunal went on to consider Dr Mouton’s oral evidence following questions from Mr Ramasamy, stating:

   “Q Importantly, you have asked about depression, you have got the arrow and then the two lines. Analysing that, when you speak to a patient about depression how reassuring is it to you as a clinician that the patient accepts that they have had depression? Is that reassuring compared to a patient who denies they have ever had any problem of that sort?

   A In this case it was reassuring because I knew from the father’s email that there was this concern, but I then thought that the father might have kept this worry because of what happened at uni but that the student back home was happy not to go to the uni anymore and just wanted to look at other pathways in his future life than studies, which you can accept from a young adult I feel, so he had depression, that was confirming what the father suspected but he felt he was on a different path now and this was not suggestive of depression because depression you would look into the mood; you would look into the sleep. I felt this was not sounding to me, from what the patient expressed, as depression.”

94. The Tribunal considered Dr Mouton’s oral evidence in response to Mr Sephton’s questions:

   “Q We have seen how the patient first presented to you. We have looked at your notes on page 1330. Looking at that point in time, do you think you failed to make a differential diagnosis of a psychiatric disorder at that point?

   A There was absolutely nothing showing up of any kind during our consultation, and that was true for the following consultations... and none of those four consultations would allow me to suspect any psychiatric symptom nor of attitude or what the psychiatrist Dr N has spoke (sic), and also the GP has spoke during their consultations but all that was in 2015, it came after. None of that would be noticeable.”

95. The Tribunal considered the information which has already been set out in the medical notes from Dr Mouton’s consultation with Patient A on 10 April 2013 and 18 November 2014. It noted that whilst Dr Mouton recorded Patient A’s symptoms, there is no evidence that he undertook any detailed exploration or interpretation of these symptoms. Given the email from Patient A’s father outlining his concerns and
those presenting symptoms Dr Mouton himself had identified, the Tribunal concluded there were typical signs of depression and a clear signal that further assessment of Patient A’s mental state was required.

96. Dr Mouton had a duty of care to ensure that Patient A was not at risk. The Tribunal could see no evidence that Dr Mouton enquired about Patient A’s risk of self-harm or harm to others. This is a duty which is incumbent and expected of any reasonable competent doctor when dealing with a patient who has shown signs of a depressive illness.

97. The Tribunal determined that the issues arising from the consultations with Patient A were important indicators with respect to Patient A’s mental state and that Dr Mouton failed to adequately assess them and consider if further referrals were necessary.

98. The Tribunal therefore finds paragraph 1b of the Allegation proved.

c. attributed Patient’s A’s symptoms to
   iii. obsessive compulsive disorder;  
   Found not proved

99. The Tribunal noted that it was Ms O’s conclusion upon her consultations with Patient A which led her to suggest that he presented as a ‘perfectionist with OCD’. The Tribunal could not see any evidence to suggest that Dr Mouton had attributed Patient A’s symptoms to obsessive compulsive disorder.

100. The Tribunal therefore finds Paragraph 1c(iii) of the Allegation not proved.

d. failed to make a differential diagnosis of a psychiatric disorder in light of Patient A’s presenting symptoms.  
   Found proved

101. The Tribunal was aware that Dr Mouton attributed Patient A’s symptoms to autoimmune thyroiditis. In his medical notes Dr Mouton stated:

    “This young patient suffers from an autoimmune thyroiditis expressed by increased antithyroid peroxidase (TPO) autoantibodies, which explains the thyroid weakness shown through high TSH and several typical symptoms.”

102. The Tribunal also considered the joint expert report of Dr S and Professor Q, 24 November 2017, which stated:

    “Prof Q and Dr S agreed:”
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There were no grounds for suspecting a diagnosis of schizophrenia or similar, but we both felt that Dr Mouton should have considered a possible differential diagnosis of depression.”

103. The Tribunal considered the oral evidence of Professor Q, who stated:

“The social withdrawal; that is difficult, it certainly would fit with depression. Could there be anything else going on psychiatrically? Well I suppose there could be…”

104. Having previously established that there were salient signs in Patient A’s presenting history of psychiatric problems, and elicited information from Patient A about his depressed mood, there was a duty of care for Dr Mouton to consider a psychiatric condition as part of his differential diagnosis for this patient. The Tribunal do not criticise Dr Mouton for failing to diagnose psychosis but do conclude that he should have included a psychiatric disorder as part of a differential diagnosis.

105. The Tribunal therefore finds paragraph 1d of the Allegation found proved.

Allegation 3

3. The investigations referred to in paragraph 2 were not clinically indicated.

Urinary thyroid hormones

Found Proved

106. The Tribunal first of all reminded itself of Dr Mouton’s duties under Paragraphs 11 and 16 b of Good Medical Practice (2013), which state:

“11. You must be familiar with guidelines and developments that affect your work.

16. In providing clinical care you must:

b. provide effective treatments based on the best available evidence”

107. The Tribunal went on to consider the guidance as set out by the British Thyroid Association (BTA) (July 2006), which states:

“Hypothyroidism cannot be diagnosed accurately on symptoms alone. The diagnosis of hypothyroidism requires abnormal TFT results. A TSH greater than 10 mU/L combined with a FT4 below the reference range indicates the presence of overt primary hypothyroidism in ambulant subjects.

A TSH concentration above the reference range together with FT4 within the
reference range defines subclinical (mild) hypothyroidism... Subclinical hypothyroidism requires to be confirmed 3-6 months after the initial results in order to exclude transient causes of a raised TSH... Subclinical hypothyroid patients who are TPOAb or TgAb positive are more likely to have higher serum TSH 12-15 and more likely to develop overt hypothyroidism but do not have increased mortality or increased incidence of ischaemic heart disease.”

108. The Tribunal also considered the guidelines as set out by the Royal College of Physicians (RCP) (2011), which state:

“(a) The symptoms of hypothyroidism are very common, both in many other conditions and even in states of normal health. It is therefore essential that thyroid function is tested biochemically alongside a careful clinical assessment of the individual patient. Clinical symptoms and/or signs alone are insufficient to make a diagnosis of hypothyroidism.

(b) The only validated method of testing thyroid function is on blood, which must include measurement of the levels of thyroid-stimulating hormone (TSH) and free thyroxine (FT4) in serum.

(c) There is no evidence to support the use of body fluids other than blood (eg urine, saliva) to test for thyroid function, or the measurement of basal body temperature in the diagnosis of thyroid dysfunction.”

109. The Tribunal considered the review paper ‘Management of primary hypothyroidism: statement by the British Thyroid Association Executive Committee (2015)’ (referred to as the Okosieme paper) which states:

“...The diagnosis of primary hypothyroidism is based on clinical features of hypothyroidism supported by biochemical evidence that is elevated serum TSH together with low free T4 (overt hypothyroidism) or normal free T4 (subclinical hypothyroidism). Primary hypothyroidism should not be diagnosed in individuals with normal serum TSH who otherwise have intact pituitary function...”

110. The Tribunal considered that when practising in the UK, a general practitioner must follow the guidance for that regulatory jurisdiction. It noted that the RCP guidance states that the only validated method of testing thyroid function is on blood and that there is no evidence to support the use of body fluids other than blood such as urine in this regard.

111. The Tribunal considered the expert opinions of Professor Q, Dr J and Dr S. The Tribunal were particularly mindful of the oral evidence of Professor Q when questioned by Mr Sephton:
“Q    In your joint report with Dr J you appear to accept that diagnosis by testing urinary thyroid hormones is okay; would you comment on that, please?

A    For me this touched on quite a wide range of issues, one being that I think I recognise that this particular guideline is... so it is not a world guideline (this); there is a different perspective here that it might add something else. I have reservations about that because I really think that the primary clinical decision-making looks to me to hinge upon the blood measurements and I just feel very sceptical indeed that the urine test either overrides the primary decision that has been made on the blood or really adds to it in a helpful way, so I could not see that it was a useful thing to do. I think the point here, I suppose, was that if somebody in Dr J’s practice sees him and he feels that it is helping him; clearly it is not harming the patient to do it. I would disagree with Dr J in the way that he uses the test and I do not believe that it enhances clinical decision-making, but it seems to me it is another test that can be done; so I found it difficult to categorically oppose its use, but I would see it as really having a number of problems and limitations which do not really add to the clinical decision-making.

...  

Q    The next thing I would like to ask you about is on page 5 at the top and this is you and Dr J discussing the use of urinary thyroid hormone tests; just explain your comment there, please?

A    Well, as I said before, I appreciate that tests can be done in a non-standard way and this is a good example of that. I think that the guideline recommendation from the professional body of opinion, which I think it refers to, and I do acknowledge that that is really a UK opinion and I can see that various laboratories offer urinary thyroid hormone measurements, so I do not think it is something that we would go along with because of the reservations that were previously expressed. We do not think that it adds anything to the blood measurements; it probably confuses things a bit.

Yes, I think that the private sector is somewhat different. I acknowledge that there can be a degree of greater freedom to choose tests and I think I would also acknowledge that there can be some international differences here. So I could agree with it to some extent and I do not think it is a harmful thing to do, but again I did not really see it as a relevant thing to do.”

112. The Tribunal considered that Professor Q was clear overall in his view that urinary thyroid hormone testing does not add anything to the standard blood test.
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113. The Tribunal also noted the evidence of Doctors S and J who both offered the same paper by Dr Baisier et al (2000) “Thyroid insufficiency”. “Is TSH measurement the only diagnostic tool?”, as evidence of the clinical usefulness of testing urinary thyroid hormone. Having read the full paper, the Tribunal determined there were a number of issues arising in the critical evaluation of this paper that raise doubts over its quality and subsequent applicability. As such it could not override the position of papers authored by accredited endocrinologists in the UK and international experts in the field of endocrinology.

114. The Tribunal noted that Professor Q acknowledged that it was not unsafe to perform the urinary thyroid hormone test. He also stated that many practitioners undertake other tests that are not necessarily useful and on that basis it could be done. However, the Tribunal determined that this is not the same as saying the urinary thyroid hormone test was clinically indicated. The Tribunal accepted the evidence of Professor Q as the expert in endocrinology that the urinary thyroid hormone test is not clinically helpful.

115. The Tribunal therefore finds paragraph 2a in relation to paragraph 3 of the Allegation proved.

Steroid hormones
Found Not Proved

116. The Tribunal considered Dr Mouton’s rationale for testing steroid hormones. In his witness statement he explained:

“Lower adrenal function may explain fatigue as much as a low thyroid function can do. Furthermore, mainstream medicine and functional medicine agree that we should not address lower thyroid function without bothering about adrenal function. And vice versa. If no financial restrictions apply. I will systematically assess thyroid and adrenal functions together. then address whatever findings.”

117. The Tribunal went on to consider the oral evidence of Professor Q, who stated:

“One of the reasons advanced to give Pregnenolone, as I understood it, was that Pregnenolone is a precursor of steroid hormone production, so this can be converted into all sorts of steroid hormones that you will have heard of like progesterone, oestradiol, testosterone, indeed cortisol, which is the main steroid hormone of the adrenal glands. So my question over this was really what happens there if you give Pregnenolone supplementations; how can one be sure that that is not resulting in excessive production of active steroids or, indeed, does it result in any change at all. I am just not aware of any
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evidence at all on that point; that it is a helpful thing to do to give
Pregnenolone in that situation.”

118. In their joint expert report, Professor Q (PQ) and Dr J (DJ) responded:

“PQ Yes, agree, no fundamental problem with this test: hormones may be
measured in the urines if well explained to patient and other GPs and useful
(and private sector is different from the NHS, with more freedom for tests).
There may significant uncertainty about the interpretation of low and high
levels, most endocrinologists would want to investigate further to diagnose or
exclude important differential diagnoses including Addison’s disease (an
important cause of fatigue) or Cushing’s syndrome. There are
reservations about interpretation of 17 hydroxysteroids atone.”

And

“DJ Yes agree, appropriate and useful test in my large experience (>30
years)”

119. Professor Q and Dr S agreed in their joint expert report that the investigations
were justified in the context of a full health check within Functional Medicine.

120. The Tribunal noted that Professor Q was not critical of the steroid test
undertaken by Dr Mouton. Furthermore, Dr Mouton’s approach is supported by
Doctors J and S in the context of a full health check in Functional Medicine. The
Tribunal has therefore concluded that there is no evidence put before it on behalf of
the GMC to confirm that this treatment was not clinically indicated.

121. The Tribunal therefore finds paragraph 2b in relation to paragraph 3 of the
Allegation not proved.

Insulin
Found not proved

122. The Tribunal considered Dr Mouton’s oral evidence during cross examination
by Mr Sephton, in which he stated:

“Q The next topic is insulin. Again, briefly, since it is set out in the
statement, why did you refer this patient for investigation of insulin?

A That comes from the food questionnaire. You can see that he is eating
tons of sugar, even though he is not overweight the sugar indulgence is there
and that is clearly a concern. You know the story about the students at the
uni, the quality of their diet and so on, especially as he was not doing well.
I had to find out what was his nutritional status and insulin is part of that in my opinion.”

123. The Tribunal considered the oral evidence of Professor Q in which he stated:

“I think insulin – and I cannot see it is personally necessary – actually I do not think that is a big issue.”

124. In his expert report, dated 12 November 2017, Dr S states:

“6. Insulin and HbA 1 c
At the first consultation Patient A was identified as having a poor, high-carbohydrate diet, and admitted to being a "sugar-addict". His height then was 6ft 1 ins, his weight 11 st 7lbs, giving a BMI of 21.2, which is entirely normal. His Triglyceride level was significantly elevated, and his Cholesterol was not. Appropriate dietary recommendations were made by Dr Mouton. His weight was next recorded as 11 st 9lbs in July 2014, then as 11st 12lbs in November 2014. No record of his compliance with the diet is shown, but I consider that the weight gain makes it probable that he did not comply well. Reactive hypoglycaemia (an unstable blood sugar), which is predisposed to by a high-carbohydrate diet, and treated by a low-carbohydrate one, can trigger anxiety and depression, and is a risk factor for development of Type 2 Diabetes. Given the lack of either compliance or response to it, I consider that it was reasonable to consider factors concerning blood sugar control at that stage.”

125. The Tribunal considered the opinions of both Doctors J and S who provided sufficient rationales for carrying out this test. The Tribunal determined that it did not have sufficient evidence before it to demonstrate that this test was not clinically indicated.

126. The Tribunal therefore finds paragraph 2c in relation to paragraph 3 of the Allegation not proved.

DIO2 genotype testing
Found Proved

127. The Tribunal first considered Dr Mouton’s response in his witness statement with regard to DIO2 genotype testing in relation to Patient A, he states:

“This genotype testing helps doctors deciding if some therapeutic trial with L-thyronine (T3) should be considered or not for a given patient. I underline the pioneering work performed by Professor Antonio BIANCO... In Patient A’s
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...case. homozygous wild DIO2 genotype has influenced me to favour L-thyroxine (T4).”

128. The Tribunal went on to consider Mr Ramasamy’s questions of Dr Mouton in relation to the DIO2 testing:

“Q ...we are dealing with your referral for investigation in Patient A’s case of DIO2 genotype. Obviously, there has been a run of papers that we have looked at in relation to DIO2. I am not going to go back to those now but just in outline, why did you wish this patient to be tested for that particular genotype?

A If I can take you back to the private health records [Dr Mouton refers to medical notes] TSH is above the range, at the bottom of the page. We can see anti TPO antibodies elevated, significantly not massively, and the therapeutical (sic) decision there appears on [medical note] where I prescribe 25mcgs of thyroxine to begin with, the first month, and then 50mcgs afterwards. I remind you that my treatment for Patient A, prescriptive thyroid treatment for Patient A is not disputed. Then comes the second round of testing in July 2014.”

129. The Tribunal considered the Okosieme review paper (2015) which referenced the American Thyroid Association (ATA) guidelines, stating:

“Should genetic characterization according to type 2 deiodinase gene polymorphism status be used to guide the use of combination synthetic L-T3 and LT4 therapy in hypothyroidism, in order to optimize biochemical and clinical outcomes?

Currently, genetic testing is not recommended as a guide to selecting therapy for 3 reasons. (i) Although there are data suggesting that specific polymorphisms of the type 2 deiodinase gene might be associated with therapeutic response to combination synthetic L-T3 and L-T4 therapy, controlled confirmatory studies are needed. (ii) Currently, genetic testing for these specific deiodinase polymorphisms is only available in the research setting. (iii) The small effect of the type 2 deiodinase gene variants identified so far that do affect thyroid hormone concentrations suggests that other factors (e.g. yet unidentified genetic variants) may play a far greater role in determining an individual patient’s thyroid hormone concentrations”

130. The Tribunal noted Dr Mouton’s reliance on the Panicker Paper (2009): “Common variation in the DIO2 gene predicts baseline psychological wellbeing and response to combination thyroxine plus triiodothyronine therapy in hypothyroid patients”, as scientific evidence for his use of DIO2 genotype testing. The Tribunal also considered Professor Q’s oral evidence when questioned by Mr Sephton:
“Q    Let us deal with what Dr J had to say about DI02 ...

A    ... we are not debating that T4 is turned into T3 and that T3 is the active form of the hormone responsible for, we think, all or nearly all of the biological activity of thyroid hormone. I think Dr J’s reference list is a general reference list, so I think most of it, as far as I can see, is... yes, it is quite encyclopaedic. I think a lot is largely irrelevant, although of course one accepts it is 225 references and that would be quite a lot of work to go through one by one to show that it is irrelevant.

I am not contesting the point that T4 is turned into T3; absolutely accepted. I think that it is also quite accepted that the DI02 gene is responsible for the DI02 enzyme which regulates the conversion. It may be that these common polymorphisms have some small effect on the activity of the DI02 enzyme, so that is the point. So given that the evidence is really, as far I can see, principally the research of Dr Panicker and Dr Dayan, I do not think that the evidence really suggests that DI02 genotyping is helpful in clinical practice.”

131. The Tribunal went on to consider the joint expert report of Professor P (PP) and Dr T (DT), dated 21 November 2017, which stated:

“(a) Whether the result of the test was diagnostic of some condition amenable to treatment (and if so, identify the diagnosis)

DT: None of these tests are diagnostic, but they give a genetic risk/tendency of a certain phenotype (genetic effect on the body). This can be used in a preventive setting to counteract with correct nutrition/choice of medication/lifestyle intervention. The aim of these tests is NOT to diagnose, but to identify risks and strategies.

PP: Agrees with the statement of DT. In relation to the use of DIO2 testing, significant reliance appears to be placed on the paper by Panicker et al (2009). However, the authors themselves acknowledge the marginal nature of the benefits identified by their research and suggest that current practice should not change, and further more emphasise the need for their research to be validated by other studies. DT agrees that DIO2 testing is not mainstream, but disagrees in relation to the extent that this is clinically useful.”

132. The Tribunal noted the expert report of Professor P and Doctor S with respect to DIO2 gene testing, which stated:

“3. The value and Justification of DIO2 SNiP Gene Testing

a. There is agreement on these points:
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i) The proportion of people in the general population with common polymorphisms in \textit{DIO2} is high.

ii) In general, the finding of polymorphisms in \textit{DIO2} has no diagnostic value.

b. \textit{There is disagreement concerning}:

i) The weight of evidence that \textit{DIO2} testing informs treatment practice in thyroid disease.

\textbf{PP:} With reference to paper described as 'key' in DT’s report (p.12, para 6), namely Panicker \textit{et al} (2009), PP highlights the view of the authors themselves, who stated that their findings do not justify any change in the investigation and management of thyroid disease. Furthermore, PP had the opportunity to speak to two of the authors (three of the authors of this paper are, coincidentally, professional colleagues), and their private views are exactly the same as those published. They did not, for example, state in private that they had to lower the stated significance of their findings for the sake of getting the paper past the reviewers and editors in order to be published, but rather confirmed that the statements in the paper reflect their on-going views that there is currently no place for the role of testing \textit{DIO2} in informing treatment for thyroid disease.”

133. The Tribunal went on to note the oral evidence of Professor P:

“Q Can we just look at that, please? Could you look at D7, page 60? I think that is the first page of Panicker \textit{et al}, 2009. I would like you to turn now, please, to the conclusion at page 65:

“Genetic polymorphisms in the DIO2 gene may affect psychological well-being in patients on T4 replacement and predict those who will have improved well-being in response to combination therapy with T3.”

That is supporting what Dr T was saying to a point.

“Replication of this result, including prospective studies with genotype-selected populations, are required before changes in treatment approach can be recommended in routine practice.”

Why do they say that?

A Because studies of this nature – a number of reasons. First of all, the significance value of their findings was at a very weak level; it was not a dramatic difference. Secondly, they highlighted some of the reasons in the
previous paragraph, the previous section. There are potentially other
candidate genes in addition to DIO2 and so the wider genetic environment in
which this study is taking place, if you like, and that there are other things
going on to influence psychological well-being and the principle that if this
effect, a weak effect, means anything at all, it should be seen in studies that
replicate this and are well-founded and from scientific methodology, good
methodology. I think it is earlier on in the paper that they cite work which is
not unlike this, but which is contradictory and not reaching clear-cut
conclusions. It just so happens that I am personally acquainted with three of
the authors of this paper. That is a coincidence. I have had the opportunity
to speak directly to two of them about this field and their scientific
credentials, all three of them that I know anyway, and certainly these two are
highly respected. They would not put their names to a piece of work which
was not conducted on a sound research basis. They say this work has not
been replicated. They also emphasise their belief that current practice is not
at a position where it can change, should change, at the moment and the
principle as well that if somebody is shown to be, in this case, euthyroid, or to
not have an obvious hormone abnormality, then treatment with hormones in
the face of everything apparently being normal is potentially dangerous. It
may not be dangerous, but it is certainly potentially dangerous and not part
of mainstream practice at the moment. That is the response of those
experts, whose opinion I would trust as much as anybody.”

134. The Tribunal considered Dr S’s opinion in his expert report, dated 12
November 2017, which stated:

“7. DIO2 genotype
The enzyme for which this gene codes converts the prohormone thyroxine
(known as T4) into the more active hormone liothyronine (T3). It is subject to
a reduced-function polymorphism in a minority of people. When such people
become hypothyroid, they respond less well to T4 alone than do people with
the normal (wild-type) version, and respond better to combined T4 and T3.
Dr Mouton has provided an adequate list of references on this matter. Given
that Patient A had a known autoimmune thyroiditis and had symptoms that
did not respond to normalising the T4 level, it was reasonable to investigate
whether this could be part of the clinical picture. In fact the result showed
that it could not.”

135. The Tribunal considered the reasons given by Dr Mouton and the two
references he cited (Panicker and Bianco) in his witness statement providing his
understanding of the conversion of T4 to T3. It considered that Dr Mouton is
supported by Dr S and Dr T who both rely on the Panicker paper. The Tribunal also
considered that Professor P suggested that the approach as set out in the Panicker
paper is not mainstream practice.
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136. The Tribunal noted it had also been able to read the Panicker paper (2009) in full, as a primary scientific source as opposed to a review article. The authors conclude their paper as followed:

“...prospective studies with genotype selected populations, are required before changes in treatment approach can be recommended in routine practice.”

Professor P enumerated his reasons, as set out in paragraph 71, for criticising the reliance placed by Dr Mouton on the Panicker Paper: namely, the significance value of their findings was at a very weak level, there are potentially other candidate genes in addition to DIO2 and other studies contradict their findings. The Tribunal agrees with the views expressed by the authors and Professor P, that without further scientific evidence, the clinical use of DIO2 genotype testing is not indicated in the management of patients with biochemical hypothyroid disease.

137. The Tribunal therefore finds paragraph 3 in relation to 2d of the Allegation proved.

Allegation 4

4. You prescribed dietary treatments for Patient A which were not:

a. clinically indicated;

b. supported by scientific guidelines;

c. supported by sound scientific evidence ("not evidence-based").

Found proved for iodine and selenium, otherwise found not proved

138. The Tribunal considered the joint expert report from Professor Q and Dr J, dated 24 November 2017, in which they stated:

"PQ: We both agree that these nutritional treatments can be given. We may have some disagreement about the usefulness of the treatments. For some of them-not specified – there might be a need for more studies. Dr J has experience with the majority of these treatments and does find them clinically useful and is of the opinion that there are sufficient studies for prolonged use.”

139. The Tribunal went on to consider the expert report of Dr S and Professor Q, which states:

"Prof Q and Dr S agreed:

The prescribed treatments were indicated and valid provided they were not
continued without review.

While Professor Q agreed with the scientific plausibility for benefit, he questioned the reliability of the actual evidence for long term health benefits of most of the treatments. However he acknowledged that there might be benefit and there was not likely to be harm. Therefore, in this respect the treatments could be justified.”

140. The Tribunal noted Dr S’s expert report, in which he stated:

“Nutritional status (vitamins, minerals, fatty acids)
A functional medicine approach mandates that nutritional factors are taken into account. I consider that Dr Mouton acted appropriately in running these tests... Moreover, Patient A had self-identified as a sugar addict with a consequent poor nutritional intake. This makes nutritional factors all the more relevant.

Dr Mouton also prescribed a long list of nutritional supplements for which there is no evidence of deficiency. I have reviewed the dosages... and I see no risk of overdose or adverse effect from this protocol. Given Patient A’s poor diet, multiple nutritional deficiencies were certainly possible at this point, and benefit from this prescription was probable, even in the absence of confirmatory tests. Moreover I see no risk whatsoever of harm (sic) the dosages employed. I consider Dr Mouton’s actions here to be appropriate. He also recommends diet changes to greatly reduce intake of ‘fast sugars, junk food, biscuits, cakes, fruit juices, fructose.’ This is unquestionably appropriate and indeed important.”

141. The Tribunal went on to consider the paper: Guidelines for the treatment of hyperthyroidism, prepared by the American Thyroid Association Taskforce on Thyroid Hormonal Replacement (2014).

"Dietary supplements and nutraceuticals in the treatment of hypothyroidism"

“We recommend against the use of dietary supplements, nutraceuticals, or other over-the-counter products, either in euthyroid individuals or as a means of treating hypothyroidism. We particularly caution against the use of pharmacologic doses of iodine because of the risk of thyrotoxicosis and hypothyroidism in those with intact thyroid glands susceptible to becoming further dysregulated because of underlying thyroid pathology.”

142. In the ATA/AACE guideline (2012) Paper: with regard to selenium, the authors state:
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“Selenium has notable theoretical potential for salutary effects on hypothyroidism and thyroid autoimmunity including Graves’ eye disease, both as a preventative measure and as a treatment. However, there are simply not enough outcome data to suggest a role at the present time for routine selenium use to prevent or treat hypothyroidism in any population.”

The Tribunal were mindful of the particular comments regarding selenium and the ATA’s advice against its use.

143. The Tribunal noted that in Dr Mouton’s medical note for Patient A, dated 12 December 2014, his prescription included:

“ELTB L-THYROXINE: 112 comprimes de levothyroxine (hormone thyroidienne T4) dosee a 125 mcg ; a prendre le matin a jeun I

TRCTR THYROCSIN: 60 capsules provide nutrients and herbs (including 112.5 mcg of iodine and 150 mg of ashwagandha extract) to support optimal thyroid function; THORNE RESEARCH product.

TRFBD TIRFORM : 60 gelules contiennent une plante et les cofacteurs necessaires pour transformer les hormones thyroidiennes T4 en T3; produit BIODYNAMICS.

... Comments

Given the on-going damage delivered by the autoimmune thyroid disease (AITD), the patient will benefit from an additional increase of L-THYROXINE (+ 25 mcg), especially regarding energy levels and mood.

We rely on selenium, zinc and copper (THYROCSIN 8: TIRFORM) as the converting cofactors to transform prohormones T4 into active thyroid hormones T3.”

144. The Tribunal noted Professor Q and Dr J’s opinions from their joint expert report:

“We both agree that these nutritional treatments can be given. We may have some disagreement about the usefulness of the treatments. For some of them—not specified - there might be a need for more studies. Dr J has experience with the majority of these treatments and does find them clinically useful and is of the opinion that there are sufficient studies for prolonged use.”
145. The Tribunal also noted Dr S and Professor Q’s opinions from their joint expert report:

“The prescribed treatments were indicated and valid provided they were not continued without review.

While Prof Q agreed with the scientific plausibility for benefit, he questioned the reliability of the actual evidence for long term health benefits of most of the treatments. However he acknowledged that there might be benefit and there was not likely to be harm. Therefore, in this respect the treatments could be justified.”

146. In general Professor Q accepted that many of the dietary supplements were safe and might potentially benefit patients. However the Tribunal noted that Professor Q made particular comment on substances that may affect thyroid hormone efficacy:

“Q So could you comment, therefore, because I know you have said, “Professor R said probiotics are not going to cause any harm”, but in relation to what is termed here “dietary supplements and nutraceuticals”, what is our position here, because this would suggest that we really should not be giving these things because they are not beneficial?

A I certainly agree with the comment about iodine, because iodine ingestion does potentially disturb thyroid function in the direction of overactivity or underactivity. So that is certainly something I would urge patients not to do and to stop taking the substances in those situations. I do not recommend these treatments at all to anybody. I get asked quite often, “Dr Q, do you think there are any other vitamins or minerals I can take to help the situation?” I will say, “In my opinion, no. The general view is not. Some people do decide to take them anyway. As long as you are not taking iodine, I am not sure that I see any particular hazard from it.” So I think to that extent, I kind of understood the drift of the functional medicine approach and it seemed to be relatively benign. I am against it because of this general view that it is not really appropriate in the treatment of hypothyroidism, or, of course, giving it to euthyroid individuals. I cannot see the purpose of that.”

147. In respect of iodine, Professor Q expressed a clear view that iodine is an unsafe supplement for patients with hypothyroidism. The Tribunal noted that his view is supported by the American Thyroid Association.

148. In respect of selenium, Dr Mouton stated that this was being prescribed to assist in the conversion of T4 inactive hormones to T3. This is particularly contradicted by the ATA guidelines as noted above. The Tribunal takes the view that
selenium is not clinically indicated and that it is not supported by scientific guidelines or supported by sound scientific evidence.

149. Professor R and Dr J, both of whom gave evidence that there is some benefit in the use of these dietary supplements.

150. The Tribunal determined that in respect of Thyrocsin (which contains iodine) evidence indicates potential harm and therefore its use was not clinically indicated, supported by scientific guidelines or evidence based.

151. With respect to Tirform (which contains selenium), evidence supported by the ATA (2012 guidelines) suggest there is no role at the present time for routine selenium use to prevent or treat hypothyroidism. Therefore the Tribunal determined its use was not clinically indicated, supported by scientific guidelines or evidence based.

152. The Tribunal therefore finds paragraphs 4 a, b and c of the Allegation in relation to Thyrocsin and Tirform is proved. The Tribunal finds Paragraphs 4 a, b and c of the allegation not proved in respect of the other dietary supplements.

Allegation 5

5. You failed to communicate with Patient A’s General Practitioner regarding Patient A’s:

   a. diagnosis of autoimmune thyroid disease with hypothyroidism;
      Found not proved

   b. prescriptions for thyroxine and T3 therapy.
      Found not proved

153. The Tribunal considered a letter sent to Dr Mouton on the 15 December 2014 from Dr K, which stated:

   “I believe you are looking after this 22 year old gentleman. He has recently come to our attention at the surgery following an attendance at Wimbledon Police Station where he was making a number of allegations of harassment and abuse.

   On speaking to [x] he tells me that he has been seeing you over the previous years and he has been diagnosed with a thyroid problem. I would greatly appreciate if you would send us his thyroid blood test results and inform us of what treatment he is receiving from you.”

154. Dr Mouton responded on 19 December 2014 in a letter stating:
“Thank you for writing to me regarding [x] who I first met when he was 5 years old but who I have only been treating since April 2013.

[x] came to see me at that time complaining of having had lethargy for a couple of months. The initial questionnaire showed several symptoms which could be linked to a low thyroid function, such as difficulty waking up in the morning (feeling groggy), poor memory, poor concentration, low motivation and cold extremities.

The blood and urine assessments done at that time (11th April 2013) enabled me to diagnose an autoimmune thyroid disease with Ab anti-TPO: 425 U/ml (range: <60 Neg), and hypothyroidism with a TSH at 4.55 mU/ml.

I then started a gentle thyroid hormonal supplementation based on 25 mcg daily of T4 to be increased to 50 mcg per day after one month.

I saw again [x] on November 25th this year and had a new set of tests done which showed a better T4 level at 1.33 (0.7-1.8) and a better TSH at 1.88 but both levels not yet optimal. His Ab anti TPO were increased to 566 kU/L. I therefore decided to increase his treatment to 125 mcg of levothyroxine daily. My prescription was sent to on December 15th so he should by now be taking this higher dosage.”

155. Dr K responded to Dr Mouton on 13 January 2015, stating:

“Thank you for your letter dated 19 December 2014. I wonder if you would be kind enough to give me some more detailed information.

You sent me a number of thyroid related blood tests however not all of the reference ranges were indicated and certainly it would appear that the T4 reference range is very different to what our lab uses here. I would very much appreciate if you could send me copies of the lab result with all the T4, TSH and anti-TPO levels along with their reference ranges.

My reason for requesting this is that when I saw [X] was concerned with regard to elements of his mental state and I am very keen to see all his thyroid blood tests both before and after treatment.”

156. Dr Mouton responded on 21 January 2015: stating:

“I am sorry to contradict you but I did provide the reference ranges, except maybe for TSH but they are the same everywhere in the world (eg between 0.3 and 4.5).
If you look closer, you will see that the Belgian lab, which provided [x] tests, uses the same reference ranges as the English labs but the units are different. The units used in Belgium are international.

I have copy/paste (and highlighted the ranges) here under the relevant sentences from my previous letter:

"The blood and urine assessment run at that time (11th of April 2013) enabled me to diagnose an autoimmune thyroid disease with Ab anti-TPO: 425 U/mL (range: <60 Neg), and hypothyroidism with a TSH-1 at 4.55 mU/ml."

"I saw again[x] on November the 25th and had a new set of test which show a better T4 level at 1.33 (0.7- 1.8) and a better TSH-1 at 1.88 but both levels not optimal yet. His Ab anti TPO were increased to 566 kU/L"

Besides, I join the full lab work where you can find the units.“

157. The Tribunal considered whether there was a duty on Dr Mouton to provide a diagnosis and prescription history to Patient A’s general practitioner. It referred to paragraph 44 of GMP, which states:

“44. You must contribute to the safe transfer of patients between healthcare providers and between health and social care providers. This means you must:

   a. share all relevant information with colleagues involved in your patients’ care within and outside the team”

158. The Tribunal considered the expert report from Professor Q and Dr J, which stated:

   “We both agree that Dr Mouton's communication was mainly indirect in the beginning by providing all information to the patient, who was supposed to transmit this information to the GP. Later on, he proved that he improved his communication, but, because his functional medicine is not well-known by other physicians, he would be well-advised to improve his communication further by adding more explanations and also scientific evidence.”

159. The Tribunal noted that there was no effective communication of a diagnosis or his prescribing before December 2014, until requested by Dr K on 15 December 2014. Thereafter, Dr Mouton did give the details of the prescriptions written for Patient A and therefore the Tribunal concluded that he did communicate with the GP.

160. The Tribunal therefore finds paragraphs 5a and b of the Allegation not proved.
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Allegation 6

6. In an email to Patient A’s father dated 15 December 2014, you:
   b. failed to communicate clearly with Patient A’s father, in that you provided contradictory advice regarding referring Patient A to a psychiatrist.  Found proved

161. The Tribunal noted the email from Patient A’s father to Dr Mouton sent on 15 December 2014 at 12:58. It stated:

“Dear Dr Mouton

Following your consultation with [x], you advised [x] the [x] should also be treated/seen by a psychologist/psychiatrist for issues not related directly to your treatment.

A GP saw [x] for a standard appointment which lasts 5 minutes and on [x] suggestion asked perhaps he ought to see Dr. N who does consultation locally in order to avoid [x] having to travel etc.

You are the only medical doctor treating [x] and we felt that your recommendation will be more meaningful as you have much better in-sight into the situation therefore allowing the consultant to address the issue properly instead of putting him through the process of trial and error, which can be quite conducive. We would therefore request you to prepare a short letter specifying lethargy and very low self-esteem along with the medical issues…”

162. Dr Mouton responded by email on 15 December 2014 at 13:15, stating:

“Well, in fact the situation is quite different to what you describe. I rather recommended counselling with a psychologist but not precisely a psychiatrist – two things that can are quite different.

It has been suggested that I could prescribe an anti-depressant to [x] to help him with his present situation, and I do not agree doing that by myself, because I do not believe in it and I find it dangerous.
However, I cannot be sure – and I and not qualified, let us say- that he does not need some psychiatric support in the form of psychotropic drugs. I wonder and I think he could see a psychiatrist, indeed.
However, I have not asked him to see a psychiatrist and I am not referring him to a psychiatrist, thus it would not be right to provide a referral letter. It is only because this has been raised I reacted.
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Personally, I manage him through thyroid support, food supplements, and dietary advice, all of that being stated and detailed in my report (even though it’s designed for the patient rather than a doctor).

I am rarely comfortable to see young patients seeing psychiatrists who too often react with a very heavy chemical load that I find dangerous and sometimes definitively destructive: you see the issue here!
I therefore feel that using my report as an independent source of information remains my best suggestion. I do not wish to write to a psychiatrist as it would necessarily imply suggesting some diagnosis.
If the psychiatrist could show useful for [x] it would be in my opinion by independently establishing some form of diagnosis, thus I am naming that I am against seeing one, but I did not recommend it.

Anyhow, would it be psychologist or psychiatrist, their advice will be implemented without any link to what I am doing. I very well know those professionals are not interested in functional medicine.
Or, if it was the case, it would be really exceptional and I of could of course deliver any further explanations about my treatments if they wished to understand more about it. It is very unlikely, shall I repeat.”

163. The Tribunal were mindful that Dr Mouton’s first language is not English. However, he had a duty to respond to Patient A’s father’s request and that he should do so clearly in a manner the recipient could easily understand.

164. The Tribunal noted that Dr Mouton contradicted himself stating initially that Patient A could need some psychiatric support and “could see a psychiatrist indeed”, but then states “I am rarely comfortable to see young patients seeing psychiatrists who too often react with a very heavy chemical load that I find dangerous and sometimes definitively destructive”. Dr Mouton also stated that he would not provide a letter to be used for a psychiatrist to form a basis for their diagnosis, however he would provide his independent report as a source of information but reiterated that he was not in favour of Patient A seeing a psychiatrist. The Tribunal therefore determined that it would be unclear to the reader what Dr Mouton’s position was.

165. The Tribunal therefore determined that Dr Mouton failed to communicate clearly with Patient A’s father, in that he provided contradictory advice regarding referring Patient A to a psychiatrist

166. The Tribunal therefore finds paragraph 6b of the Allegation proved.

Patient B

Allegation 8
8. The investigations referred to in paragraph 7 were:

   a. not clinically indicated;

   **Found proved in relation to CTLA-4**
   **Found not proved in relation to VDR**

**CTLA-4 genotype testing**

**Found Proved**

167. The Tribunal first considered Dr Mouton’s oral evidence following questions from Mr Ramasamy as to why CTLA-4 was clinically indicated with regard to Patient B:

   “CTLA-4 will tell us if the autoimmune thyroiditis has likely a genetic origin, so if the patient is homozygous variant, so has received let us say the wrong or the upset gene from both parents, it has to be from both parents, then this patient is prone to autoimmune thyroiditis. My algorithm is the following: if his tests come back suggesting that the autoimmune thyroiditis has a genetic route that is bad luck; it is family; both parents perhaps should not have met or whatever, the outcome you can imagine. That is a fact, we cannot change that. On the other side, if the CTLA-4 is either let us say normal, what we call the Y type or heterozygous, one good/one bad copy, well then the genes do not play into the autoimmune condition and then my reaction to that information is to put more pressure on the patient to improve the diet in order to increase intestinal health and strengthen intestinal wall because that is protective, it is a means, a very modern means, heavily scientifically documented, to help patients with autoimmune conditions, not from a mainstream point of view. From a functional medicine point of view this is powerful and we help autoimmune patients with those dietary and intestinal managements.”

168. The Tribunal considered the view of Dr S, who stated in his expert report:

   “9.4.1. CTLA-4 genotype testing

   The CTLA-4 gene codes for Cytotoxic T-lymphocyte Associated protein number 4. It is an inhibitory messenger molecule that down-regulates inflammatory responses. Alterations in this gene have been associated with insulin dependent diabetes mellitus, Graves disease, Hashimoto thyroiditis, celiac disease, systemic lupus erythematosus. thyroid-associated orbitopathy, and other autoimmune diseases. It therefore has clear relevance to this patient.”

169. The Tribunal went on to consider the expert report of Dr T, which stated:
"6.4. CTLA-4 genotype testing

This genetic variation is located within the cytotoxic T lymphocyte-associated antigen-4 gene has been the subject to more than 100 genetic studies to date (source PubMed). Jia-Jun et al (2015) demonstrated, that the genetic variation in question has a strong link to the development of Ulcerative colitis (UC) in Caucasians and Asians, but not in Africans [1]. Patients with the highest risk genotype were shown to have a 2.21 fold risk of developing UC when compared to individuals with the low risk genotype and this finding was highly significant. While the science in this aspect is very clear, this genetic variation would in my opinion not qualify for a screening approach of healthy individuals due to the fact, that a more than 2-fold higher risk of disease appears to be significant, but factoring in the fact, that the disease is relatively rare (approximately 1 in 400 individuals develop it), this still does not amount to a very high likelihood of developing the disease (2)."

170. The Tribunal noted the opinions of Professor P and Dr T in their joint expert report, which stated:

"CTLA-4 Type 1 diabetes, autoimmune endocrinopathies, rheumatoid arthritis, Hashimoto's thyroiditis and Graves' disease

DT: This was not the intended interpretation. The intended interpretation relates to the risk of developing ulcerative colitis (UC).

PP: The approximate two-fold risk for UC potentially conferred by this test does not justify the test, ie it is not indicated. DT agrees that the risk modification is very small and provides little justification for the test being performed, but adds that he would not disagree with a clinician saying he considers this genetic risk as a relevant factor he wishes to know when handling a patient."

171. The Tribunal concluded that the experts in this field are Professor P and Dr T. Their evidence was that CTLA-4 was relevant to ulcerative colitis (UC) to the limited extent that an unfavourable genotype increased the risk of disease by a factor of 2.21. Professor P stated that the increase in this risk is not sufficient to change the clinical management and that the test is not therefore clinically indicated. With regard to Patient B there were no clinical features to suggest a potential diagnosis of UC. Furthermore, the Tribunal heard Dr Mouton’s oral evidence regarding Patient B which did not include a diagnosis of UC. Taking account of the generic scientific utility of CTLA-4 testing and the specific absence of clinical need for testing in Patient B, the Tribunal concluded that CTLA-4 genotype testing was not clinically indicated."
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172. The Tribunal therefore finds paragraph 8a in relation to paragraph 7a of the Allegation proved.

VDR genotype testing
Found not Proved

173. The Tribunal first considered Dr Mouton’s oral evidence following questions from Mr Ramasamy as to why he suggested VDR testing was clinically indicated with regard to Patient B:

“This is a patient where I have identified in the first consultation an autoimmune condition, which is an autoimmune thyroiditis, so we are in this background with autoimmune issue. For autoimmune patients, and I would say only or especially, like those two genotypes, one is VDR, vitamin D receptor, because that has a clear impact as I follow the results from the genotype, it has a clear impact on how much vitamin D I am going to prescribe, keeping in mind that vitamin D – we have a lot of science demonstrating it is very helpful for autoimmune patients. I do not think that can be challenged. It is everywhere in the medical literature and I have a lot of such articles in my scientific justification bundle. It might get you through that for further patients but it is there so that is my vitamin D prescription according [to] the outcome of that genotype.”

174. The Tribunal considered the view of Dr S, who stated in his expert report:

“9.4.2. VDR genotype testing

VDR encodes the nuclear hormone receptor for vitamin D3 (the active form of vitamin D in the body). Vitamin D3 and its receptor are crucial for bone formation, but also for modulation of the immune system; Vitamin D3 is a potent anti-inflammatory agent, capable of down-regulating the inflammatory response to, for instance, autoimmune antibodies. It therefore has relevance to this patient.”

175. The Tribunal went on to consider the expert report of Dr T, which stated:

“6.5. VDR genotype testing

...As this genetic variation is so common and has such a strong effect on a severe food intolerance, this genetic variation is one of the most widely used genetic tests today.

Dr Mouton claims to have used the outcome of this genetic test to personalise the level of Vitamin D intake for the patient. The choice of genetic variations to test for as well as the approach of personalising Vitamin D intake is a
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sound and advanced approach in the field of personalised medicine. Current science strongly supports this approach.”

176. The Tribunal noted the opinions of Professor P and Dr T in their joint expert report, which stated:

“DT: The aim of this test was to determine Vitamin D-requirement (nutritional advice)... I agree with using this test for both nutritional advice and risk of osteoporosis, though I clearly state, that this test was used for NUTRITIONAL ADVICE ONLY in the case of Dr. Mouton.

PP: PP accepts that the response of DT may be correct, but would maintain that the use of the test to identify 'risk factors' is questionable in terms of health benefit, and does not currently have a place in mainstream practice (DT agrees with the latter point, as preventive and Nutrigenetics is not fully established in mainstream medicine, but the field is growing rapidly.).

... 

DT: The VDR polymorphism reduced the effectiveness of the Vitamin-D3 receptor...This genetic test is NOT a diagnosis, but gives information about the likely response to vitamin D3 supplementation.

PP: I would again defer to the expertise of an adult metabolic physician regarding the potential health benefit and intervention.”

177. The Tribunal noted the joint expert report of Professor Q and Dr J, which stated:

“We both agree that in private practise (sic), there is more freedom for different testing and that all these tests may be accurate (Professor Q has still some reservations for the VDR test, would like to have more studies in support of its usefulness). Dr J has made some research and found several double-blind placebo studies that may be considered to support the use of this genotype test for optimizing vitamin D treatment).”

178. The Tribunal determined that the evidence before it suggested that Dr Mouton was linking autoimmune hypothyroidism to vitamin D deficiency. It considered Dr S’s opinion that Vitamin D modulates autoimmune hypothyroidism and that there is a clear link between the two. The Tribunal also considered that Dr T stated in his joint expert report with Professor P that there is strong scientific support that polymorphism testing can indicate how well a patient may respond to vitamin D supplementation, albeit Professor P stated that this test is not carried out in mainstream medicine. The Tribunal therefore concluded that the GMC have not proved VDR genotype testing was not clinically indicated in this case.
179. The Tribunal therefore finds paragraph 8a in relation to paragraph 7b of the
Allegation not proved.

8. The investigations referred to in paragraph 7 were:

   b. conducted without obtaining informed consent from
      Patient B; \textbf{Found not proved}

   c. conducted without appropriate counselling of Patient B;
      \textbf{Found not proved}

180. The Tribunal saw evidence of a consent form signed by Patient B dated 30
June 2015 and on a second page, a list of the genotype tests to which Patient B was
consenting.

181. The Tribunal considered Dr Mouton’s oral evidence in relation to consent and
counselling in which he stated:

   “Q We can see that the particular ones you are asking for are crossed in
   the boxes, is that right?

   A Yes, I tick the corresponding boxes so they can ... I stress the cost
   because that is part, of course, of explaining to the patient and finding out if
   they agree for the cost, as well as for the test, and then I go through these
   different points with the patient and that is what I call my first part of
   counselling in the initial consultation.

   Q To what extent do you go into the specific tests, so taking page 2 for
   example, we can see VDR and CTLA all appear to be crossed. To what extent
   do you go into that with the patient as opposed to “I am doing genetic testing
   generally”?

   A I already announce what outcome could come from those tests in
   terms of my programme, the impact it will have on my programme because it
   is a fact that first patients are entitled to know why you test something. As
   soon as you go into the genetic field patients are always a bit more
   concerned; they want to know about what you are going to test, and it is
   a fact that they also need to be explained what could be the findings – for
   some tests it might be significant – for the majority of those tests they are
   purely benign, it is purely mean, manipulating their diet and adapting the
   dosage of vitamin D if it is about precisely the VDR, the vitamin D receptor.
   The only consequence of that is me adapting the dosage of the possible
   vitamin D supplementation, so I say that. I say, “This is a test for me
   because this might indicate that you need 1000 units more vitamin D per day
than what you would expect from the blood reading”. But then for some other tests that goes to the patient where they will have to adapt their diet according to the outcome of the test.

Q One of the points in the form, and it is just before the bullet points, is:

“You have to be aware that any genetic analysis will provide information on health status that might either reveal an increased or decreased susceptibility to develop multi-factorial diseases linked partially or entirely to hereditary factors, therefore you should address any questions related to genetic testing beforehand to your prescribing physician.”

What is that designed to encourage happening in the consultations?

A To make sure that the patients will not regret afterwards that they are aware about some result that might have an implication for their future health.

Q How do you deal with that part of the form when you are speaking to the patient prior to sending a test request off?

A I stress the fact and then you have different types of reactions, some patients feel that is okay, they prefer to know, and then some patients usually prefer not to know and then sometimes we just abandon the testing or sometimes I go a bit further through the topic with them and for many situations I can reassure them that even though we know about some genomic position I can help them compensate or combat the genetic outcome and that is precisely interesting to have a functional medicine approach into that but then the patient has to decide if he/she or wants to go for it or if he/she prefers just ignoring, and that is where it is proper consent, I mean, that is the consent process.”

182. The Tribunal noted that the VDR genotype testing was done in July 2015 and the consent form was signed on 30 June 2015. Furthermore, there is a letter from Patient B sent some time after March 2015, which stated:

“...I have now been under Dr Mouton’s care for 3 months and I am very happy with his attention and interest and thoroughness of his testing procedures. I am given clear explanations and kept informed of the plan of action. I have no reason to believe that the care that I am receiving from Dr Mouton is not of an exceptionally high standard, and this is treatment that I wish to continue.”
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183. The Tribunal was satisfied with Dr Mouton’s explanation of his consultation regarding obtaining consent for CTLA-4 and VDR genomic testing of Patient B. Dr Mouton spoke distinctly about how he went through the process and that there had been occasions where he said he would not do the test if a patient was not happy to proceed. The Tribunal noted the testimonial from Patient B supporting the care Dr Mouton had provided. This, in combination with a signed consent form, provided enough evidence to demonstrate to the Tribunal that Dr Mouton had obtained informed consent from Patient B.

184. In respect of counselling for genomic tests, the Tribunal considered that the counselling required will vary depending on the test being carried out and its potential results. The Tribunal concluded that the GMC have not provided sufficient evidence to prove the allegation, on the balance of probabilities, that there was no appropriate counselling in respect of the two particular tests that were carried out for Patient B.

185. The Tribunal therefore finds paragraph 8b and c in relation to paragraph 7a and b of the Allegation not proved.

Allegation 10

10. The investigations referred to in paragraph 9 were not clinically indicated.

   a. urinary thyroid hormones;  
      Found proved

   b. steroid hormones;  
      Found not proved

   c. insulin.  
      Found not proved

Urinary thyroid hormones  
Found proved

186. The Tribunal determined that for the reasons outlined with regard to Patient A, the investigations in respect of urinary thyroid hormone treatment were not clinically indicated.

187. The Tribunal therefore finds paragraph 10 in relation to paragraph 9a of the Allegation proved.

Steroid hormones  
Found not proved
188. The Tribunal determined that for the same reasons outlined with regard to Patient A, the investigations in respect of steroid hormones were clinically indicated.

189. The Tribunal therefore finds paragraph 10 in relation to paragraph 9b the Allegation not proved.

**Insulin Found not proved**

190. The Tribunal considered the oral evidence from Dr Mouton in which he expressed his reasoning as to why insulin testing was clinically indicated for Patient B:

   “...And he expresses some weight loss desire, that is clearly part of what he wants me to help with. Obese, this is probably on the brink of obesity, I am not a big believer in BMI so I do not find that very accurate, but we know from a clinical perspective this is a rather obese patient, certainly heavily overweight. Insulin for me is essential. I mean, how could I not test insulin for an obese patient because we need to know if he is insulin resistant or if he is even moving further into diabetes.

   Q Hence the reason for your referral under the heading “Insulin”?

   A Yes. Insulin which I also measured was looking better in fact, was not very high, not optimal, which I still quote but not very high, so insulin did not give a clear clue but HbA1c did. I would do those two tests automatically for any overweight obese poorly eating patient.”

191. The Tribunal noted the expert report from Dr S in which he stated:

   “9.4.5. Insulin

   Patient B was overweight and probably had metabolic syndrome...insulin resistance can significantly impact symptoms such as fatigue and mood changes, and therefore needs to be considered in this context. Its utility is increased when its considered alongside measures such as glucose, triglycerides and glycosylated haemoglobin. In the context of functional medicine consultation this investigation is appropriate.”

192. The Tribunal considered the joint expert report from Dr S and Professor Q, where they agreed:

   “the investigations were justified in the context of a full health check and functional medicine context.”
193. The Tribunal determined that Dr Mouton had sufficiently explained his rationale for testing insulin levels in Patient B and was supported in this approach by Dr S. The Tribunal noted the non-specific acceptance of this approach in the joint expert reports from Professor Q, Dr S, and Dr J. It also noted that the GMC had not provided any evidence to prove insulin testing should not be done. The Tribunal concluded, given the evidence before it, that insulin testing in Patient B was clinically indicated.

194. The Tribunal therefore finds paragraph 10 in relation to paragraph 9c of the Allegation not proved.

**Allegation 11**

11. You attributed Patient B’s symptoms of acute lethargy to hypothyroidism when there was no sound clinical basis for doing so.  
**Found not proved**

195. The Tribunal first considered Dr Mouton’s witness statement in which he remarked:

“There is no issue of “acute lethargy” here. My initial anamnesis states:  
“feeling very tired and lethargic --- always”... I have not labelled the patient as suffering from hypothyroidism. But I have instead commented that the patient suffered autoimmune thyroiditis. Which had “weakened the thyroid function”...”

196. The Tribunal went on to consider Dr Mouton’s oral evidence:

“A Yes, I do not know where “acute lethargy” comes from. It is not my statement.  

...  

A Yes, I also dispute that I would have labelled this patient with hypothyroidism. I did not say that. I thought that his autoimmune thyroiditis, which is mild, and that is obvious, it is not a severe case, had weakened the thyroid function which I thought was a more subtle way ...  

Again, we have this continuum, we have hypothyroidism at one end, euthyroidism at the other end. You cannot say that this patient is euthyroid but he is not hypothyroid either. He is in-between...”

197. The Tribunal considered the joint expert report from Professor Q and Dr S, which stated:
“Dr S and Professor Q's views contrasted to some extent. Dr S considered that the TSH was increased, albeit within the normal range, and therefore that there were reasonable grounds for a trial of treatment in this situation. Professor Q had reservations that such a mild borderline result would explain a non-specific symptom like lethargy, but he acknowledged that this might present an argument for a trial of treatment.”

198. The Tribunal considered the joint expert report from Professor Q and Dr J which stated:

“We both agree that lethargy is a symptom of hypothyroidism and that there is some evidence for hypothyroidism with lab tests within reference ranges (Professor Q would have had more differential diagnosis, because lethargy is so non-specific and he did not think it particularly likely that the patient was hypothyroid, although we both agreed that TSH was high in the normal range and there was a positive thyroglobulin antibody test indicating underlying autoimmune thyroid disease).”

199. The Tribunal accepted Dr Mouton’s evidence, supported by his notes, that he did not make a diagnosis of hypothyroidism, in the case of Patient B, but simply referred to “autoimmune thyroiditis” which had weakened the “thyroid function”. The Tribunal also noted that lethargy can be a symptom of hypothyroidism. The Tribunal concluded therefore that the GMC had not proved, on the balance of probabilities, that Dr Mouton had attributed Patient B’s symptoms of acute lethargy to hypothyroidism when there was no sound clinical basis for doing so.

200. The Tribunal therefore finds paragraph 11 of the Allegation not proved.

Allegation 12

12. You prescribed Patient B the treatments set out in Schedule 1, which were not:

   a. clinically indicated;
      Found proved in relation to iodine
      Found proved in relation to selenium
      Found proved in relation to Novothyral
      Found proved in relation to pregnenolone
      Otherwise Found not proved

   b. supported by scientific guidelines;
      Found proved in relation to iodine
      Found proved in relation to selenium
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Found proved in relation to Novothyral
Found proved in relation to pregnenolone

Otherwise Found not proved

c. evidence based.

Found proved in relation to iodine
Found proved in relation to selenium
Found proved in relation to Novothyral
Found proved in relation to pregnenolone

Otherwise Found not proved

Iodine
Found Proved

201. For the reasons outlined in respect of Patient A, the Tribunal found this paragraph of the Allegation proved with regard to iodine.

202. The Tribunal therefore finds paragraph 12 a, b and c of the Allegation in relation to the treatment of iodine for Patient B proved.

Selenium
Found Proved

203. For the reasons outlined in respect of Patient A, the Tribunal found this paragraph of the Allegation proved with regard to selenium.

204. The Tribunal therefore finds paragraph 12 a, b and c of the Allegation in relation to the treatment of selenium for Patient B proved.

Novothyral (100ug T4 & 20 ug T3)
Found Proved

205. The Tribunal noted that Dr Mouton stated in oral evidence that Patient B was neither euthyroid nor hypothyroid, but had mild autoimmune thyroiditis. It noted that the experts were of the view in general terms that Patient B had mildly borderline results of hypothyroidism. On 14 September 2014 Patient B was prescribed Novothyral, containing both T4 and T3.

206. The Tribunal noted the RCP guidelines (2011) with regard to the diagnosis of primary hypothyroidism, which state:

“a) Patients with suspected primary hypothyroidism should only be diagnosed with blood tests including measurement of serum TSH.”
(b) Patients with primary hypothyroidism should be treated with T4 using levothyroxine tablets (listed in the British National Formulary) alone.
(c) There is no indication for the prescription of levothyroxine or any preparation containing thyroid hormones to patients without an established diagnosis of thyroid disease and thyroid blood tests within the reference ranges.
(d) In patients with suspected primary hypothyroidism there is no indication for the prescription of levothyroxine or any preparation containing thyroid hormones to patients with thyroid blood tests initially within the normal range. Thus patients with normal levels of T4 and TSH do not have primary hypothyroidism, and even if they have symptoms which might suggest this, they should not be given thyroid hormone replacement therapy.”
(e) The RCP does not support the use of thyroid extracts or levothyroxine and T3 combinations without further validated research published in peer-reviewed journals. Therefore, the inclusion of T3 in the treatment of hypothyroidism should be reserved for use by accredited endocrinologists in individual patients.”

207. The BTA guidelines (2006) and the ATA guidelines (2012) do not recommend or support the use of combined therapy with thyroxine and tri-iodothyronine in comparison to thyroxine alone.

208. The Okosieime review (2015) cited these guidelines and went on to state:

“... 10 L-T4/L-T3 combination therapy in patients with hypothyroidism should not be used routinely, as there is insufficient evidence to show that combination therapy is superior to L-T4 monotherapy.
11 Clinicians have an ethical responsibility to adhere to the highest professional standards of good medical practice rooted in sound evidence. This includes not prescribing potentially harmful therapies without proven advantages over existing treatments.
12 If a decision is made to embark on a trial of L-T4/L-T3 combination therapy in patients who have unambiguously not benefited from L-T4, then this should be reached following an open and balanced discussion of the uncertain benefits, likely risks of over-replacement and lack of long-term safety data. Such patients should be supervised by accredited endocrinologists with documentation of agreement after fully informed and understood discussion of the risks and potential adverse consequences. Many clinicians may not agree that a trial of L-T4/LT3 combination therapy is warranted in these circumstances and their clinical judgement must be recognized as being valid given the current understanding of the science and evidence of the treatments...”
The Tribunal noted that there is clear clinical guidance that combination therapy is not clinically indicated and should not be used, unless the patient is under the care of an accredited endocrinologist.

209. The Tribunal went on to consider the joint report of Professor Q and Dr S, which stated:

“...Regarding thyroid hormone treatment, Dr S and Professor Q's opinions contrasted in that Prof. Q considered that most endocrinologists around the world would not have treated with thyroid hormone, and that is what the major guidelines suggest...”

210. The Tribunal considered the joint expert report from Professor Q and Dr J, which stated:

“...We both agree that a certain degree of hypothyroidism may exist with thyroid tests within reference range (at borderline low T3, T4, levels, high-normal TSH levels), that there is also a place for a trial of thyroid treatment of hypothyroidism with T3-T4 treatments (and not on thyroxine), and desiccated thyroid.

We also agreed that one role for thyroid guidelines is to support less experienced physicians, and that thyroid treatments not included in the guidelines may be prescribed by more experienced physicians, on conditions that safety and efficacy is adequate or can adequately be shown. We both agree that there is general scientific evidence to support this. For treatments that might be contentious or obviously falling outside a guideline, the clinician should also be able to justify this.”

211. The Tribunal is of the opinion that guidelines are put in place to protect patients from harm and to provide clinical benefit. They exist to assist healthcare professionals at all levels of training and experience, both expert and non-expert. Guidelines are not a replacement for clinical judgement and the Tribunal accepts that doctors should not 'slavishly follow them’. However, when exercising clinical judgement and derogating from a guideline, doctors have a duty of care to document and explain their judgment and reasons for departing from the guideline. This is for the benefit of patients and any other healthcare professional involved in that patient’s care.

212. The guidelines set out above are clear on the diagnostic criteria required for commencing thyroid hormone treatment. Patient B did not meet these criteria. Further, the guidelines are explicit in detailing that any combined treatment of T4 and T3 should only commence if treatment with T4 alone is unsuccessful. Professor Q confirmed this in his evidence. The Tribunal noted Patient B did not receive T4 hormonal monotherapy. Additionally, the guidelines stipulate that combination
therapy should be supervised by an accredited endocrinologist. In his evidence, Dr Mouton accepted and confirmed that he is not an accredited endocrinologist. The Tribunal did not have any evidence before it from Dr Mouton to explain his reasons for derogating from established guidance.

213. The Tribunal therefore finds paragraphs 12 a, b and c of the Allegation in relation to the treatment of Novothyral for Patient B proved.

PGNLG (pregnenolone 100mg & magnesium glycinate)
Found Proved

214. The Tribunal considered the oral evidence of Dr Mouton:

"Q Why was Pregnenolone used in this patient’s case?"

... 

A So I bring you back to my typical algorithm, fatigue, adrenal function likely has a role. I support the adrenal function with Pregnenolone, which is low in the blood. I would never prescribe Pregnenolone without a blood test. That is just not even thinkable from my point of view, so I prescribe Pregnenolone, I adapt the dosage according to the blood level and in my opinion this supports the adrenal function. May I just stress the fact that Pregnenolone in the United States is seen as a food supplement? In Europe you need a prescription and, of course, I obey the European rules, I make a prescription and that is for compounding capsules because otherwise we just do not find the Pregnenolone as such in a pharmacy, so this comes from a pharmacy. It is compounded to the strength I have chosen for my patient.”

215. The Tribunal considered the oral evidence of Professor Q:

"Q ..."

"It is plausible that Pregnenolone supplementation might be harmful."

Could you explain that, please?

A One of the reasons advanced to give Pregnenolone, as I understood it, was that Pregnenolone is a precursor of steroid hormone production, so this can be converted into all sorts of steroid hormones that you will have heard of like progesterone, oestradiol, testosterone, indeed cortisol, which is the main steroid hormone of the adrenal glands. So my question over this was really what happens there if you give Pregnenolone suppletations; how can one be sure that that is not resulting in excessive production of active steroids or, indeed, does it result in any change at all. I am just not aware of
any evidence at all on that point; that it is a helpful thing to do to give Pregnenolone in that situation.

Perhaps the other comment of reflection I have found in the course of all this, Dr J felt that Pregnenolone is a useful substance. His references relate to a slightly separate suggested use of Pregnenolone, which is in various effects on the brain. I had a lot of uncertainty over this, so (a) is there adrenal underactivity anyway; (b) is Pregnenolone an appropriate treatment – we would not generally see that it is – are we entirely satisfied that it is safe? Do not know. Is it being used for effects on the brain? Well, I do not know, so maybe some of that depends on what the patient’s expectations were, but it is not a treatment that has really gained any traction, I would say, in endocrine circles. So if we consider there is adrenal underactivity and we do not have enough cortisol or aldosterone we would replace those. I think if there is some concern about neuropsychiatric function, then maybe we could see a neurologist and I just felt I did not see the justification, but I acknowledge I am not a neurologist.”

And further:

“...My general view about pregnenolone is that this is not regarded as an appropriate treatment to support the adrenal glands in medicine and it is unclear what it does. Going back to the evidence that Dr J touched on, he believed it was useful and safe, but I just came at this rather differently and did not see any evidence base for it, so I do not really see it as an appropriate treatment at all.”

216. The Tribunal went on to consider the oral evidence of Dr J, who stated:

“A Pregnenolone is the first step of cholesterol being converted to sex hormones or other adrenal hormones like aldosterone and cortisol and others so it is almost cholesterol slightly modified.

Q Do you use it your practice?

A Most of my patients are, let us say, above aged 40 and we give them very often because it is an excellent molecule. It is very safe to improve our short-term memory loss and other complaints, attention deficit.

Q Did you have any special or specialist training in order to use pregnenolone?

A It was actually self training. If I had my book I could show that I have abundantly researched literature and explained to physicians how to do it. I lecture maybe yearly on it. At least for five years ago, it was two or three
times a year. For me it is an excellent treatment with very – generally you do not have any side effects to it...”

217. The Tribunal considered that Dr Mouton was prescribing pregnenolone in order to stimulate the adrenal glands. It noted that there was no evidence to suggest Dr Mouton had taken into consideration the neuropsychiatric effect of pregnenolone. None of the scientific papers before the Tribunal advocate pregnenolone for use in support of adrenal function.

218. The Tribunal therefore accepts the evidence of Professor Q that pregnenolone is potentially unsafe and has questionable evidence of benefit. The papers put forward by Doctors J and Mouton regarding the use of pregnenolone relate primarily to psychiatric function.

219. The Tribunal therefore determined that the use of pregnenolone to support the adrenal function in Patient B was not clinically indicated, supported by scientific guidelines or evidence based.

220. The Tribunal therefore finds paragraph 12 a, b and c of the Allegation in relation to the treatment of pregnenolone for Patient B proved.

221. The Tribunal determined that all other nutritional dietary supplements in schedule 1, for reasons already discussed in relation to Patient A, were clinically indicated, supported by scientific guidelines and evidence based and therefore were found not proved.

Allegation 13

13. You failed to communicate adequately with Patient B’s General Practitioner regarding:

   a. Patient B’s diagnoses, including:
      
      i. hypothyroidism;  
      Found proved
      
      ii. iodine deficiency; 
      Found proved
      
      iii. hypogonadism; 
      Found proved

   b. Patient B’s prescriptions as set out in Schedule 1  
   Found proved
222. The Tribunal first considered the correspondence between Dr Mouton and Dr K, Patient B’s General Practitioner. Dr K sent a letter to Dr Mouton on 30 January 2015, stating:

“I have recently taken over the care of this 52 year old gentleman whose previous practice has recently closed down.

I have gone through his past medical history with him today including his drug history. He tells me that he is taking a small dose of Thyroxine from you. I would very much appreciate if you could send me copies of his results both before and after his Thyroxine treatment.”

223. Dr Mouton responded on 16 February 2015, stating:

“I have just found your letter dated January the 30th coming back from a couple off weeks of the country.

I am seeing [Patient B] precisely this morning for his first follow-up after the treatment prescribed mid-September last year. I am planning to run more tests today to evaluate the impact of the treatment and to adapt it if needed.

I confirm that [Patient B] has been prescribed a tiny dosage of thyroid hormones, both T4 and T3 (25 mcg of T3 and 5 mcg of T4).

A more detailed report will be issued and sent to you when I will have received the new test results as it will now show more interesting to include his evolution.”

224. The Tribunal noted that Dr Mouton sent Dr K a letter dated 7 August 2015, which stated:

“I saw your patient today hence I wish to keep you informed of the patients status within my practice.

I therefore send you a copy of the results as well as the report that I have also handed over to your patient during today’s appointment.

The patient has received prescriptions and dietary recommendations for a period of 6 months and has booked a follow-up appointment in my practice in 5 months.

I am available for any further information that you may wish to receive.”

225. Dr Mouton asserted in his witness statement:
“In 2014 and early 2015, I was always providing a copy of my results to the patients and recommended them to hand it to their GP...”

The Tribunal noted the expert report of Dr S and Professor Q, which stated:

“Prof Q and Dr S agreed:

Clearer communication with rationales and treatment plans was warranted and would have prevented complaints.”

As the Tribunal had evidence before it that Patient B was between General Practices before January 2015, the Tribunal considered that Dr Mouton could not be criticised for failing to contact another surgery prior to receiving the letter from Dr K on 30 January 2015. It noted that when he received the letter from Dr K he did respond and offered an opinion, though with no clear diagnosis.

The Tribunal noted that Dr Mouton stated in his letter dated 16 February 2015, that he would send a more detailed report to Dr K once he had received the new test results. On the evidence before the Tribunal he did not do so.

The letter of 7 August 2015 was merely a proforma that enclosed the latest test results. It contained no diagnosis or explanation for the prescriptions he had made in relation to Patient B. Specifically there was no mention of any diagnosis of autoimmune thyroiditis, iodine deficiency or hypogonadism. Some of the treatments were in French. Many would have been unfamiliar to a mainstream general practitioner. The Tribunal considers that simply sending a list of his prescriptions without explanation was not adequate communication with the general practitioner.

The Tribunal therefore finds paragraphs 13a and b of the Allegation proved.

**Patient C**

**Allegation 14**

14. Between 15 December 2013 and 22 September 2015, you consulted with Patient C and you failed to record:

a. any clear diagnosis;

   **Found proved**

The Tribunal first of all considered Dr Mouton’s witness statement:

“I refer you to a letter written by the Consultant Gastroenterologist Dr U on 10 December 2013... He could not reliably decide if the patient suffers from ulcerative colitis or from Crohn’s disease (not infrequent situation). plus he
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adds “there may be a component of irritable bowel syndrome” (i.e IBS). I
have treated Patient C on the basis that she may suffer from both IBS and
IBD, would that be ulcerative colitis or Crohn’s disease.”

232. The Tribunal reviewed Dr Mouton’s patient records, test results and
correspondence for Patient C between 15 December 2013 and 22 September 2015.
A number of potential diagnoses may be inferred including UC, hair loss (attributed
to hypothyroidism), fungal dysbiosis, bacterial overgrowth, lactose intolerance and
pancreatitis.

233. The Tribunal also considered the letter sent by Dr Mouton to Dr V (Patient C’s
GP) on 12 July 2015, which stated:

“I saw your patient today hence I wish to keep you informed of the patient’s
status within my practice.

I therefore send you a copy of the results as well as the report that I have
handed over to your patient during a follow-up appointment.

The patient has received prescriptions and dietary recommendations for a
period of 6 months and has booked a follow-up appointment in my practice in
5 months...”

Dr Mouton adds a hand written note at the bottom of the letter:

“ The patient has performed a thyroid US scan on last Thursday (9 days ago)
and they had promised I would receive results yesterday but I didn’t! I will
forward them to you when I get them. The idea is to find out if she needs
thyroid hormone support or not (and I have reduced her dosage by 12.5 mcg
of l-thyroxin in the meantime).”

234. The Tribunal noted that Dr Mouton did not specify a diagnosis, but alluded to
diagnoses in his medical notes and correspondence through hand written
annotations. The Tribunal were mindful that the letter to the GP above contained no
clear diagnosis.

235. The Tribunal also noted Dr Mouton’s oral response following questions from
Mr Ramasamy:

“Q Dr Mouton, we are following your statement, and we have reached
page 14, and we are about to deal with Patient C. Patient C, the first
allegation that is being dealt with in the statement is failing to record any
clear diagnosis...?”
As it often happens with IBD, inflammatory bowel disease, there is a lot of uncertainty about deciding if a patient suffers from either ulcerative colitis or Crohn’s disease. This is one more case in that scope... this case is made even a bit more complex because there is clearly a touch of IBS with this patient, irritable bowel syndrome. It is a combination of IBS and IBD...”

The Tribunal considered the joint expert report of Professor Q and Dr J, which stated:

“We also both agree that communication to the GP should include new diagnoses.”

The Tribunal also considered the joint expert report from Dr S and Professor Q, which stated:

“We agree that it is appropriate in clinical practice to record the reasons for investigations and treatments given, and any specific diagnoses reached. This may help other doctors and the patients better understand the rationale and long term plans for treatments.”

The Tribunal considered the oral evidence of Professor Q:

“Q I would like to ask you about Patient C, page 19, please... can you just help us with what you would expect a doctor to do when faced with a patient for the first time presenting with a series of symptoms for which they were seeking treatment?

A The approach here I feel would be for any doctor to identify the presenting complaint and to try to interpret that in terms of a diagnosis, which may be possible on first encounter; on the other hand, it may require further investigation to attach a diagnosis to the presenting complaint. There may, of course, be several possible diagnoses, in which case we would also recognise the relevance of identifying a differential diagnosis, so two or more differential diagnoses.

Perhaps lastly, the other variation on the theme, so many people have multiple problems and that can turn into a problem list, but I think to summarise one would expect to see some clear identification of what the symptom was for which the patient was seeking help and, if possible, all relevant diagnoses that had been made.”

The Tribunal noted that whilst Dr Mouton suggested Patient C’s treating gastroenterologist did not assign a specific diagnosis, Dr Mouton did not demonstrate to the Tribunal that he himself had provided a clear diagnosis between 15 December 2013 and 22 September 2015.
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240. The Tribunal concluded that whilst there was evidence that Dr Mouton had documented symptoms with regard to patient C, there was no clear diagnosis. Dr Mouton’s medical notes as a whole recorded in this period, only show that Patient C had been diagnosed with ulcerative colitis by another clinician.

241. The Tribunal concluded from the expert opinions of Professor Q and Dr S that where a patient presents with numerous and potentially non-specific symptoms, there is a duty of care on doctors to provide an explicit statement of a working diagnosis in order to document and explain the subsequent treatment strategy. This is applicable to Patient C and the Tribunal found that Dr Mouton failed to provide a clear analysis or interpretation of the symptoms to formulate a diagnosis.

242. The Tribunal therefore finds paragraph 14a of the Allegation proved.

   b. any differential diagnoses.
   Found not proved

243. On review of Dr Mouton’s medical records for Patient C the Tribunal was able to identify multiple notations throughout such as fungal dysbiosis, bacterial overgrowth, low thyroid, lactose intolerance and pancreatitis. Even though these notations were scattered throughout the medical notes, the Tribunal established that taken collectively they represented a differential diagnosis for patient C. The Tribunal therefore found the Allegation that Dr Mouton failed to record any differential diagnosis not proved.

244. The Tribunal therefore finds paragraph 14b of the Allegation not proved.

Allegation 16

16. The investigations referred to in Schedule 2 were:

   a. not clinically indicated;

      DIO2 genotype testing          Found proved
      CTLA-4 genotype test           Found proved
      Apo E genotype testing         Found not proved
      MTHFR genotype testing         Found not proved
      LCT genotype testing           Found not proved
      FUT2 genotype testing          Found not proved
      VDR genotype testing           Found not proved

      DIO2 genotype testing
      Found proved
245. For the reasons outlined with regard to Patient A, the Tribunal found this paragraph of the Allegation proved.

246. The Tribunal therefore finds paragraph 16a in relation to paragraph 15 of the Allegation proved in relation to DIO2 genotype testing.

**CTLA-4 genotype testing**

**Found proved**

247. The Tribunal considered Dr Mouton’s witness statement with regards to Patient C and the treatment of CTLA-4:

“CTLA-4 gene helps me understand if a given patient has developed ulcerative colitis (or Crohn’s disease) in connection with genomic background, or if at the contrary environmental triggers, such as increased intestinal permeability linked to alcohol or gluten intake, should be blamed in priority. In this case, her heterozygous genotype does not suggest genetic factor being key component in the aetiology, thus I emphasize avoiding alcohol and zero gluten.”

248. The Tribunal considered the joint expert report of Professor P and Dr T, which stated:

“**DT**: The CTLA-4 polymorphism has a scientifically backed, but small effect on UC-Risk. Whether the risk increase of 2.21-fold is relevant in clinical management is a matter of opinion. It is not diagnostic nor an indication of disease.

**PP**: See comment under CTLA-4 above. There is no clear benefit or medical/lifestyle intervention that is a consequence of knowing the risk has approximately doubled.”

249. The Tribunal noted Dr J’s expert report in which he stated:

...clinically indicated in people with suspicion of leaky gut and autoimmune disease...”

250. The Tribunal noted the expert report of Dr T, which stated:

"...Dr Mouton justifies the test being done on an individual already affected by UC. The case can be made that in this case it is not used as a disease risk prediction in a healthy individual, but as a diagnostic tool to evaluate the factors (genetic or lifestyle) that contribute to the present condition. This is a valid argument. In my opinion the likely effect and benefit of this genetic test
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is backed by current science, but likely to be a small factor in the cause of the disease in the patient.”

251. The Tribunal noted the expert evidence of Professor P who stated that there was little clinical relevance for the use of CTLA-4 in this patient’s treatment.

252. It noted the joint expert report of Professor P and Dr T, which stated:

“[Conditions associated with CTLA-4 testing] “Type 1 diabetes, autoimmune endocrinopathies, rheumatoid arthritis, Hashimoto’s thyroiditis and Graves’ disease

DT: This was not the intended interpretation. The intended interpretation relates to the risk of developing ulcerative colitis (UC).

PP: The approximate two-fold risk for UC potentially conferred by this test does not justify the test, ie it is not indicated. DT agrees that the risk modification is very small and provides little justification for the test being performed, but adds that he would not disagree with a clinician saying he considers this genetic risk as a relevant factor he wishes to know when handling a patient.”

253. Dr Mouton’s rationale for carrying out this test was that it would help him understand if the already diagnosed UC was caused by a genetic factor or was caused by diet. Having considered all of the expert evidence, the Tribunal concluded that there was no sound basis for this approach. Professor P and Dr T both agree that the test merely indicates an increased risk by factor of 2.21 if the unfavourable genotype is present. It is not causative. The Tribunal accepted Professor P’s evidence that the increased risk does not justify the test. Accordingly the Tribunal concluded that this test was not clinically indicated for Patient C.

254. The Tribunal therefore finds paragraph 16a in relation to paragraph 15 of the Allegation proved in relation to CTLA-4 genotype testing.

Apo E genotype testing
Found not proved

255. The Tribunal considered Dr Mouton’s rationale for the Apo E genotype testing. In his witness statement he stated:

“Considering the critical importance of dietary guidance in my medical practice. I often suggest testing apoE polymorphism. While informing the patient about the consequences. Even regarding connections with AD and cardiovascular diseases. Manipulating the diet and making life-style recommendations (such as increasing physical activity seems to represent an
256. The Tribunal went on to consider Dr Mouton’s oral evidence in which he stated:

“Autoimmune patients are, from a dietary point of view, which is a bit my field, are generally driven into something you would call paleo diet. You might have heard that at some point, but let us make that clear, this is going to be a rather high fat, low carb diet. For instance, meat is going to be recommended to those patients and rather not carbs or grains, in fact less carbs and perhaps no grains. That is the typical advice, but if the patient presents an Apo E-4 allele - even one, not necessarily two; one is enough - red meat is not recommended at all, should stay away from red meat, and same thing for dairy products.

Even worse, there is a big trend now, and this is international, especially the US but now in Europe as well, and you see that everywhere is this coconut oil recommendation, which apparently is a good source of fat especially for autoimmune patients. Coconut oil is contraindicated for ApoE-4 patients. I feel I need to know for those autoimmune patients if they have an Apo E-4, because it completely changes my recommendations.”

257. The Tribunal then went on to consider the joint expert report from Professor P and Dr T, which stated:

“DT: The Apo E genetic test has a scientifically back recommendation on nutrition. The general scientific consensus of experts in the nutrigenetic field is that individuals who carry the E4-variant benefit from a low-fat high card diet whilst individual that carry the E3 variant benefit from the opposite, a high-fat, low-carb.

PP: on this point I would defer to the expertise of a metabolic physician.”

258. The Tribunal considered Dr J’s expert report which stated:

“Impairments of breakdown of triglycerides and transport of cholesterol to neurons (Apo E is the principal cholesterol carrier in the brain): increases in risks of hypertriglyceridemia hypercholesterolemia, Alzheimer’s disease, and cardiovascular disease.

Indicated as preventive screening”

259. The Tribunal noted the expert report from Dr T, which stated:
“Besides the risk of Alzheimer's disease; this genetic variation has been shown to influence how people respond to various aspects of the diet, particularly with their LDL-cholesterol levels. The general scientific consensus is, that individuals that carry the E4-variant benefit from a low-fat, high carb diet while individuals that carry the E3 variant benefit from the opposite, a high-fat, low-carb diet [10,11]. This application of genetic results of the APOE genotype has become widely used in the field of nutrigenetics, the science of eating based on one’s genetic profile. In patients who do not wish to learn of their Alzheimer's disease risk, the typical approach is to ignore the link to Alzheimer's disease and not communicate it to the patient and to focus on the nutritional intervention aspects of the genetic test only.

Dr Mouton claims to have used this genetic variation to give dietary intervention advice. This represents generally accepted nutrigenetic practice and is supported by a large body of science.”

260. The Tribunal noted the joint expert report from Professor P and Dr S, which stated:

“The Value and Justification of APOE Genotyping

a. There is agreement on these points:

i) The APOE E4/E4 genotype is associated with an increased risk of developing Alzheimer disease.

ii) APOE genotyping has weak associations with cardiovascular disease and hyperlipidaemia type III, but is not necessarily considered useful in NHS lipid clinics.

b. There is disagreement on these points:

i) Informed consent relating to testing APOE genotypes

PP: holds the view, as expressed (1.b.iii) above), that the known potential consequences of the outcome of an ApoE genotype should be explained in pre-test counselling...

DS: holds the view that APOE testing may inform lifestyle/nutrition options and this may justify testing.”

261. The Tribunal considered Dr Mouton’s rationale for carrying out this test and that it may have assisted in his dietary advice for Patient C. It also noted Dr T’s report which indicated that in the field of nutrigenetics, this test was frequently
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carried out for the reasons that are present in Patient C’s case. The Tribunal determined that the GMC have not supplied any evidence to disprove this rationale and therefore it does not consider that it has been proven that Apo E testing was not clinically indicated.

262. The Tribunal therefore finds paragraph 16a in relation to paragraph 15 of the Allegation not proved in relation to Apo E genotype testing.

MTHFR genotype testing
Found not proved

263. The Tribunal first considered Dr Mouton’s rationale for the MTHFR genotype testing. In his witness statement he stated:

“I usually do not test for this genotype (I did not for other cases) except when I face a specific situation where it shows relevant. Which was the case for Patient C. In first, her family history reveals breast cancer on her mother side. whilst her father presently suffers from prostate cancer...In second, she has been recommended to consider azathioprine to treat severe flare-ups that have occurred during the year when I did not prescribe thyroid hormonal support. These two facts have triggered a certain cancer scare among Patient C, who has asked me to further assess her risk levels through personalized tests. I have then thought about MTHFR genotype testing, especially as I had read, just a few weeks ago before meeting the patient on 1 April 2017, a new meta-analysis about MTHFR C6777T polymorphisms and breast/ovarian cancer risk. Fortunately, her MTHFR genotype is heterozygous variant (CT) and not homozygous variant (TT). I have therefore been able to reassure her about the corresponding risk. This genotype was indicated for her.”

264. The Tribunal considered the joint expert report of Professor P and Dr T, which stated:

“Breast cancer

**DT:** A large meta study has confirmed this link (19,260 cases and 26,364 controls). This was confirmed by 2 other meta studies including 446+ other publications, so the science in this area is unusually strong and well validated. I agree that this can be used to get a better insight into the risk of breast cancer, though I would extend this panel by even more genes to get a better diagnosis of risk.

**PP:** There are other risk factors for breast cancer, eg variants in *PALB2*, but even this has not entered mainstream testing practice as the specific risks and effect that such genetic status has on screening advice has not yet been fully elucidated and validated.”
265. The Tribunal then went on to consider the expert report of Dr T, which stated:

“...A large meta-study comprised of 19,260 cases and 26,364 controls has demonstrated, that this genetic variation has a moderate, but significant effect on breast cancer risk in Asians [4]. Several other meta-analyses confirmed this finding [5-7].

...As these mutations are so rare, testing is usually only warranted if several members of the close family have already had cases of breast cancer. If this is the case in patient C, BRCA1 and 2 testing would be more appropriate. Assuming that this is not the case, testing far common genetic variations that have a smaller but more common genetic risk of disease is the better-alternative. The genetic variations of this gene qualify as such and I agree with Dr Mouton's approach of testing. My only recommendation would be to extend the panel for breast cancer with a number of other genetic variations considered to be in the same as to improve the accuracy of the test.”

266. The Tribunal considered that although Professor P indicated this is not a test in mainstream practice, Dr T confirmed that there is some scientific backing for this test and given Dr Mouton’s rationale, the Tribunal concluded that in the circumstances of this case the GMC have not proved that the MTHFR genotype testing was not clinically indicated.

267. The Tribunal therefore finds paragraph 16a in relation to paragraph 15 of the Allegation not proved in relation to MTHFR genotype testing.

LCT genotype testing
Found not proved

268. The Tribunal first considered Dr Mouton’s rationale for LCT genotype testing. In his witness statement he stated:

“Genotyping LCT gene represents a useful alternative to breath tests. not only because of the lack of reliability (damaged microbiota for many different reasons, which will not allow bacteria to digest lactose and to render the test positive), but also because it shows easier and cheaper. Patient C precisely presents homozygous variant (CC) genotype and she is lactose intolerant. I did advise her accordingly and I have recommended following a lactose-free diet.”

269. The Tribunal went on to consider Dr Mouton’s oral evidence:
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“Q  One of the points that has been made is if that is known, why are you sending her off for those tests. Help the tribunal to understand that, please?

A  ...So I did consider when I ordered these tests is there any chance we can increase her dietary choices by re-introducing some dairy products beyond what she was taking and rather not taking.

I think that made sense because her breath test which had determined this lactose intolerance dated back to a number of years, so you cannot guarantee that this is still the case because a breath test evaluates in vivo situation. It is very different from a genetic test. A breath test that leads to a diagnosis of lactose intolerance has two downsides...

...it is not that accurate and that reliable. So I felt that it was worth the very small expense for this NCT genomic test to have either confirmation or information and see if I could expand the diet.”

270. The Tribunal considered the expert report of Dr T, which stated:

“Dr Mouton has used this genetic test to diagnose, that the patient is of the affected CC genotype and is therefore highly likely to suffer from lactose maldigestion. The recommendation of a lactose-free diet is scientifically correct and standard genetic practice in this case. The only additional comment that could be made during counselling is, that this food intolerance typically worsens with age; Some young individuals with the CC genotype can tolerate small to moderate amounts of lactose and younger ages but most will eventually need to switch to a lactose-free diet to avoid severe symptoms.”

271. The Tribunal considered the expert report of Dr J, which stated:

“LCT: What it detects:

Impairments in the ability to detect lactose: increases in risks of lactose and milk product digestive problems...

Clinically indicated in people with digestive problems and consuming milk products”

272. The Tribunal considered Dr Mouton’s rationale, that the LCT testing was to confirm the previous diagnosis of LCT due to the potential unreliability of a breath test which is prone to false positives and false negatives. The Tribunal therefore determined that it was appropriate for Dr Mouton to carry out this genotype testing.

273. The Tribunal therefore finds paragraph 16a in relation to paragraph 15 of the Allegation not proved in relation to LCT genotype testing.
FUT2 genotype testing
Found not proved

274. The Tribunal first considered Dr Mouton’s rationale for the FUT2 genotype test:

“Non-secreters, i.e. homozygous variant FUT2 genotypes, present numerous disadvantages, especially are more prone to ‘intestinal dysbiosis’ and to intestinal inflammation, which may predispose to the development of IBD. It is not the case for Patient C who is heterozygous variant, thus no connection but I feel this genotype was indicated for her because as a non-secretor, she would have needed a higher intake of probiotics and further survey of gut microbiota.”

275. The Tribunal went on to consider Dr Mouton’s oral evidence:

“Q In relation to the genotype testing, over the page, top of page 32, FUT2 genotype testing, again briefly?

A ...in brief, the interests of this genotype, which I really find super important and I like it very much, is that for the patients who have two "bad" variants from both father and mother, they have the complete inactivation of this gene. The FUT2 gene is just not delivering the corresponding enzyme, which is fucosyltransferase. Fucosyltransferase is an interesting word because it tells you what is going on with that enzyme. It attaches a sugar called fucose, not fructose but fucose, on the intestinal lining. This fucose layer, which needs a functional FUT2 gene and enzyme, is a bit of magic. It is pretty impressive what it does. It attracts good bacteria because it feeds them, and it repairs a lot of bad guys, bad bacteria, viruses. It is really very helpful.

Patients who are called non-secretors of the fucosyltransferase, they do not secrete fucosyltransferase because the gene is deadly wrong, are much more prone, as you will see at the top of page 32, much more prone to intestinal dysbiosis, so to an upset intestinal microbiota. They have an inflamed gut. All that is scientifically very well documented.”

276. The Tribunal considered the expert report of Dr T, which stated:

"It is my opinion that there is sufficient scientific evidence, that this genetic variation significantly influences the microorganisms in the intestine [1-3]. Whether this information is beneficial for clinical use is beyond my expertise to judge.”

277. The Tribunal considered the expert report of Dr J, which stated:
“... clinically indicated in people with digestive problems and gut dysbiosis”

278. Dr Mouton justified using this test on the basis it would give him information about Patient C that would affect the dietary recommendations he would make. Dr T confirmed that there was some scientific backing for this approach. Accordingly, in the context of the medical history presented to Dr Mouton by Patient C, the Tribunal concluded that the use of this test was clinically justified for Patient C.

279. The Tribunal therefore finds paragraph 16a in relation to paragraph 15 of the Allegation not proved in relation to FUT2 genotype testing.

**VDR genotype testing**

**Found not proved**

280. The Tribunal noted the evidence as previously set out in this determination in relation to VDR genotype testing. It considered that this type of testing can assist in making it more efficient for a doctor to understand how to manage this patient’s condition. This was particularly relevant in this case where there was documented evidence that Patient C had a low vitamin D level. The Tribunal therefore determined that VDR genotype testing was clinically indicated.

281. The Tribunal therefore finds paragraph 16a in relation to paragraph 15 of the Allegation not proved in relation to VDR genotype testing.

b. conducted without obtaining informed consent from Patient C;

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apo E genotype testing</td>
<td>Found proved</td>
</tr>
<tr>
<td>MTHFR genotype testing</td>
<td>Found proved</td>
</tr>
<tr>
<td>LCT genotype testing</td>
<td>Found not proved</td>
</tr>
<tr>
<td>FUT2 genotype testing</td>
<td>Found not proved</td>
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<tr>
<td>VDR genotype testing</td>
<td>Found not proved</td>
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<tr>
<td>DIO2 genotype testing</td>
<td>Found not proved</td>
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<tr>
<td>CTLA-4 genotype test</td>
<td>Found not proved</td>
</tr>
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282. conducted without appropriate counselling of Patient C;

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apo E genotype testing</td>
<td>Found proved</td>
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<tr>
<td>MTHFR genotype testing</td>
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<tr>
<td>FUT2 genotype testing</td>
<td>Found not proved</td>
</tr>
<tr>
<td>VDR genotype testing</td>
<td>Found not proved</td>
</tr>
</tbody>
</table>

MPT: Dr MOUTON
282. The Tribunal noted the oral evidence of Professor P, which stated:

“Q. I would like you, before we go on to deal with APOE and Alzheimer’s disease, just to explain to the Tribunal what counselling you would expect a competent practitioner to give his patient before subjecting them to a genetic test.

A. We are talking here about genetic counselling; we are not talking about therapeutic counselling. Just to make it absolutely clear, genetic counselling is something fairly specific. We have debated long and hard over the years about whether that is a good word to use for communicating genetic information. We have not over the years come up with a better term, but not to confuse it with therapeutic counselling.

Genetic counselling is around explaining the nature of the genetic condition, the risks that may apply to other family members, the risks that may apply in the future for a condition which has not yet manifested itself. The counselling in relation to genetic investigations will be first of all telling the patient exactly what tests they are having done, why those tests are being done and the indications for taking blood for that purpose and the expectations from those tests, what the tests will tell us and what the tests will not tell us and the possibility that, by doing such a test, we will find something which is extremely difficult to interpret. That last piece of counselling advice might not apply if you are doing a highly specific test on one particular mutation, but it would apply to more generic tests that are being undertaken. Those are all the things we would do prior to a test. We would sometimes say, “We think it is a good idea for you to have this test because of this, that and the other.” In other situations, we may say, “The choice is yours obviously. We cannot force anybody to have a test.” That applies to all situations, but depending on the particular medical problem, we might say, “This is going to give us good information” and steer it that way.

Then of course the test is done and you wait a few weeks for the results. We either communicate the results by letter or bring them back to clinic to talk about it. We will go through the result, what it means for the individual, whether we have found anything or not and, if we found something which is uncertain, where we might go from there and how we would handle that and of course what it might mean for other members of the family.

Q. What would your view be of somebody who told a patient, “I’m going to give you such-and-such a test” and when the test results came back, he then offered an explanation for the test and counselling?
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A  In general, that is not the way we do it in clinical genetics. Where we have time to discuss all this before, where people are not acutely ill is what I mean, then we would always aim to give an explanation beforehand and allow people the chance to know exactly what is being done. The fact that the sample of their DNA is being extracted from their blood and stored – actually, by law, we are supposed, according to the Human Tissue Act, to explain to patients that their DNA will be stored, “Is that okay?” We do not actually have to get signed consent for doing that in the UK; we are supposed to make a record of it somewhere.”

The Tribunal accepted his evidence that the process of obtaining informed consent and providing genetic counselling are inextricably linked. The Tribunal therefore considered allegation 16b and c together.

Apo E genotype testing
Found proved

283. The Tribunal first considered Dr Mouton’s response in his witness statement:

“All European genetic laboratories will always request a signed and dated consent form. Otherwise they will not undertake the tests. Furthermore, the genetic Laboratory requests consent forms signed and dated for each of the tests, which means that a patient needs to fill the consent form each time that I decide to test for any single nucleotide polymorphisms (SNPs) i.e. genomic testing.”

284. The Tribunal noted Dr Mouton’s oral evidence in which he stated:

“ApoE, I think I need a short explanation. There is a huge interest in evaluating ApoE polymorphism. Is the patient showing an ApoE-4 allele or not? That is a big question. We do not really mind ApoE-2 or ApoE-3. ApoE-4 is a big deal, has a lot of consequences. My interest is not Alzheimer's disease for such young patients. My interest is diet.”

285. The Tribunal noted the oral evidence of Professor P in relation to Apo E genetic testing and the informed consent and counselling a patient might expect:

“Q   I would like to deal finally with APOE. Could you help the Tribunal with the associations that are well-known for APOE?

A   Yes. To my knowledge, APOE and its different subtypes according to genetic variations can be associated with different types of lipid profile, different types of cardiovascular risk – most of these are very weak
associations – and also a well-known association with a risk for Alzheimer’s disease. Those are the main things according to my knowledge.

Q You have already told us that there is some association between APOE, for example, or some of the allele of APOE, and Alzheimer’s disease. That is why you were asked in the joint report of you and Dr T at page 4 what counselling ought to be given in relation to patients taking the APOE test in respect of the risk of Alzheimer’s disease. Dr T’s answer is:

“This was not the intended interpretation. The intended interpretation is nutritional advice.”

What is your view about that?

A I would say that the individual needs to have it explained what all the implications for that test are. In the same way that I would say to patients doing a microassay test that we might find something which is very difficult to interpret and only confuses and muddies the water – that is regarded as absolutely standard practice now – I would not be able to ignore the consequences of doing the test, an APOE test, which potentially confers a risk of Alzheimer’s. Even though that risk is not particularly high, it is nevertheless a piece of information that the patient is entitled to receive. Many patients will go away and read about all of this anyway. They will look it up for themselves and, if you have not told them, they may well come back to you and say, “Why haven’t you told me that?”

I have changed my practice since the internet became widely available, simply because I know that patients, or many of them, will look everything up and come back to you with a list of questions based on their own research, some of which are perfectly reasonable and others of which are a little bit misguided. The default position for me is that the patient will go away and look something up and learn for themselves.”

286. The Tribunal also noted Dr Mouton’s response to Mr Sephton’s cross examination questions with regard to this Allegation in respect of the Apo E test:

“Q You administer Apo E test to determine what dietary advice to give the patient, but some alleles of the Apo E gene have association with Alzheimer’s disease. Do you notify your patient that the test may reveal that they have an increased risk of Alzheimer’s disease?

A I have to.

Q And that the family may also have an increased risk of Alzheimer’s disease?
A The family, depending how the genes of course are transmitted and so forth. I have to because of course the test is going to come back from the genetic lab and, as you may see in all the examples that show up into the bundle that the genetic lab clearly states that in the case of Apo E4 allele, when that comes out, there is an increased risk for Alzheimer’s, so the patient is going to be exposed to the fact in a written format because I always provide a copy of that to the patient and to the GP, and I highlight these things when relevant, so I have to discuss that...

Some people may express a certain reserve and they ask me, "Yes, but if we find that, can you help me not develop Alzheimer’s disease with your functional medicine approach?" and I have to say yes. I have to say yes because if you have an Apo E4 allele, I should know and instruct you to exercise more. That is what I said to the patients. Avoid red meat, dairy products and use coconut oil to make sure you do not aggravate the inflammation, including the brain inflammation. That may start very early in your 40s or 50s so we may reduce the odds for reducing Alzheimer’s. I have never had in the last 20 years that I am doing this functional medicine full time, I have never had a patient under long-term care, which I had plenty of patients of that type in Belgium for 20 years and as such, develop Alzheimer’s.

...

Q You say, you systematically explain which tests you are going to run, their cost and the detailed reasons why you find them useful. This helps in their personalised programming.

"They may ask questions at that stage, which I will of course answer. Counselling mostly takes place when I explain the results".

...Yes, because the counselling mostly takes place - I think that has already been said - and is a matter of time. I think it is fair to agree that the discussion is more about the results than announcing what the results may lead to because otherwise that is a bit putting the pyramid on its tip.

Q I do not see in ... your witness statement, or in any paragraph on what you do with Apo E, that you had explained to the patient before they took this test that this might tell them that you have a risk of Alzheimer’s disease and your parents or your children may have such a risk?

A ...I have just further explained Apo E now because there is a big incidental risk that I have to disclose, otherwise I am in trouble when the
patient sees the results. That is very clear, very obvious to my view. I test along 20 or 30 genotypes. You only have 7 in the bundle but you know you have many different clinical situations so you have 30 genotypes on my list nowadays. It has expanded since what you see in the bundle where there was only 50.”

287. The Tribunal noted that with regard to the Apo E test, there is a signed consent form and that there was no complaint from Patient C.

288. The Tribunal noted that the Apo E test results dated 26 June 2015, for Patient C stated:

“Presence of a genetic predisposition for atherosclerosis, hypercholesterolemia and hypertriglyceridemia with regard to the analyzed polymorphisms. The relative risk of Alzheimer’s disease is increased by a factor 3 compared to negative E4 carriers. The E31E4 genotype shows the highest response rate to a diet low in calories, fatty acids and cholesterol in order to decrease the LDL cholesterol levels. Carriers of the E4 allele respond less to hypolipidemic medication (except for Probucol).”

289. The Tribunal considered the importance of obtaining informed consent and providing counselling for Patient C. It considered that obtaining informed consent would include Dr Mouton explaining the implications that test results may bring, not only for the dietary possibilities which were his focus, but also the unintended consequences in relation to Alzheimer’s disease. The Tribunal noted that there are well established links between the Apo E genotype testing and Alzheimer’s disease. This was not disputed by either party.

290. The Tribunal noted that Dr Mouton was dismissive of the significance of the Alzheimer’s Association in the test result and stated that there will be a cure by the time Patient C reaches the age where this may be of concern. He stated that he could give advice which stops his patients developing the disease.

291. The evidence demonstrated that Dr Mouton disregarded the implications of the tests in relation to Alzheimer’s disease. The Tribunal considered that if he did obtain informed consent and counsel Patient C about the full implications that may have arisen from the Apo E test, there would be a note to reflect this but the Tribunal saw no evidence of this.

292. The Tribunal heard evidence from Dr Mouton both orally and in his witness statement that his particular focus with Apo E testing was dietary. The Tribunal considered that when obtaining informed consent from a patient for a genetic test that may have significant consequences in relation to Alzheimer’s disease, if Dr Mouton’s focus was diet, there was potential for him to not fully inform a patient of all the ramifications of the results regarding potential consequences in relation to
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Alzheimer’s. There was no evidence before the tribunal that he counselled Patient C before blood was taken. This was therefore a failure in the consent process.

293. The Tribunal believed it was particularly significant that Patient C had a negative result from her Apo E test. There was no evidence to show that Dr Mouton discussed Patient C’s three-fold increased risk of Alzheimer’s disease based on her Apo E testing. The Tribunal would have expected Dr Mouton to document that some form of counselling had taken place between himself and Patient C given the implications that the result now held for herself and the members of her family.

294. The Tribunal therefore determined that on the balance of probabilities Dr Mouton did not obtain informed consent or provide appropriate counselling to Patient C in relation to the Apo E genotype testing.

295. The Tribunal therefore finds paragraphs 16b and c in relation to paragraph 15 of the Allegation proved in relation to Apo E genotype testing.

MTHFR genotype testing
Found proved

296. The Tribunal noted that the MTHFR genotype testing was undertaken by Dr Mouton after Patient C expressed concerns of developing breast cancer. Dr Mouton had not described a process of counselling and obtaining consent that included explaining to the patient that there were alternative and more widely recognised tests for assessing her risk of breast cancer. Furthermore, there is no documentary evidence he had that discussion. The Tribunal concluded therefore that he did not appropriately counsel her or obtain informed consent for this test.

297. The Tribunal therefore finds paragraphs 16b and c in relation to paragraph 15 of the Allegation proved in relation to MTHFR genotype testing.

All other Genotype testing undertaken
Found not proved

298. Dr Mouton provided the Tribunal with a description of his process in prescribing genotype tests for patients and how he obtained informed consent. Furthermore, it had copies of the consent forms from Patient C for genetic testing dated 19 November 2014, 15 June 2015, 22 February 2016, 5 October 2016 and 1 April 2017. The Tribunal determined therefore that in respect of LCT, FUT2, VDR, DIO2 and CTLA-4 genotype tests, the procedures which he explained to the Tribunal were sufficient in those cases.

299. The Tribunal therefore finds paragraphs 16b and c in relation to paragraph 15 of the Allegation not proved in relation to LCT, FUT2, VDR, DIO2 and CTLA-4 genotype testing.
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Allegation 18

18. The investigations referred to in paragraph 17 were not clinically indicated.

Investigation of urinary thyroid hormones
Found proved

Thyroid ultrasound scan
Found proved

Investigation of urinary thyroid hormones
Found proved

300. The Tribunal determined that, for the reasons as outlined with regard to Patient A, the investigation of urinary thyroid hormones was not clinically indicated.

301. The Tribunal therefore finds paragraph 18 in relation to paragraph 17a of the Allegation proved.

Thyroid ultrasound scan
Found proved

302. The Tribunal noted Dr Mouton’s witness statement response regarding the clinical indication of a thyroid ultrasound scan in relation to Patient C, he stated:

“I had suspicions about low thyroid function for a number of reasons, thus I found it logical to complete my thyroid enquiry with an US scan. This brings many advantages, such as reasonable cost, absolute safety (no radiation), quick, and comfortable to pass, but no disadvantage. It may uncover nodules or reveal criteria for inflammation and autoimmunity, especially among a patient who is already presenting an autoimmune and inflammatory condition. I also have to underline major differences in thyroid management between Belgian doctors and UK doctors. Firstly, many Belgian GP’s do not refer thyroid cases to endocrinologists. as long as they are within classic hypothyroid presentations. but they will always refer severe ones and, most often, hyperthyroid patients. Secondly, prescribing thyroid US scans for all thyroid patients are seen as good practice in Belgium, whereas not prescribing scans comes close to malpractice.”

303. The Tribunal took into the consideration the 2013 ETA Guideline: Management of Subclinical Hypothyroidism, which stated:
“A significant proportion of otherwise healthy people have asymptomatic chronic AIT...
While aspiration cytology is the most sensitive method for diagnosing AIT, a non-invasive examination by US also constitutes a reliable diagnostic tool. Nevertheless, unless there are additional clinical indications, such as goitre, US is not routinely required in the management of SCH.”

304. The Tribunal considered Professor Q’s oral evidence in which he stated:

“...I do not really see it as necessary to undertake a thyroid ultrasound. Of course I work primarily within the NHS, but I also do a small amount of private practice and actually I would not request a test and suggest the patient pays for that if it is a test that does not really inform the clinical treatment decision. So that was where I was coming from there. That is a fairly widely held view. If you do undertake thyroid ultrasounds on people who do not have a palpable goitre or a thyroid nodule, then sometimes they show abnormalities in the size of the gland and autoimmune hypothyroidism, sometimes they show abnormalities of the texture of the gland, so I suppose it can support the diagnosis that is being made on the basis of the antibodies and the thyroid function test, but it does not change it. Of course you can get unsuspected findings occasionally. You might find somebody has a lump in their thyroid that needs to be further investigated, but I think that would be a coincidental finding. So those are my personal opinions on it.

I think Dr J presented a not unreasonable argument that this is a somewhat widespread practice and I would agree that sometimes I do see patients having thyroid ultrasound scans requested which I do not think were really needed. So in my original report, as you now see, the principal question in my head was relating to the evidence base for justifying lots of tests and treatment, so in my own mind there is a degree of uncertainty over that point, so I felt it was not really a helpful thing to do. It was not an unhelpful thing to do and clearly one is within one’s rights to request the test and the patient may be interested to find out the result.”

305. The Tribunal considered the expert report from Dr S and Professor Q, which stated:

“Prof. Q and Dr S agreed:
These tests were reasonable in the context of a thyroid assessment in the private sector and could have added relevant information.”

306. The Tribunal noted Dr Mouton’s rationale for carrying out the thyroid ultrasound scan was because he had concerns about Patient C’s ongoing thyroid
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function. However, the Tribunal also noted the joint expert report of Professor Q and Dr S which stated:

“We did not consider that the patient had hypothyroidism...”

307. The Tribunal bore in mind the 2013 European guidelines as set out above which stated that this course of action is not routinely required.

308. The Tribunal noted that the scientific justification Dr Mouton cited in his evidence for undertaking the ultra sound scan: 'Review Article - Thyroid ultrasound, Indian Journal of Endocrinology and Metabolism (Mar/Apr 2013) discusses the benefit of high resolution ultrasonography and quite clearly stated:

“The limitation of thyroid imaging is that it cannot determine thyroid function; i.e., whether the thyroid gland is underactive, overactive or normal in function; for which a blood test or radioactive isotope test is generally required.”

309. The Tribunal therefore determined that in all the circumstances it did not consider that the thyroid ultrasound scan was clinically indicated.

310. The Tribunal therefore finds paragraph 18 in relation to paragraph 17b of the Allegation proved.

Allegation 19

19. You prescribed Patient C the treatments set out in Schedule 3, which were not:

a. clinically indicated;

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<td>Iodine &amp; Tyrosine</td>
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<td>Pregnenolone</td>
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<td>Selenium and selenomethionine</td>
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<td>Armour Thyroid and l-thyroxine</td>
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<td>Otherwise</td>
<td>Found not proved</td>
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b. supported by scientific guidelines;

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c. evidence based.

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<td>Otherwise</td>
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Iodine & Tyrosine

**Found proved**

311. The Tribunal noted that Professor Q and Dr S did not consider that Patient C had hypothyroidism. Nevertheless, she was given a preparation of Tyrosine which was formulated with Iodine. Therefore for the reason previously set out in this determination with regard to Patient A’s treatment this combined iodine and Tyrosine medication was not clinically indicated, supported by scientific guidelines or evidence based.

312. The Tribunal therefore finds paragraph 19 a, b and c in relation to the treatment of Iodine & Tyrosine for Patient C as set out in schedule 3 proved.

Pregnenolone

**Found proved**

313. The Tribunal determined that as Patient C did not have hypothyroidism, and for that reason and those already set out in this determination with regard to Patient B, treatment with pregnenolone was not clinically indicated, supported by scientific guidelines or evidence based.

314. The Tribunal therefore finds paragraph 19 a, b and c in relation to the treatment of pregnenolone for Patient C as set out in schedule 3 proved.

Selenium and selenomethionine

**Found proved**

315. The Tribunal determined that as Patient C did not have hypothyroidism, and for the reasons previously set out in this determination with regard to Patient A, treatment with selenium and selenomethionine (a naturally occurring amino acid which contains selenium) treatment was not clinically indicated, supported by scientific guidelines or evidence based.
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316. The Tribunal therefore finds paragraph 19 a, b and c in relation to the treatment of selenium and selenomethionine for Patient C as set out in schedule 3 proved.

Armour Thyroid and l-thyroxine

Found proved

317. The Tribunal noted the letter dated 23 April 2015 from Dr W, Consultant Physician and Endocrinologist to Dr V, in which he stated:

"I have reviewed the bloods taken by Dr Mouton and note the recent results from the clinic. I have discussed these results with [Patient C].

I am not sure that she has any significant underlying thyroid disease, particularly in the context of her negative thyroid antibodies. I realise that Dr Mouton is using thyroxine as a treatment for her colitis. I am not quite sure of the logic of adding extra thyroxine or whether this would be treatment for her hair loss. Changing thyroid hormone levels can in fact increase hair turnover and make things transiently worse.

In conclusion. I do not think that she has underlying thyroid disease at present but if the medications help her colitis then that would be Dr Mouton’s expertise rather than mine.”

318. The Tribunal went on to consider the oral evidence of Professor Q:

"Q Moving on, can we look at page 76(e):

"Overwhelming evidence supports the use of... T4... alone in the treatment of hypothyroidism, with this usually being prescribed as levothyroxine. We do not recommend the prescribing of additional... T3 in any presently available formulation, including Armour Thyroid..."

Just help us; what is "Armour Thyroid"?

A It is a brand name for a combination of T4 and T3 in roughly the sorts of proportions that you would expect in human blood levels, so it is quite widely used actually in Europe and North America.

Q Help us with what the source of the hormones is in Armour Thyroid; do you know?

A It is a dried animal extract.

Q Help us on what the guideline says next:
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"... as it [I think combination therapy] is inconsistent with normal physiology, has not been unequivocally proven to be of any benefit to patients, and may be harmful."

That may be Armour Thyroid actually?

A ...overall reservations and explains really why there is this position that the T4 alone is the standard treatment and that generally the Armour Thyroid, for example, would not be the recommended first treatment."

319. The Tribunal noted that the TSH results for Patient C were all within normal ranges. The Tribunal noted that the 2011 RCP guidelines, the 2012 ATA guidelines, the 2013 ETA guidelines and the 2015 Okosieme paper all indicate that a patient presenting as Patient C did would not be treated with Armour Thyroid or l-thyroxine. It also noted that both Professor Q and Dr S stated that they would not have prescribed Armour Thyroid treatment for Patient C.

320. The Tribunal noted that Armour Thyroid contains T3 and T4. This was started as a first line of treatment in December 2013. The guidelines indicate that T3 and T4 combination treatment should not normally be given. The evidence of Professor Q was that he would expect combination treatment only to be trialled after treatment with T4 alone has been attempted first and had been unsuccessful.

321. The Tribunal placed particular weight on the evidence of Professor Q who is the specialist endocrinologist in this case. It determined therefore that thyroid hormone treatment in this case was not clinically indicated with regard to Armour Thyroid and l-thyroxine. It followed that this treatment was neither supported by scientific guidelines nor evidence based.

322. The Tribunal therefore finds paragraphs 19 a, b and c in relation to the treatment of Armour Thyroid and l-thyroxine for Patient C as set out in schedule 3 proved.

323. The Tribunal determined that all other nutritional dietary supplements in schedule 3, for reasons already discussed in relation to Patient A, were clinically indicated, supported by scientific guidelines and evidence based and therefore were found not proved.

Allegation 20

20. You failed to communicate adequately with:

a. Patient C regarding her therapeutic treatment plan;

Found not proved
324. The Tribunal noted that Dr Mouton had an ongoing professional relationship with Patient C over a number of years. It also noted that there was no evidence of any complaint from Patient C stating that Dr Mouton failed to communicate adequately with her at any stage. Furthermore, there was no indication that she was unhappy at any stage with his communication.

325. Accordingly, the Tribunal do not consider the GMC have produced any evidence to demonstrate that Dr Mouton had not adequately communicated with Patient C.

326. The Tribunal therefore finds paragraph 20a of the Allegation not proved.

b. Patient C’s General Practitioner regarding the tests referred to in Schedule 2 and the implications of them; Found proved

327. The Tribunal noted that Dr Mouton’s communication with Dr V, dated 12 July 2015, as already set out in this determination is a demonstration of his communication. However, it noted that Dr Mouton sent results to Dr V without clear interpretation and explanation of the results or a treatment plan.

328. The Tribunal finds it proved that Patient C consulted with Dr Mouton in December 2013 and he prescribed thyroid hormone treatment to her from 8 January 2014 onwards. He prescribed a variety of treatments to her from January 2014 onwards. It noted that at that time, Dr Mouton’s system appears to have been to pass the results to the patient to pass onto the General Practitioner. The Tribunal considered that there was an obvious risk that the patient would fail to do so and that accordingly there was a danger that the General Practitioner would not be aware of important information that might affect the patient’s general management.

329. Dr Mouton began to write directly to the General Practitioner from the 12 July 2015 onwards. There was a letter that included the results and prescriptions that he had made at that time. For the reasons given in respect of Patient B, the Tribunal did not consider that the sending of a proforma letter including a large number of test results and prescriptions, some of which were in French, amounted to adequate communication.

330. The Tribunal also noted that Dr Mouton failed to copy the General Practitioner into his communication with the gastroenterologist. It took the view that this was also not adequate communication with a General Practitioner who is the central coordinator of a patient’s medical records.

331. The Tribunal therefore finds paragraph 20b of the Allegation proved.
c. Patient C’s treating Gastroenterologist regarding the intestinal and dietary treatments referred to in Schedule 3.

**Found proved**

332. The Tribunal noted Dr Mouton’s response to this Allegation:

“Patient C has always kept her Gastroenterologist Consultants fully aware about her treatment, and that clearly appears in several letters sent by them to her NHS GP. As an example, Consultant Gastroenterologist Dr X has sent a letter to the GP on 11 July 2014 that shows supportive of my treatment with probiotics and turmeric ... Initially, Patient C came to me for an alternative treatment regarding her colitis, especially as she had badly reacted to mesalazine. She had seen several different gastroenterologists in 2015, but she now trusts Dr Y who regularly oversees my programmes. I have sent him a letter on 20 December 2016 to ask him his views about resuming L-thyroxine treatment and he answered on 26 January 2017 by giving his formal acceptance.”

333. The Tribunal considered Dr Mouton’s oral evidence when questioned by Mr Sephton:

“Q I heard your explanation in chief about why you did not consult directly with Patient C’s treating gastroenterologist. Do you not think it would be common courtesy to do that?

A Certainly, yes, but first the patient was given the report and she did provide the report through the gastroenterologist. That appears in the NHS help records and the gastroenterologist she was seeing at the time was pretty supportive of my treatment. That is pages 261 and 262 in the NHS records. In the middle of the second paragraph you have this sentence:

“[The patient] had completely lost faith in mainstream medical therapy at this point.”

You see, these things happen as well and something should be done for those patients. Otherwise, what is it about?

“I was pleased to hear that the combination of interventions used by Dr Mouton appeared to make her colitis better.”

Then on page 262 there is more about that management in the second paragraph, and I fully agreed with his recommendation and I have endorsed it. He has suggested, still through the patient, there was no direct communication, I fully accept that this is not optimal and I have improved that since, he says at the end of the second paragraph, page 262”.

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**MPT: Dr MOUTON**
334. Patient C had consulted with a gastroenterologist as recently as 13 days before her first appointment with Dr Mouton in 2013. In the initial consultation Dr Mouton recorded she had a pre-existing diagnosis and accompanying treatment for ulcerative colitis. Dr Mouton then prescribed a variety of treatments to Patient C from January 2014 onwards. It was clearly important for any treating gastroenterologist to know exactly what medication Patient C was taking so they would be able to consider potential pharmacological interactions. Dr Mouton relied exclusively on Patient C to pass the results on. In fact Patient C did so as evidenced by the letter sent from Dr X to Dr V in which reference to those results are made. However, this system was far from fail-safe and created an obvious risk whereby the treating gastroenterologist would not have all the information they needed. In fact Patient C consulted with Dr Mouton regularly throughout 2014, 2015 and 2016 and received a variety of prescriptions from him. However, it was not until 2016 that Dr Mouton communicated directly with Patient C’s treating gastroenterologist. Given that he must have been aware during this period that she was being treated by a gastroenterologist there was a clear duty on him to communicate with him during this period.

335. The Tribunal therefore finds paragraph 20c of the Allegation proved.

**Patient D**

**Allegation 22**

22. The investigations referred to in paragraph 21 were not clinically indicated. *Found proved*

**Investigation of urinary thyroid hormones**

*Found proved*

336. For the reasons outlined in respect of Patient A, the Tribunal found the investigation of urinary thyroid hormones was not clinically indicated.

337. The Tribunal therefore finds paragraph 22 in relation to paragraph 21a of the Allegation proved.

**Thyroid ultrasound scan**

*Found proved*

338. For the reasons outlined in respect of Patient C, the Tribunal found the investigation of Thyroid by ultrasound scan was not clinically indicated.

339. The Tribunal therefore finds paragraph 22 in relation to paragraph 21b of the Allegation proved.
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**Allegation 23**

23. You referred Patient D for the investigation of DIO2 genotype testing and such investigation was:

a. not clinically indicated; **Found proved**

340. For the reasons outlined in respect of Patient A, the Tribunal found the investigation of DIO2 genotype testing was not clinically indicated.

341. The Tribunal therefore finds paragraph 23a of the Allegation proved.

b. conducted without obtaining informed consent from Patient D; **Found not proved**

c. conducted without appropriate counselling of Patient D; **Found not proved**

342. The Tribunal considered that Dr Mouton had given oral evidence about his general approach regarding consent and counselling for this particular test. There was also evidence of a signed consent form for this patient. Given these circumstances, and for the reasons outlined in respect of Patient C the Tribunal found that the DIO2 genotype testing was carried out with informed consent and the appropriate counselling of Patient D.

343. The Tribunal therefore finds paragraphs 23b and c of the Allegation not proved.

**Allegation 24**

24. You failed to adequately interpret or act upon the findings that Patient D was biochemically euthyroid. **Found proved**

344. The Tribunal noted Dr Mouton’s response to this Allegation in his witness statement:

“I stress that my holistic approach to thyroid patients, and especially those who suffer from autoimmune thyroid disease, should be appreciated in the context of new trends in Primary Care. Paradigm is changing slowly but surely, as shown in an article published by Elsevier in 2016 entitled “Clinical challenges in thyroid disease: Time for a new approach?”...I also underline that Patient D: a) has been showing high TSH level (6.47 at certain time #11/464); b) has been showing free T4 and free T3 blood levels close to the lower end of the ranges at multiple times and that appears in both Private
345. The Tribunal considered the letter sent from Consultant Endocrinologist, Dr Z, to Dr AA (Patient D’s General Practitioner), dated 29 April 2015, which stated:

“...We had a long discussion, and I share your concerns about a particular physician she went to see. The advice she was given is quite against what we as an endocrine society specialist group would recommend. Particularity my concern is excessive thyroid replacement and long term result of osteoporosis and atrial fibrillation. She has already had some side effects such as light periods and weight loss. In addition it will be difficult to know her true thyroid function test results as she is on combined T3 and T4 therapy. I would not recommend an ultrasound scan of her thyroid glands...”

346. The Tribunal considered the expert report of Dr J, which stated:

“In scientific controversies such as treating with thyroid therapy patients with thyroid levels within the serum reference range, it seems to me correct for evaluations of a physician’s practice to rely on scientific and experienced-based evidence and not to rely on personal opinions without practical experience.

There is here, following my experience, a fundamental misunderstanding of what biochemically euthyroid levels are.”

347. The Tribunal noted the joint expert report by Professor Q and Dr S in relation to Allegation 24, which stated:

“Neither of us would have considered a trial of thyroid hormone treatment.”

348. The Tribunal also noted the joint expert report of Professor Q and Dr J, which stated:

“We both agree that a certain degree of hypothyroidism may exist with thyroid tests within reference range (at borderline low T3, T4, levels, high-normal TSH levels), that there is a place for thyroid treatment of hypothyroidism with T3-T4 treatments (and not on thyroxine), and desiccated thyroid.

We also agree that thyroid guidelines are especially designed for low experienced or unexperienced physicians, and that thyroid treatments not included in the guidelines may be prescribed by more experienced physicians,
on conditions that safety and efficacy is adequate or can adequately be shown. We both agree that there is scientific evidence.

Dr J remarks that in patient D there is evidence of relatively higher and fluctuating TSH levels between 2.93 and 6.47, a low free T4 under the lower reference limit, and that a therapeutic trial would be acceptable. Professor Q considered that the recent TSH result of 1.47 appeared very normal, and therefore not justifying thyroid treatment in his opinion. He acknowledged that TSH had fluctuated and that there had been a higher previous result 6.47. He mentions that most physicians wouldn't have treated at those levels. Dr J points out that internationally several 1000s of physicians (not only the signers of the consensus 9 of the International Hormone society) would similarly administer a thyroid treatment trial in patients with similar thyroid tests if their clinical signs and symptoms also suggested hypothyroidism and the treatment could be beneficial to the patient's health. We discussed this case at length. Dr J considered that there was a fair case for at least a trial period of thyroid hormone treatment to determine if there was any clinical benefit, and also subject to ruling out differential diagnoses, close monitoring and avoidance of overtreatment. Dr J's rationale is set out in Consensus Statement 9 from his Society. If Dr Mouton was going to treat, then Professor Q thought a clearer explanation to the GP of why a therapeutic trial was justified would have been appropriate.”

349. The Tribunal noted that Patient D’s results were all within the reference range and therefore supported a diagnosis of being biochemically euthyroid.

350. The Tribunal noted that Dr J relied on results that were obtained in 2008 and 2010, at least 4 years before Patient D presented to Dr Mouton. The Tribunal also noted that subsequent NHS test results demonstrated that the TSH levels corrected themselves without treatment. The Tribunal therefore preferred the evidence of Professor Q and Dr S that Patient D was euthyroid and therefore should not have been started on a trial of treatment.

351. The Tribunal therefore finds paragraph 24 of the Allegation proved.

Allegation 25

25. You prescribed the treatments set out in Schedule 4 which were not:

a. clinically indicated;

    Found proved in relation to Pregnenolone
    Found proved in relation to Novothyral

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b. supported by scientific guidelines;

**Found proved in relation to Pregnenolone**
**Found proved in relation to Novothyral**

Otherwise **Found not proved**

c. evidence based;

**Found proved in relation to Pregnenolone**
**Found proved in relation to Novothyral**

Otherwise **Found not proved**

**Pregnenolone**

**Found proved**

352. For the reasons outlined with regard to Patient B, the Tribunal found that treatment with Pregnenolone was not clinically indicated, supported by scientific guidelines or evidence based.

353. The Tribunal therefore finds paragraphs 25a, b and c of the Allegation proved in relation to the prescription of pregnenolone treatment to Patient D.

**Novothyral**

**Found proved**

354. For the reasons outlined with regard to Patient B, and given the Tribunal’s finding that Patient D was euthyroid, The Tribunal found that treatment with Novothyral was not clinically indicated, supported by scientific guidelines or evidence based.

355. The Tribunal therefore finds paragraphs 25a, b and c of the Allegation proved in relation to the prescription of Novothyral treatment to Patient D.

356. The Tribunal determined that all other nutritional dietary supplements in Schedule 4, for reasons already discussed in relation to Patient A, were clinically indicated, supported by scientific guidelines and evidence based and therefore were found not proved.

**Patient E**

**Allegation 27**
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27. The investigations referred to in paragraph 26 were not clinically indicated. **Found proved**

Investigation of urinary thyroid hormones
**Found proved**

357. For the reasons outlined with regard to Patient A, the Tribunal found investigation of urinary thyroid hormones was not clinically indicated.

358. The Tribunal therefore finds paragraph 27 in relation to paragraph 26a of the Allegation proved.

Thyroid ultrasound scan
**Found proved**

359. For the reasons outlined with regard to Patient C, the Tribunal found the investigation of the thyroid by ultrasound scan was not clinically indicated.

360. The Tribunal therefore finds paragraph 27 in relation to paragraph 26b of the Allegation proved.

**Allegation 29**

29. The investigations referred to in paragraph 28 were:
   a. not clinically indicated;
      **Found proved in relation to DIO2 genotype testing**
      **Found not proved in relation to Apo E genotype testing**
      **Found proved in relation to CTLA-4 genotype testing**

DIO2 genotype testing
**Found proved**

361. For the reasons outlined with regard to Patient A, the Tribunal found the investigation of DIO2 genotype testing was not clinically indicated.

362. The Tribunal therefore finds paragraph 29a in relation to paragraph 28a of the Allegation proved with regard to DIO2 genotype testing.

Apo E genotype testing
**Found not proved**

363. For the reasons outlined with regard to Patient C, the Tribunal found there was insufficient evidence provided by the GMC to establish that the investigation of Apo E genotype testing was not clinically indicated.
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364. The Tribunal therefore finds paragraph 29a in relation to paragraph 28b of the Allegation not proved with regards to the clinical indication of Apo E genotype testing.

CTLA-4 genotype testing
Found proved

365. The Tribunal reminded itself of the opinion of the experts in this field Professor P and Dr T. Their evidence stated that CTLA-4 was relevant to ulcerative colitis (UC) to the limited extent that an unfavourable genotype increased the risk of disease by a factor of 2.21. The Tribunal further reminded itself that Professor P stated the increase in this risk is not sufficient to change the clinical management. Patient E had a pre-existing diagnosis of Crohn’s disease. Taking account of the generic clinical utility of CTLA-4 genotype testing and Patient E’s clinical history the Tribunal concluded that this test was not clinically indicated.

366. The Tribunal therefore finds paragraph 29a in relation to paragraph 28c of the Allegation proved with regards to the clinical indication of CTLA-4 genotype testing.

b. conducted without obtaining informed consent from Patient E;  
   Found not proved in relation to DIO2 genotype testing  
   Found proved in relation to Apo E genotype testing  
   Found not proved in relation to CTLA-4 genotype testing

c. conducted without appropriate counselling of Patient E;  
   Found not proved in relation to DIO2 genotype testing  
   Found proved in relation to Apo E genotype testing  
   Found not proved in relation to CTLA-4 genotype testing

DIO2 genotype testing
Found not proved

367. The Tribunal considered that Dr Mouton had given oral evidence about his general approach regarding consent and counselling for this particular test. There was also evidence of a signed consent form for this patient. Given these circumstances, and for the reasons outlined in respect of Patient C the Tribunal found that the DIO2 genotype testing was carried out with the informed consent and appropriate counselling of Patient E.

368. The Tribunal therefore finds paragraph 29b and c in relation to paragraph 28a of the Allegation not proved with regard to obtaining informed consent and counselling of Patient E for DIO2 genotype testing.

Apo E genotype testing
Found proved
369. The Tribunal noted that there is a signed consent form for this test. However Patient E was 32 years old when she first consulted Dr Mouton. The Tribunal also noted that the result of her Apo E test showed an increased risk of developing Alzheimer’s disease. Given these circumstances, and for the reasons outlined in regard to Patient C, the Tribunal found that Dr Mouton should have made explicit reference to this during his process of consent and counselling of Patient E. The Tribunal determined that Dr Mouton had conducted this test without obtaining informed consent or undertaking appropriate counselling of Patient E.

370. The Tribunal therefore finds paragraph 29b and c in relation to paragraph 28b of the Allegation proved in that Dr Mouton did not obtain informed consent or appropriately counsel Patient E prior to conducting Apo E genotype testing.

**CTLA-4 genotype testing**

**Found not proved**

371. The Tribunal considered that Dr Mouton had given oral evidence about his general approach regarding consent and counselling for this particular test. There was also evidence of a signed consent form for this patient. Given these circumstances, and for the reasons outlined in respect of Patient C the Tribunal found that the CTLA-4 genotype testing was carried out with the informed consent and appropriate counselling of Patient E.

372. The Tribunal therefore finds paragraph 29b and c in relation to paragraph 28c of the Allegation not proved with regard to obtaining informed consent and counselling of Patient E for CTLA-4 genotype testing.

**Allegation 30**

30. You failed to adequately to interpret or act upon the findings that Patient E was biochemically euthyroid.

**Found proved**

373. The Tribunal first considered Dr Mouton’s witness statement with regard to this Allegation:

“In first, I stress the fact that I did not prescribe thyroid hormones in my first programme dated 4 September 2015 (sic) which shows that I did consider her as biochemically euthyroid at that time. However, despite having prescribed specific food supplements, non-prescriptive thyroid glandular GTA, and adrenal support made on the back of pregnenolone, the patient did show low blood T3 level when she came back in January 2015. Corresponding results have been provided to the GMC but they do not appear in the hearing bundle. Blood free T3 was 3.05 pmoI/L. whereas range goes from 3.23 to 6.47
pmoI/L. She was then not biochemically euthyroid. Given the appearance of new symptoms such as irregular periods and constipation, I opted for a therapeutical trial with a combination of T4 and T3. This decision was besides supported by heterozygous variant DIO2 genotype, which has likely contributed to abnormally low blood free T3 level. I also relied on scientific articles already provided regarding Patient C, which relate to "the association of inflammatory Bowel Diseases with thyroid disorders"…"

374. Whilst the Tribunal acknowledged that Dr Mouton stated he did not prescribe thyroid hormone treatment after the first consultation, it noted that he did prescribe GTA which contains both T3 and T4.

375. The Tribunal noted the expert report from Dr S, which stated:

“18.6. Euthyroid diagnosis
Dr Mouton wrote to the patient after the initial consultation: “Low endocrine functions, both thyroid and adrenal glands considerably weaken intestinal immune defences, thus we implement an individualised glandular support treatment strengthened by numerous cofactors (often severely deficient due to the leaky gut)”

From what I have seen of Dr Mouton’s work and notes I believe that if he had thought the patient had actual hypothyroidism, he would have stated it more clearly than this. He would also have been swift to prescribe hormone replacement; he did not. He prescribed “glandular support treatment” – a mixture of nutritional supplements (trace minerals including iodine) and herbal extracts, all at low doses. Plus a porcine Glandular Concentrate at a very low dose. These are the “numerous cofactors” to which he refers. nutritional supplements and indeed herbal treatments at such low doses are very different from hormone replacement. I therefore consider that at the initial consultation Dr Mouton did not fail to “adequately interpret or act upon the findings that Patient E was biochemically euthyroid.”
It was only at 4-month follow up assessment that Dr Mouton wrote: “The severe lack of active thyroid hormones T3 leads to plenty of issues…” and he prescribes T4 25 mcgs and T3 5 mcgs in addition to the existing “glandular support”.

He appears to have based his assessment on 3 direct laboratory findings: low blood Free T3, low 24 hour urinary T3 and borderline low 24 hour urinary T4, on 2 indirect laboratory findings; elevated Cholesterol and elevated Carotene to Retinol ratio, and on 2 new symptoms: constipation and “periods haywire”. I find this very much a borderline case. On the one hand I would not myself have regarded this assessment as sufficient justification for prescribing thyroid hormones, at however small a dose. On the other hand it is clear that Dr Mouton believed at that point that he was following paragraph 16.a of
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Good Medical practice: "In providing clinical care you must a. prescribe drugs or treatment, including repeat prescriptions. only when you have adequate knowledge of the patient's health and are satisfied that the drugs or treatment serve the patient's needs". Therefore I do not consider that in this instance Dr Mouton failed to adequately interpret or act upon the findings that Patient E was biochemically euthyroid”

376. The Tribunal noted the joint expert report from Professor Q and Dr S, which stated:

“Dr S and Professor Q agreed that this patient was biochemically euthyroid, and therefore did not have hypothyroidism or warrant treatment with thyroid hormone.

However, Dr S considered that Dr Mouton's treatment with glandular support was clearly justified, and he also considered that the thyroid hormone doses were small and unlikely to cause harm.”

377. The Tribunal also noted the joint expert report from Professor Q and Dr J, which stated:

“We both agree that a certain degree of hypothyroidism may exist with thyroid tests within reference range (at borderline low T3, T4, levels, high-normal TSH levels), that there is a place for thyroid treatment of hypothyroidism with T3-T4 treatments (and not on thyroxine), and desiccated thyroid.

We also agree that thyroid guidelines are partly designed for less experienced, and that thyroid treatments not included in the guidelines may be prescribed by more experienced physicians, on conditions that safety and efficacy is adequate or can adequately be shown. We both agree that there is scientific evidence.

However, Professor Q considered this patient was clearly euthyroid based on the normal blood results, and he would not have diagnosed hypothyroidism or prescribed thyroid hormone. He also considered that most other doctors, whatever their level of experience in endocrinology, would not have treated for hypothyroidism. Dr J considered there was sufficient evidence to justify a trial of treatment, as justified previously and set out in his Society's Consensus Statement 9. Dr J points out that internationally several 1000s of physicians (not only the signers of the consensus 9 of the International Hormone society) would similarly administer a thyroid treatment trial in patients with similar thyroid tests if their clinical signs and symptoms also suggested hypothyroidism and the treatment could be beneficial to the patient's health.”
The Tribunal noted that Professor Q and Dr S agreed that Patient E was biochemically euthyroid and therefore did not have hypothyroidism or warrant treatment for hypothyroidism.

The Tribunal reminded themselves of Dr Mouton’s oral evidence where he stressed he did not prescribe thyroid hormones in his first programme dated 27 September 2014. In January 2015, Dr Mouton prescribed combination therapy in the form of Novothyral on the basis of the new test results. Those test results from January 2015 showed the serum free T3 level to be low and outside of the normal range. The urine T3 levels were also low and outside of the normal range. Dr Mouton interpreted those results as “a severe lack of active thyroid hormones” and implemented stronger thyroid treatment, based on slowly increasing dosages of Novothyral. However, the Tribunal had already determined that the urine results did not provide a sound basis for treatment. The serum TSH and T4 results were both well within the reference range. The Tribunal determined that there was no basis for concluding that Patient E was not biochemically euthyroid. Accordingly, by prescribing thyroid hormones, particularly combination T3 and T4 in the form of Novothyral, Dr Mouton did fail to adequately interpret or act upon the finding that Patient E was biochemically euthyroid.

The Tribunal therefore finds paragraph 30 of the Allegation proved.

Allegation 31

31. You wrongly diagnosed Patient E with adrenal insufficiency; Found not proved

The Tribunal first considered Dr Mouton’s response to this Allegation in his witness statement:

“I still state that Patient E’s adrenal function was “low”, but I never pretended she was suffering from “adrenal insufficiency”, which would anyway imply much stronger treatments than pregnenolone, namely hydrocortisone, prednisone, prednisolone, or fludrocortisone. In fact, I never prescribe these molecules, because I would always refer such severe adrenal cases to a Consultant Endocrinologist, in particular to Dr BB whom I trust. The reason for labelling her adrenal function as “low” lies in the repeated DHEA deficiency: 79 mcg/dL on 4 September 2014... and again 81 mcg/dL on 16 June 2015, well under range provided by the lab going from 110 to 430 mcg/dL.”

The Tribunal noted the expert report from Dr S:

“18.7. Adrenal Insufficiency Diagnosis
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I cannot see that Dr Mouton made this diagnosis at all. As with the euthyroid diagnosis, his assessment of “Low endocrine functions, both thyroid and adrenal glands” does not amount to a diagnosis of adrenal insufficiency. Nor does his treatment with “glandular support treatment strengthened by numerous cofactors” amount to the hormone replacement that such a diagnosis could require. I consider that Dr Mouton behaved correctly in this regard.”

383. The Tribunal went on to consider the joint expert report from Professor Q and Dr J:

“It is noted that Dr Mouton denies this and considered adrenal function was "low". We both agree that the test of the 17-OH-steroids is a legitimate test and can help in the diagnosis of adrenal deficiency. Dr J thinks that the extent of the reduction of 17-OH-steroids justifies treatment with pregnenolone. So, the discussion was whether additional tests were needed to be done to ascertain whether adrenal function was low or if there was "adrenal insufficiency". For frank adrenal insufficiency (Addison's disease) the treatment is hydrocortisone rather than pregnenolone. Professor Q considered that additional endocrine tests seemed to be justified, or there ought to be a reason why they were not necessary.”

384. The Tribunal also considered the expert report from Professor Q and Dr S:

“Dr S and Professor Q recognised each other's position, although their views contrasted to some extent. Dr S considered Dr Mouton was safely following good medical practice based on his knowledge and experience.

Professor Q also considered that a diagnosis of adrenal "insufficiency", or a need for adrenal support was questionable, and he doubted whether the urinary 17 hydroxysteroid test clearly justified pregnenolone treatment. He also expressed the reservation that further investigation seemed necessary if adrenal activity was deemed inadequate. We agreed that this point refers only to the specific matter of adrenal treatment, not to the vitamin and mineral therapies.”

385. The Tribunal having considered all of the expert opinion agreed that Dr Mouton did not diagnose adrenal insufficiency. It made particular reference to Professor Q’s statement that the diagnosis of adrenal insufficiency (Addison’s disease) is a different diagnosis to a low functioning adrenal gland and therefore requires different endocrinology testing. The Tribunal therefore concluded that the GMC had not proven that Dr Mouton diagnosed Patient E with adrenal insufficiency.
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386. The Tribunal therefore finds paragraph 31 of the Allegation not proved.

Allegation 32

32. You prescribed the treatments referred to in Schedule 5 which were not:

a. clinically indicated;
   
   **Found proved for Thyrocsin (containing iodine)**  
   **Found proved for Tirform (containing selenium)**  
   **Found proved for pregnenolone**  
   **Found proved for GTA (containing porcine thyroid concentrate and selenium)**  
   **Found proved for Novothyral**  
   **Found proved for L-selenomethionine**
   
   Otherwise **Found not proved**

b. supported by scientific guidelines;
   
   **Found proved for Thyrocsin (containing iodine)**  
   **Found proved for Tirform (containing selenium)**  
   **Found proved for pregnenolone**  
   **Found proved for GTA (containing porcine thyroid concentrate and selenium)**  
   **Found proved for Novothyral**  
   **Found proved for L-selenomethionine**
   
   Otherwise **Found not proved**

c. evidence based.
   
   **Found proved for Thyrocsin (containing iodine)**  
   **Found proved for Tirform (containing selenium)**  
   **Found proved for pregnenolone**  
   **Found proved for GTA (containing porcine thyroid concentrate and selenium)**  
   **Found proved for Novothyral**  
   **Found proved for L-selenomethionine**
   
   Otherwise **Found not proved**

**Thyrocsin**
**Found proved**

387. Thyrocsin is a compound containing iodine and ashwagandha extract. For the reasons outlined in relation to Patient B regarding the use of iodine, the Tribunal
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found this prescription not clinically indicated, supported by scientific guidelines or evidence based.

Tirform
Found proved

388. Tirform contains selenium. Therefore, for the reasons outlined in relation to Patient B, the Tribunal found this prescription not clinically indicated, supported by scientific guidelines or evidence based.

Pregnenolone
Found proved

389. For the reasons outlined in relation to Patient C, the Tribunal found the use of pregnenolone not clinically indicated, supported by scientific guidelines or evidence based.

GTA
Found proved

390. The Tribunal heard oral evidence from Dr Mouton that GTA contains T3 in small doses:
   “GTA is 0.8 micrograms of T3. I cannot accept that this is seen as a T3 treatment when we know that the tablet is 20 micrograms”.

391. The Tribunal noted Dr S’s expert report:
   “Although concentrations of hormones within thyroid glandular extracts do vary somewhat, it is likely that 5 milligrams of this would provide approximately 3.3 micrograms of T4 (normal daily dose being 100 – 150 micrograms) and 0.8 micrograms (800 nanograms) of T3 (normal daily dose being upwards of 5 micrograms)”

392. The Tribunal also considered the joint expert report of Professor Q and Dr S:
   “We both agreed that we would not have considered the patient hypothyroid. Dr S considered that thyroid support (non-hormonal support) was justified and that thyroid hormone doses were very small.
   ...
   Dr S considered that thyroid support therapies including those with very small doses of active thyroid hormone was a legitimate and safe approach”.

393. The Tribunal concluded that as Patient E was biochemically euthyroid, treatment with GTA which contains thyroid hormones albeit in small doses was not clinically indicated, supported by scientific guidelines or evidence based.
Novothyral
**Found proved**

394. For the reasons outlined with regard to Patient B, and given the Tribunal’s finding that Patient E was euthyroid, the Tribunal found that treatment with Novothyral was not clinically indicated, supported by scientific guidelines or evidence based.

L-selenomethionine
**Found proved**

395. L-selenomethionine is a naturally occurring amino acid which contains selenium. Patient E was biochemically euthyroid. Therefore the Tribunal found that use of L-selenomethionine for this patient was not clinically indicated, supported by scientific guidelines or evidence based.

396. The Tribunal therefore finds paragraphs 32a, b and c of the Allegation proved in respect of Thyrocsin, Tirform, Pregnenolone, GTA, Novothyral and L-selenomethionine.

397. The Tribunal determined that all other nutritional dietary supplements in schedule 5, for reasons already discussed in relation to Patient A, were clinically indicated, supported by scientific guidelines and evidence based and therefore were found not proved.

**Allegation 33**

33. You failed to communicate adequately with:

   a. Patient E’s General Practitioner regarding the tests referred to in paragraph 28 and the implications of them;
   **Found proved**

398. The Tribunal noted that after Dr Mouton’s consultations with Patient E in September 2014 and January 2015, he did not contact Dr L (Patient E’s General Practitioner).

399. The Tribunal noted Dr Mouton’s first correspondence to Dr L, dated 12 July 2015:

   “I saw your patient today hence I wish to keep you informed with the patient’s status within my practice.”
The patient has received prescriptions and dietary recommendations for a period of 6 months and has booked a follow-up appointment in my practice in 5 months.”

Hand written notes on the correspondence:

- “undefined inflammatory bowel disease (Crohn’s –ve)
- Positive antibodies (ANA+)
- Overtly low thyroid hormones T3 (explained partly by)
- Heterozygous variant DIO2 genes (TA)
- Treated with 3/4 tablet of NOVATHYRAL (T4-T3 combination) providing 75 micrograms of T4 + 15 micrograms of T3
- Thyroid US scan requested to fine tune it

400. The Tribunal noted a letter dated 15 July 2015, sent from Dr L to Dr Mouton, which stated:

“If I have understood this correctly you have sent off blood and urine samples and on the basis of these results and your examination you have started her on 18 different medications. Most of these seem to be harmless vitamins and food supplements but you have also taken it upon yourself to start her on thyroid supplements (using a fixed dose preparation of T4 and T3 that is unlicensed in the UK) despite her having normal blood thyroid function tests.

The rationale for this appears to be a variety of borderline changes you have noted in the results, none of which are specific for hypothyroidism. Indeed the specific blood tests you did to check her thyroid function were normal. As you know overtreatment with thyroid hormones is not without long-term risks including osteoporosis and atrial fibrillation.

I realise that you had sent me your report for my information and that you are not asking for my opinion or for me to prescribe any medication. However I do feel that I have an ethical duty to write to you and copy [Patient E] in lest she thinks that I agree in anyway with this management plan”.

401. Thereafter, until 5 October 2015, Drs Mouton and L correspond regarding Dr Mouton’s management strategy for Patient E.

402. The Tribunal then went on to consider the joint expert report of Professor Q and Dr J, which stated:

“We both agree that Dr Mouton's communication was mainly indirect in the beginning by providing all information to the patient, who was supposed to transmit this information to the GP. Later on, he proved that he improved his communication, but, because his functional medicine is not well-known by
other physicians, he would be better advised to improve his communication further by adding more explanations and also scientific evidence.”

403. The Tribunal considered that Dr Mouton had a clear duty to inform Patient E’s General Practitioner of the results and the implications of these tests. In the United Kingdom the General Practitioner has an important and well recognised role as the central coordinator of the patient’s care and medical records. This is not restricted to an individual practitioner but may be shared among a number of General Practitioners within an individual practice. The DIO2 test was carried out in January 2015. Dr Mouton did not correspond with Dr L until 12 July 2015. In that correspondence the only reference to the DIO2 test was a handwritten note ‘Heterozygous and variant DIO2 genes”. The Tribunal did not consider that this would have been readily comprehensible to a General Practitioner. Dr Mouton did give a further explanation for this test in his letter dated 29 September 2015 but this was only when his treatment plan was being challenged by Dr L. The Tribunal concluded that Dr Mouton’s communication with Patient E’s General Practitioner concerning the result and implication of this test was not adequate in that it was not timely, it was unclear initially on 12 July 2015 and Dr Mouton did not supply the actual result of the test to Dr L.

404. In respect of the Apo E and CTLA-4 tests, the results of these tests were sent to Dr L on 12 July 2015, relatively soon after they had been carried out. However they were accompanied by a large volume of other test results and a list of 18 prescriptions made by Dr Mouton for Patient E. There was no accompanying explanation by Dr Mouton as to why the tests had been done or the significance of the results. Accordingly, the Tribunal concluded that Dr Mouton’s communication in respect of these tests was also not adequate.

405. The Tribunal determined that in respect of Dr Mouton adequately communicating with Patient E’s General Practitioner, this allegation is proved for these reasons.

406. The Tribunal therefore finds paragraph 33a of the Allegation proved.

   b. a Gastroenterologist regarding the intestinal treatments you intended to prescribe.

   Found not proved

407. The Tribunal considered the oral evidence of Dr Mouton:

   “Q Focusing on this document, the allegation is that you did not communicate adequately with a gastroenterologist regarding treatments. Here you have a document dated March 2013 which appears to relate to
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gastroenterology. Is there any particular reason why you did not attempt to make contact with Kings College Hospital?

A    Because she was pretty clear saying that she was not seeing that gastroenterologist anymore, and in fact, she made it clear as well that she was not seeing any gastroenterologist. With the benefit of hindsight, I think she did not want really me to go into that direction. She has put an obstacle there which I only found out about. When I got the NHS health records, I found out that she was in fact seeing a gastroenterologist. I did not know about it”

408.    The Tribunal was mindful that Patient E first consulted with Dr Mouton in September 2014. It noted correspondence from Dr CC (another General Practitioner at the same practice as Dr L) addressed to a Consultant Gastroenterologist, dated 1 October 2014:

    “Thank you for seeing this patient find below a copy of my last consultation with them.

    Notes:
    H: New pt. Diagnosed with Crohns disease at Kings College Hospital in 2013. Presented at the time acutely unwell with abdominal pain, pr bleeding. Was put on asacol and steroids.

    Moved to Canada and had normal colonoscopy there in June 2014. She has been off all medication since December 2013. She has no further attacks. Denies any abdominal pain/bleeding and is well.

    She requires a referral for routine follow up with a Gastroenterologist.”

409.    The Tribunal noted Dr L’s letter to Dr Mouton dated 20 August 2015:

    “She also informed me that she was returning to live in Australia in a couple of weeks time and will be having follow up there”.

And a further letter dated 5 October 2015:

    “She is no longer registered with me”.

410.    The Tribunal noted the letter from the Imperial College Healthcare NHS Trust, dated 30 November 2015, to Dr CC:

    “We are writing to inform you that the patient below has cancelled their outpatient appointment for Gastroenterology on 08 April 2016 at 15:10, and has informed us that they do not wish to reschedule at this point...
We have therefore discharged them back to your care. If your patient’s circumstances change you will need to refer them again…”

411. The Tribunal noted that the evidence before it did not clearly demonstrate that in the time period concerned, Patient E was under the care of a gastroenterologist. It noted Dr Mouton’s oral evidence as set out above and determined there was no alternative evidence to contradict this assertion. Accordingly, the GMC failed to prove that Dr Mouton would have been aware of a gastroenterologist with whom he would have had a duty to communicate.

412. The Tribunal therefore finds paragraph 33b of the Allegation not proved

**Patient F**

**Allegation 35**

35. The investigations referred to in paragraph were:

   a. not clinically indicated;

   Found not proved in relation to Apo E genotype testing
   Found proved in relation to DIO2 genotype testing
   Found not proved in relation FUT2 genotype testing

**Apo E genotype testing**

**Found not proved**

413. The Tribunal considered Dr Mouton’s rationale for these treatments as set out in his witness statement:

   “As already explained for previous patients, I value this genomic test to help me personalize my dietary advice. Given Patient F’s E4/E4 genotype, I have not allowed him to consume excessive amounts of dairy products and coconut oil.”

414. The Tribunal noted Dr Mouton’s response to Dr M (Patient F’s GP) dated 16 December 2015, which stated:

   “Thank you for your letter dated 10th December 2015 and for your positive comments. It is true that the two boys seem thriving, and they are much more happy with their health and mood.

   They had strange dietary habits that I have largely reformed according their respective tests. In particular, they still remain always hungry despite eating loads of good quality fresh foods prepared by their mother. As they both tend
to suffer from intestinal overgrowths of yeasts and bacteria within the frame of what we can call a fermenting gut issue, I wanted to make sure they should reduce their intake of carbs and replace them with much more healthy fats.

The apoE E3/E3 genotype appears compatible with higher fat intake without any downsides, whereas the presence of one E4 copy of the DIO2 gene leads to increased cholesterol levels in such cases. Thus, E3/E4 leads to recommending a balanced diet with carbs-proteins-fats.

I had explained this to the mother who lives the struggle of feeding them correctly/ sufficiently on a daily basis. These kids have of course nothing to do with other implications of that gene in Alzheimer’s disease odds, which anyhow are perfectly well corrected by a balanced diet...

What exactly is the point thinking Alzheimer for children of this age? None to my point of view!”

415. The Tribunal also noted a letter sent to Dr Mouton from the mother of Patient F and G, dated 26 January 2016:

“I am writing to let you know that I was asked to go to a meeting with my GP today concerning [Patient F] and [Patient G] genetic tests. The GP expressed her concerns about carrying out such tests on minors and advised me that the practice is raising this with the GMC. I am aware that the boy’s tests were carried out following the findings from my tests when the lab recommended that the same analysis should be carried out for family members of the first degree, i.e. my sons. As we have health issues in common, I wanted the tests to be carried out. I am happy that the results meant that you could treat them accordingly and they are now so much healthier than they were when they first came to see you and are really thriving. So, I trust this will not cause you any problems.”

416. The Tribunal noted that there is some evidence that Patient F failed to thrive prior to this test being carried out in September 2015. It therefore accepted that for the reasons already given in this determination, there was a clinical indication to carry out Apo E genotype testing to assist in providing dietary advice.

417. The Tribunal therefore finds paragraph 35a in relation to paragraph 34a of the Allegation not proved.

DIO2 genotype testing
Found proved
418. For the reasons outlined with regard to Patient A, the Tribunal found the investigation DIO2 genotype testing was not clinically indicated.

419. The Tribunal therefore finds paragraph 35a in relation to paragraph 34b of the Allegation proved.

**FUT2 genotype testing**

**Found not proved**

420. The Tribunal considered Dr Mouton’s rationale for these treatments as set out in his witness statement:

> “Homozygous variant FUT2 genotypes (non-secretors), present numerous disadvantages, especially they are more prone to intestinal dysbiosis, which throws more light on reason for Patient F to frequently complain of indigestion, stomach pain, colicky pains, burping, gas, halitosis, and upset stools...”

421. The Tribunal noted that there was some documented evidence that Patient F showed signs of failure to thrive. It therefore accepted that for the reasons already given in this determination, there was a clinical indication to carry out FUT2 genotype testing to assist in providing dietary advice.

422. The Tribunal therefore finds paragraph 35a in relation to paragraph 34c of the Allegation not proved.

b. conducted without obtaining informed consent from Patient F and/or from his parent or guardian;  
   **Found proved**

c. conducted without appropriate counselling of Patient F and/or his parent or guardian;  
   **Found proved**

**Apo E genotype testing**

**Found proved**

423. The Tribunal noted the oral evidence of Dr Mouton in relation to consent and counselling for genotype testing in children:

> “Q We then have the usual pairing of consent and counselling. We have the documents in D8. We perhaps do not need to turn them up because we are familiar with the layout. Obviously the difference here is you are dealing with a young person. How, if at all, do you vary your practice? Who is in the room and how are you dealing with consent and counselling?”
A In the room we have both siblings because they always come together, and the mother. I explain, as I have already detailed earlier today, I explain the whole topic. Clearly here I wanted an ApoE assessment, or I made very clear the outcome. By the way, the mother had already been tested for similar reasons, and she was well aware about the possible outcome. Everybody agreed. I speak with the kids in front of the mother. The kids have to agree one at a time, and then the mother signs. In fact, the mother did sign initially for both, but then... No, it is not even the case because for this I followed the European rule, the German rule, and the limit of age is 18. So I think that we always needed the mother's signature. I still see those two boys.”

And further:

“Q Can I ask you, Dr Mouton, what the word “Gillick” means to you?
A Which word?
Q Gillick.
A Gillick? Gillick is, I think, a female activist who has triggered some change in the regulations, and it is about making a child, or a teenager I think under 16, able to make medical decisions for his own health management beyond the parents’ authorisation, if I am ... this is accurate.

Q Do you understand what steps a medical practitioner is required to take when obtaining consent from someone who is under the age of 18?

A What steps? Well, between 16 and above 16, that is the threshold. Now it is a bit confusing because, as I have already said, in Germany it is above 18, so between 16 and 18, I mean, a sort of in between situation. I have not been put in this situation to my understanding so I do not see why I should have gone further into the regulation. If it was the case, I would study the regulation as I do. I am fully Care Quality Commission registered, so I go through tons of regulations when relevant.

Q You sought consent in relation to the genetic testing on Patients F and G, did you not?
A Yes, and when the age limit was 16 I have asked the mother to sign the consent form, even though I have explained the whole topic, including the counselling, including the Apo E counselling because that was critical for
Patients F and G, and I have explained that to the child and the mother has signed. I have not been told that was wrong.

Q Did you give any thought to the question whether Patient F, who was then 13, would be able to understand what you were asking him?

A Well, of course, he is a very bright young little guy and the mother had already explained to him that she was willing to have this Apo E test, which I was very satisfied with because that was helping me to address their diet, which is a complicated topic because these two kids eat loads of food, so it is my whole ... to tidy up a little bit the kind of food they eat and they have improved massively, which has contributed to healthy improvements. That was very easy to understand, and because it became from the mother being herself carrying an Apo E4 allele, so all this concern about counselling before the testing in fact is much more straightforward than what you would think because this is not about genetic diseases. You need a lot of pre-counselling for genetic diseases, but I do not deal with genetic diseases, I deal with polymorphisms and there is still a need for counselling. It is very light compared to genetic diseases. All the algorithm that has been used for my case is based on something that I do not do. I do genomic testing. You need counselling. You need the patient to be aware that you are going to test for this Apo E polymorphism, which is relevant to potential risk of Alzheimer’s, but here it was not really something to be overly worried about because the mother has an Apo E polymorphism and the question was to know if this was transmitted to her boys. She was willing to know, the boys were willing to know. That was very clear. We had a thorough discussion about this.

Q Your answer so far has given me the impression at least that what you are saying is that mum had decided it was all right, so as far as you were concerned that was all that needed to be asked?

A No, because the patient is in the room. The patient is part of the discussion. You know, for these boys, it could be different with children, but these boys, teenagers, pretty well advanced, you know, on computers and things and smart phones and everything, they are intelligent human beings, so they participate to the discussion and they know what they are going to be tested about, plus if the concern is Alzheimer’s disease, can we come back to what I have tried to explain in my letter, perhaps not with such good wording, that for a child who is 13, the fear of becoming Alzheimer’s 60 years later, let me say, is not something extremely relevant. The topic has been about the insurers. The insurers could see this information that might have changed something in the cost of the indemnity or something like that. That is the only topic. Nobody there in this family was afraid of being labelled with an ApoE4 allele. They knew what it was. They were perfectly conscious about it because the mother had already gone through that and that was explained to
the kids. They have not been forced to a blood extraction for testing these genes. This is completely wrong. Why not ask the kids then?

...

Q Just remember I was asking you about the conversation where you sought consent.

A That is all what we discussed. We discussed what was going to happen with the results of the polymorphism and in this case the Apo E4 linked to Alzheimer’s, I did not even have to disclose that, which I told you I typically disclose because the results, when you come back to the patient and the results clearly show if there is an increased risk of Alzheimer’s, so I have to anticipate any concern about that from the patient – sometimes I drop the test. In this case that was very clear, the mother has this Apo E4 gene which is how one of the boys has the E4 from both parents, so obviously from the mother and the father in his case, not the other one, only one parent, and that was clearly presented to the patient and likely – I cannot remember that, but likely discussed in the previous cycle which maybe next time it will be relevant to test for this. It is not something that I have forced in any means. I think the mother in her letter that is in the bundle – and if you want we can go to that letter, it is easy to spot – does make clear that she was happy for the kids to do that and the kids were happy as well. You could pretend they have been influenced by their mother, but do you not think a child, 13-year-old, is going anyhow to be influenced by the mother? I mean, am I supposed to see him alone and get a confession? I do not totally get the point, I have to say. This has been a fair discussion and they knew exactly what all this was about, as always in my practice. I spent one hour with these patients, you know. What do we do for one hour? We speak about the weather? No, we do those medical things step-by-step. It is not all recorded in a written format, agreed. That may be something I have to change to a certain point, make sure some of those things are recorded but ...

...

I am sure you will ask my expert, Dr T, how much counselling is adequate for genomic polymorphism. We cannot truly rely on Professor P to tell us about that because Professor P does not do this and Professor Q does not do this. I do this. I know how much counselling I have to deliver and that is what I deliver.

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...Apo E is a bit more complex. Each situation is different. For some patients I even feel that the patient is not going to be very happy with the idea that there might be something related to Alzheimer’s disease, and then I do not even test for it. You see, it does not need a lot of counselling because I cherry-pick the patients who are happy to go through Apo E polymorphism.

...

Q I will suggest in due course that this letter indicates that you minimised the importance given to the connection between the ApoE allele and Alzheimer’s disease. Do you think that is a fair characterisation?

A I think that is totally unfair. That is an insult to my intelligence. I cannot imagine one second that anyone would believe that with my experience, with my practice, with my knowledge of genomic testing I would ignore or minimise or, how to say, dismiss the link between Apo E and Alzheimer’s disease, but, you know – well, I was going to say everybody knows that, which is certainly not true, but this is – this is the A of an alphabet that goes down to Z. I minimised the implication of Apo E Alzheimer’s link for kids who are 13 because they will have to deal with that link in 60 years. If you do not mind me going back 60 years ago, what was happening 60 years ago? We did not – the state of medicine in 1957, you know, it is not exactly what it is today. Most of the things we are discussing here were even not known, certainly not genes and polymorphisms, so that is irrelevant. But I did not dismiss the link. The link is so obvious. How could I dismiss that?

...

Coming back to this case, the concern here on my behalf was diet, not Alzheimer’s. Maybe the mother had that in her mind, but I am the one who prescribes. I always have a reason for prescribing a test, or a treatment by the way, and my reason was dietary management. Now, if we both agree on her behalf because she thinks about Alzheimer’s, on my behalf because I think about diet, well, we still agree and we run the test and I explained the consequences”.

424. The Tribunal noted the oral evidence of Professor P:

“Q Finally, on the matter of consent, could you help the Tribunal about what issues are involved in consenting children?

A This has been the subject of much debate and written papers and books even over the years. I came into genetics in the UK in 1990. It was a very hot topic over the early 1990s while the availability of genetic testing
was beginning to grow. Some of our colleagues with a lot of foresight realised that this was potentially going to be an issue for the future and so they tried to get one step ahead of the game in terms of what sort of policy decisions, what issues should be out there.

We have a very clear-cut principle that we do not undertake genetic testing on children unless there is a clear clinical indication for doing so, i.e. a child is symptomatic with something. We do not undertake predictive testing on children unless there is very clearly value in the information we are going to gain from that test. In other words, if it is positive, is that result going to change the situation for that child, looked after by his parents or guardians, at that time or during childhood? There are some very good examples where that is the case. In the field of endocrinology, we have a condition called multiple endocrine aplasia – actually multiple endocrine aplasia type 2, to be more precise – where if you have a pathogenic mutation in the gene, actually you should have your thyroid removed in childhood, because we know there is a significant risk of cancer at almost any age. It certainly can occur young. You try to take parents through that, saying, “We’re going to do this test on your healthy child and, if it’s positive, we’re going to send them to a surgeon to take their thyroid out.” That is quite a big piece of information, but there are very good grounds for doing that. There are some others as well. With Huntington’s disease, as an example at the other end of the spectrum, so to speak, you will never get a clinical geneticist to do that test on a child who is under age, by which we mean – it is a moveable feast slightly – we normally talk about the age of 18. It would be extremely rare for us to do that test on an under 18 year-old unless they are symptomatic, but then it is not a predictive test, it is a diagnostic test and Huntington’s only rarely presents under the age of 18, because the individual themselves need to make their own decision about whether they want to know that they will develop Huntington’s disease at some point in the future. That is not the parents’ decision; that is the individual’s decision. So we would never accede to a parent twisting our arm or trying to twist our arm to do that test. There are some exceptions to the rule. I have even had some in my own practice, whereby a 17 year-old with a family history of Huntington’s turned up pregnant and she wanted to know. These are exceptions to the rule, but they are rare occurrences. Therefore in principle we do not do predictive genetic testing on children when there is no clear-cut medical intervention that will benefit them at that time.”

425. The Tribunal also noted an email dated 18 December 2015 from Professor DD, consultant in clinical genetics to Dr M:

“I am extremely concerned about the activities of Dr Mouton. Having read his letter to you alarm bells started to ring and I immediately searched the GMC register to see if his name was listed. In my view the GMC should be informed
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that he appears to be offering testing in a way that is not compliant with professional guidelines and this is dangerous. (As it happens, I chaired the Human Genetics Commission committee that wrote the Framework of Principles for the Regulation of Direct to Consumer Genetic tests.)”

426. The Tribunal accepted that Patient F’s mother requested the Apo E genotype test. However, the Tribunal heard detailed evidence from Professor P as to what is acceptable practice in respect of the genetic testing of children. He was clear that all genetic testing of a child ought to be deferred until the patient is able to consent for themselves, ideally when the patient is over the age of 18 years, unless there is a clear clinical urgency for doing so before.

427. The Tribunal considered that the evidence provided by Professor P was supported by the degree of concern expressed by Professor DD when she heard of the tests being carried out in this case.

428. Dr Mouton was questioned at length before this Tribunal regarding the specific process of obtaining informed consent for genetic testing from Patient F. The Tribunal considered that Dr Mouton’s oral evidence demonstrated an inadequate understanding of Gillick competence. At various points in his evidence Dr Mouton made it clear that he did not regard Alzheimer’s disease as a relevant consideration for a child of 14 years old. This is in keeping with his comments in correspondence with Patient F’s GP, Dr M, on 16 December 2015 when he stated “What exactly is the point thinking Alzheimer for children of this age? None to my point of view!” The Tribunal concluded on the balance of the evidence before it that Dr Mouton’s preoccupation was the information that the test might give him to provide dietary advice and not the potential ramifications in respect of Alzheimer’s disease.

429. The Tribunal also noted that Dr Mouton’s explanation was that all European genetic laboratories requested signed and dated consent forms. There was no evidence that he discussed with the patient the generally accepted guidance for deferring genetic testing to the age of 18 unless there was a clinically urgent indication to do so. Dr Mouton also failed to discuss and inform Patient F that ultimately the decision and consent to the process lay with himself and not his mother. There was no evidence to suggest that Dr Mouton gave this advice to either Patient F or his mother. The Tribunal also considered that if Dr Mouton had been fully mindful of his responsibilities to counsel Patient F appropriately and obtain proper informed consent, he would have made some record of the conversation he had had with Patient F. This is especially so as a result of the Apo E test in respect of Patient F indicated an increased risk of developing Alzheimer’s disease by a factor of 15. The Tribunal concluded that he did not discuss the issue fully with Patient F and did not obtain informed consent or counsel him appropriately.

430. The Tribunal therefore finds paragraph 35b and c in relation to paragraph 34a of the Allegation proved.
DIO2 and FUT2 genotype testing
Found proved

431. In respect of DIO2 and FUT2, the Tribunal was mindful of the fact that these tests do not have the same potentially serious implications attached to them. However the Tribunal considered that in the light of Professor P’s evidence, in order to properly counsel Patient F and obtain informed consent, there should have been a discussion as to whether the tests should be deferred until Patient F was 18 years of age and emphasised that this was not a decision for the parent to make, but a patient decision.

432. The Tribunal therefore finds paragraphs 35b and c in relation to paragraph 34b and c of the Allegation proved.

Allegation 36

36. You failed to adequately interpret or act upon the findings that Patient F was biochemically euthyroid.
Found proved

433. The Tribunal considered the oral evidence of Dr Mouton:

“Q  First of all, I am looking at paragraph 36 of the allegation. Do you accept that Patient F was biochemically euthyroid?

A  Well, you know, again you suggest me to answer to a switch question: hypothyroid, euthyroid. There is no good answer to that type of question. My view is clearly stated at the end of my answer to allegation 36, which is page 33 around the middle:

“Such a combination of clinical and biological data cannot guarantee Patient F was euthyroid...

...So, technically I am entitled to suspect he is not so euthyroid. This is what happens in the US... if I work in the UK I am blamed for treating. If I look at it from an American point of view I am blamed for not treating, so I am always blamed. The problem is that treating this boy has helped him immensely.

Q  ... put quite clearly to you the allegation which is that this child was biochemically euthyroid. On that occasion, you gave him thyroid support complex..
A  Yes, so as you can see I did not treat him with anything hormonal from scratch. I have tried, of course I have privilege *(sic)* another route. I have not been able to fully answer because besides this controversy around the ranges...

“... recent studies suggest perhaps [a serum TSH level] between [2.5 and 3 ml units per litre as the proper cut-off].”

So, we are in that situation. That is one point. The second point is hypocholesterolaemia. How can we explain that a boy this age is already hypocholesterolaemic, whereas he was not necessarily eating a lot of red meat and those things? It is likely that the thyroid has a hole. Then something that apparently is of very limited value for some endocrinologists, but that is not our Belgian point of view, are the symptoms: constipation, fatigue, cold feet, sleeps with socks, terribly dry skin, eczema was horrendous, puffy eyes, low concentration. So, of course one of those symptoms would not be specific at all. Two, maybe, three, four. What do we do with four?

Q  Do you count cold feet and sleeping with socks as two separate symptoms?

A  No, I count constipation, fatigue, feeling cold, being cold and dry skin – these are four major thyroid symptoms. One, no value. Two, maybe. Three, perhaps. Four, we have to consider. I told you, I am not going to treat those symptoms. I am not going to treat the TSH above three, otherwise I would give thyroxine to tons of kids because that is not uncommon. I treat when everything comes together – high TSH, a lot of symptoms, high cholesterol, ongoing fatigue, after one year I give a trial. What trial do I give? What therapeutical trial do I give? I give a quarter grain of Armour thyroid. Why do I give Armour Thyroid – because it is something I can understand there is some concern about – because it is the smallest treatment I can give. I am not attracted to Armour thyroid. I told you, it is big, it is too much T3, but when it is a matter of giving 9 mcg of T4 and 2 mcg of T3, what else can I do? Am I going to ask to split the T3 tablet available in this country in eleven – in ten bits? It is impossible, so this is the smallest thing I can give.

Q  The answer, Dr Mouton, to your question is you do not prescribe thyroid supplements to children who are euthyroid.

A  First, I am demonstrating you that the child is not guaranteed being euthyroid. Two, you instruct me here something, but I am the medical doctor. I make the final decision in the patient’s best interest. What did you read me a few days ago about the 16(a) paragraph of *Good medical practice*? It is to ---
Q Can we stick to the point?

A I do not know it by heart. I have to be satisfied that the drugs or treatment serve the patient’s needs. That is exactly what I have done. I have satisfied that and I am following the Good medical practice. It is not my fault if the Good medical practice guidelines do not always cope with other guidelines. You have tons of guidelines. You will always find guidelines against me, but I am going to find guidelines in my favour or scientific evidence in my favour. What does that mean? It is controversial. Professor Q would not have treated. I did. But is it something to blame or prohibit? My view, but you are not going to follow that, is that because it has massively changed this child’s life, and the mother is stating that in further letters that you will be able to see later on in this hearing, I did well.”

434. The Tribunal went on to consider the joint expert report from Professor Q and Dr S:

“Professor Q and Dr S agreed that this patient was biochemically euthyroid.”

435. The Tribunal also noted the joint expert report from Professor Q and Dr J:

“We both agree that a certain degree of hypothyroidism may exist with thyroid tests within reference range (at borderline low T3, T4, levels, high-normal TSH levels), that there is a place for thyroid treatment of hypothyroidism with T3-T4 treatments (and not on thyroxine), and desiccated thyroid.

We also agree that thyroid guidelines are partly designed for less experienced physicians, and that thyroid treatments not included in the guidelines may be prescribed by more experienced physicians, on conditions that safety and efficacy is adequate or can adequately be shown. We both agree that there is scientific evidence.

However, Professor Q considered that there was insufficient evidence to make a diagnosis of hypothyroidism in this child, and that such a diagnosis would not be considered by a paediatric endocrinologist. A diagnosis of hypothyroidism is a significant and life long diagnosis, with major implications for long term treatment, health monitoring and health costs. Likewise, Professor Q saw little justification for DIO2 testing. While the relative safety of closely monitored thyroid treatment is agreed, there were also reservations about the possibility of overtreating a child.
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Dr J considered that there were grounds for a diagnosis of hypothyroidism, as explained already, and therefore a trial of thyroid treatment, as outlined in his Society’s Consensus Statement 9 document. Dr J also acknowledged that in his practice he has experience of providing thyroid extracts to children.”

436. The Tribunal considered that all of the results were within the reference range. During the course of extensive reading, the Tribunal noted the 2012 ATA/AACE guidelines which clearly stated “Although most physicians can diagnose and treat hypothyroidism, consultations with an endocrinologist is recommended in the case of children and infants”. The Tribunal also paid particular regard to the joint expert report of Professor Q and Dr S who concurred that Patient F was euthyroid. Accordingly, the Tribunal concluded that by commencing the prescribed treatment of thyroid glandular support and thyroid hormone treatment, that included T3 and T4, Dr Mouton failed to adequately interpret and act upon the finding that Patient F was biochemically euthyroid.

437. The Tribunal therefore finds paragraph 36 of the Allegation proved.

Allegation 37

37. You prescribed thyroid hormone treatment, Armour thyroid, to Patient F which treatment was not:

   a. clinically indicated;  
     Found Proved

   b. supported by scientific guidelines;  
     Found Proved

   c. evidence based.  
     Found Proved

438. The Tribunal considered the joint expert report from Professor Q and Dr S, which stated:

   “Professor Q and Dr S agreed that this patient was biochemically euthyroid and so thyroid hormone treatment was not indicated.”

439. The Tribunal was mindful that Patient F was biochemically euthyroid. Despite which his first treatment regime from Dr Mouton included a prescription that contained thyroid hormones. The Tribunal had particular regard to Professor Q’s opinion that a child should not be prescribed thyroid hormone treatment by anyone other than a consultant endocrinologist and that most paediatric endocrinologists would not have diagnosed hypothyroidism or treated this child with thyroid hormones. It determined that the thyroid hormone treatment was not clinically
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indicated as Patient F was biochemically euthyroid as discussed above. The Tribunal further noted that this was Patient F’s first treatment by Dr Mouton; the guidelines clearly state that combination thyroid treatment should not be first line therapy in any patient.

440. For these reasons and those already set out in this determination with regard to the prescribing of Armour Thyroid, the Tribunal determined that in this case thyroid hormone treatment was not clinically indicated, supported by scientific guidelines, or evidence based.

441. The Tribunal therefore finds paragraph 37a, b and c of the Allegation proved.

Allegation 38

38. You failed to communicate appropriately with Patient F’s General Practitioner in that you:

   a. did not include information in your letters to Patient F’s General Practitioner regarding his:

      i. previous and/or new diagnoses;  
         **Found not proved**

442. The Tribunal had regard to correspondence sent to Dr M, Patient F’s General Practitioner, from Dr Mouton, dated 16 December 2015, referring to both Patients’ F and G:

   “As they both tend to suffer from intestinal overgrowths of yeasts and bacteria within the frame of what we can call a fermenting gut issue, I wanted to make sure they should reduce their intake of carbs and replace them with much more healthy fats.”

443. The Tribunal noted that, as drafted, this Allegation requires the GMC to prove that Dr Mouton did not include any information in his letters to Patient F’s General Practitioner regarding previous and/or new diagnoses. The Tribunal noted Dr Mouton’s letter to Dr M, dated 16 December 2015, in which he refers to Patients F and G tending to “suffer from intestinal overgrowths of yeasts and bacteria within the frame of what we can call a fermenting gut issue”, arguably indicating the diagnosis of intestinal dysbiosis. The Tribunal therefore determined that there was some information regarding a diagnosis.

444. The Tribunal therefore finds paragraph 38a(i) of the Allegation not proved.

   ii. key results; 
      **Found not proved**
445. The Tribunal considered correspondence sent to Dr M, from Dr Mouton, dated 2 November 2015:

“I saw your patient today hence I wish to keep you informed of the patient’s status within my practice.

I therefore send you a copy of the results as well as the report that I have also handed over to your patient during today’s appointment.

The patient has received prescriptions and dietary recommendations for a period of 6 months and has booked a follow-up appointment in my practice in 5 months…”

446. The Tribunal noted that within his letter Dr Mouton had enclosed results of tests that had been carried out, a report and dietary recommendations. The Tribunal determined that inclusion of these tests, reports and recommendations amounted to provision of key results to Patient F’s General Practitioner.

447. The Tribunal therefore finds paragraph 38a(ii) of the Allegation not proved.

iii. investigations;

**Found not proved**

448. The Tribunal noted that the correspondence sent to Dr M, from Dr Mouton, dated 2 November 2015 included some information regarding investigations Dr Mouton had carried out with regards to Patient F.

449. The Tribunal therefore finds paragraph 38a(iii) of the Allegation not proved.

iv. treatment plans.

**Found not proved**

450. The Tribunal noted that the correspondence sent to Dr M, from Dr Mouton, dated 2 November 2015 included some information relating to treatment plans and dietary recommendations.

451. The Tribunal therefore finds paragraph 38a(iv) of the Allegation not proved.

b. failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing to which you referred Patient F, namely:

i. why the tests were carried out;

**Found proved**
452. The Tribunal considered Dr Mouton’s letter to Dr M dated 16 December 2015:

“...The Apo E E3/E3 genotype appears compatible with higher fat intake without any downsides, whereas the presence of one E4 copy of the DIO2 gene leads to increased cholesterol levels in such cases. Thus, E3/E4 leads to recommending a balanced diet with carbs-proteins-fats.
I had explained this to the mother who lives the struggle of feeding them correctly/sufficiently on a daily basis! These kids have of course nothing to do with other implications of that gene in Alzheimer’s disease odds, which anyhow are perfectly well corrected by a balanced diet...”

453. The Tribunal noted the expert report of Dr S, which stated:

“24. 7. I note that in Dr Mouton’s letter to the GP Dr M on 16th December 2015 he discusses “The ApoE E3/E3 genotype” but then refers to “the presence of one E4 copy of the DIO2 gene”. I consider this to be a simple error, which does not imply misunderstanding on Dr Mouton’s part. I state this because I can see from what he writes throughout his Comments on Allegations that his knowledge of genomics is adequate to prevent such a misunderstanding.”

454. The Tribunal noted that in his letter to Dr M on 16 December 2015, Dr Mouton makes reference to his justification of his use of the Apo E genotype testing with Patient F. The Tribunal could see no evidence that Dr Mouton had made any attempt to adequately or appropriately explain to Patient F’s General Practitioner why the DIO2 or FUT2 genotype tests were carried out.

455. The Tribunal therefore finds paragraph 38b(i) of the Allegation proved.

ii. the degree of explanation given to Patient F’s parent and/or Patient F prior to taking the test;
   Found proved

456. The Tribunal noted that Dr Mouton asserted to Dr M that he had adequately and appropriately counselled Patient F and Patient F’s mother prior to genotype testing. However, there is no evidence to suggest he offered any further degree of detail or explanation of what that entailed. The Tribunal concluded that Dr Mouton failed to respond adequately or appropriately to the questions from Patient F’s General Practitioner as to the degree of explanation he had given to Patient F’s parent and/or Patient F prior to taking the test.

457. The Tribunal therefore finds paragraph 38b(ii) of the Allegation proved.
iii. any implications that the General Practitioner should be aware of arising from the results of the genetic testing; **Found proved**

458. The Tribunal noted that in correspondence to Dr M on 16 December 2015, Dr Mouton stated; “What exactly is the point thinking Alzheimer’s for children of this age? None to my point of view!” The Tribunal concluded that Dr Mouton was minimising the possible implications of the results in respect of Alzheimer’s disease and that this was not an adequate or appropriate response to the questions from Patient F’s General Practitioner.

459. The Tribunal therefore finds paragraph 38b(iii) of the Allegation proved.

c. suggested that the results of the genetic testing that you sent to the General Practitioner should not be entered into the medical notes of Patient F. **Found not proved**

460. The Tribunal noted the witness statement of Dr Mouton:

“The initial desire not to enter apoE genotype testing in Patient F’s file clearly came from his mother. She did notify this desire of the GP, Dr M. It now appears that Dr M responded favourably in a first move, saying that he understood the issue and was ready not to include all the material in NHS records. He suggested a meeting at the Practice with the mother, in order to decide what should go onto the records and what should not. I have learned for Patient F’s mother that Dr M did send her a letter expressing his positive attitude and suggesting her to book an appointment to solve the issue.”

461. The Tribunal considered Dr Mouton’s response letter to Dr M, dated 16 December 2015:

“Because of potential misinterpretations of this special gene implications regarding Alzheimer's disease, it is probably better not to enter this data in the children's medical notes. In fact, the concern seems to arise from possible insurers access to the NHS medical notes, which sounds odd to Belgian ears because my feeling is that such matters are covered by medical secret!

Let us be realistic: very soon, everyone will have his genome fully scanned, as it is already the case for many of my patients running the Me23 DNA test. That will obviously help us in order to prevent diseases: genes intervene for 30% but environment / diet / lifestyle represent 70%.

Let me know if these answers satisfy you. I remain available for any further queries, of course.”
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462. The Tribunal noted the letter sent from Dr M to Patient F’s mother, dated 3 December 2015:

“...I am aware you have some concerns about recent genetic analysis of her sons going on their medical records, and I quite understand this. I think it would be helpful if you made an appointment so I can clarify the rationale behind the tests being done, the implications etc. and then together we can decide which bits of the report from Dr Mouton should go on their records.

I will await to hear from you”.

463. Following questions from the Tribunal, Professor P stated:

“Q …in relation to Patients F and G, who are the paediatric patients, and the conversations which have occurred between Dr Mouton and the GP about whether the results of the genetic testing should be placed within the children’s medical records. Could you help the Tribunal a bit on that particular thorny issue?

A I do find that a difficult one. I think I put in my report or one of my responses somewhere that I was not sure what the legal position there was, but I thought that information should at least be with the family in some form or other. I think I would maintain that position. It is not just information for the doctor. Why would we be paternalistic about that information? It is not our information, it is the patient’s and the family’s information.”

464. The Tribunal noted that Dr M was open to discussing the matter of which results should be entered into the medical records. Whilst he was not against the idea of a discussion, he did not clearly agree to avoiding the genetic test results being entered into the medical records.

465. The Tribunal went on to consider the oral evidence of Dr Mouton:

“...The topic has been about the insurers. The insurers could see this information that might have changed something in the cost of the indemnity or something like that. That is the only topic...”

And further:

“...This whole thing came out because of including or not the Apo E results in the NHS records. By the way, Professor P is of the opinion that they should not be included in the health records, and Dr M did send a letter to the mother saying that he was open to the idea of not including them. That was the controversy, not the fact for testing Apo E by itself...”
466. The Tribunal considered the evidence of Professor P on the issue of whether or not genetic testing should be entered into the medical records for a patient under the age of 18. He stated that it was a difficult issue and he was not sure of the legal position. Dr M, in his letter to Patient F’s mother dated 3 December 2015 demonstrated that he was at least open to the possibility that some parts of Dr Mouton’s report should not go into Patient F’s medical record. In his letter dated 16 December 2015, Dr Mouton suggested that the result for the genetic testing in respect of Patient F in relation to Alzheimer’s disease should not be entered into the medical records because of the concern that possible insurers might have access to the NHS notes in the future.

467. The Tribunal noted that at the end his letter to Dr M, Dr Mouton indicated that he remained available for any further queries. In the absence of evidence to the Tribunal that it was an inappropriate suggestion to the General Practitioner that the results of genetic testing should not be entered into the medical notes of Patient F, the Tribunal do not find that Dr Mouton failed to communicate appropriately with Patient F’s General Practitioner in this regard.

468. The Tribunal therefore finds paragraph 38c of the Allegation not proved.

Patient G

Allegation 39

39. Between 17 November 2014 and 16 December 2015 you consulted with Patient G and you referred him for the following investigations:

   c. FUT2 genotype testing.  
   Found not proved

469. The Tribunal considered the witness statement of Dr Mouton:

   “I did not test Patient G for FUT2 genotype because my Responsible Officer’s (RO) recommendation. As soon as I heard about concerns on behalf of the boys’ GP (forwarded by their mother, even before I knew about the existence of a formal complaint). I have contacted Dr EE, my Responsible Officer at the Independent Doctors Federation (IDF). He has not immediately been able to answer, but after enquiry, his final recommendation was to limit my genomic prescriptions for children. which I have thus immediately adopted.”

470. The Tribunal noted that there was no evidence before it to demonstrate that this test had been carried out.

471. The Tribunal therefore finds paragraph 39c of the Allegation not proved.
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Allegation 40

40. The investigations referred to in paragraph 39 were:

a. not clinically indicated;

   Found proved in relation Apo E genotype testing
   Found proved in relation to DIO2 genotype testing
   Found not proved in relation to FUT2 genotype testing

Apo E genotype testing
Found proved

472. The Tribunal considered Dr Mouton’s witness statement, which stated:
“As already explained for previous patients. I value this genomic test to help me personalize my dietary advice. Given patient G’s E3/E4 genotype and presence of one E4 allele. I have advised a balanced diet without excessive saturated fats.”

473. The Tribunal noted that Dr Mouton had recorded in Patient G’s medical record that Patient G “always struggles to put on weight”.

474. The Tribunal noted that Patient G’s medical records included growth charts showing his height and weight were well within the normal expected range. Accordingly, unlike Patient F, there was no clinical picture of Patient G failing to thrive. In the circumstances, there was no clinical indication to carry out the Apo E test while Patient G was still a child.

475. The Tribunal therefore finds paragraph 40a in relation to paragraph 39a of the Allegation proved.

DIO2 genotype testing
Found proved

476. For the reasons outlined in relation to Patient F, the Tribunal found the DIO2 genotype testing was not clinically indicated.

477. The Tribunal therefore finds paragraph 40a in relation to paragraph 39b of the Allegation proved.

FUT2 Genotype testing
Found not proved

478. Dr Mouton did not carry out this test on Patient G. Therefore the Allegation that FUT2 genotype testing was not clinically indicated was found not proved.
479. The Tribunal therefore find paragraph 40a in relation to paragraph 39c of the Allegation not proved.

   b. conducted without obtaining informed consent from Patient G and/or from his parent or guardian;
   Found proved in relation to Apo E and DIO2 genotype testing
   Not applicable for FUT2 genotype testing

   c. conducted without appropriate counselling of Patient G and/or his parent or guardian;
   Found proved in relation to Apo E and DIO2 genotype testing
   Not applicable for FUT2 genotype testing

480. For the reasons outlined in relation to Patient F, the Tribunal found the Apo E and DIO2 genotype testing was conducted without obtaining informed consent from and without appropriate counselling of Patient G and/or his parent or guardian.

481. The Tribunal have already determined that FUT2 genotype testing was not undertaken. As such these Allegations are not applicable.

482. The Tribunal therefore finds paragraphs 40b and c in relation to paragraphs 39a and b of the Allegation proved.

Allegation 41

41. You failed to adequately interpret or act upon the findings that Patient G was biochemically euthyroid.
   Found proved

483. The Tribunal first considered Dr Mouton's witness statement:

   “Many symptoms presented by Patient G can be related to low thyroid function, even though not surprisingly unspecific. I list the following: fatigue, needs a lot of sleep (10 ½ hours and would need more); cold hands and feet; very sensitive to cold; very dry skin; poor memory/concentration; ‘never’ sweats; low temperature (systematically less than 36°C when taken early in the morning, still in bed). Patient G presented with ‘grey zone’ TSH levels in his three sets of tests: on 21 January 2014 with 3.77mU/L… on 10 June 2014 with 2.58 mU/L… and on 20 December 2014 with 2.63 mU/L… Such a combination of clinical and biological data cannot guarantee Patient G was euthyroid. His third test has triggered my decision to implement only ¼ grain of ARMOUR THYROID. i.e a lower dosage than ½ grain given his younger
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brother Patient F, because in this case DIO2 genotype showed homozygous wild. This meant that he is not prone to experience low T3 levels, which implies less support from T3-containing natural desiccated thyroid. I have prescribed 2.25 mcg of T3. I have used ARMOUR THYROID for these young patients for good reasons: a) it comes in very small strengths impossible to reach with prescriptive tablets: b) it is usually very well tolerated and better suits children. I could have recommended a food supplement called THYROPLEX readily available in London health shops which supplies ¼ grain of natural desiccated thyroid as well, but without the need for prescription. I did not opt for this product, which would have protected me from allegations of prescribing thyroid hormones to children because THYROPLEX shows twice more expensive. I take into account that this family has a limited budget.”

484. The Tribunal went on to consider the oral evidence from Dr Mouton:

“...That is pretty grey and to American standards that is not normal, but I did not treat with hormones at that stage. The way I formulate this is that altogether, the biology and the symptoms make me think that I cannot guarantee Patient G was euthyroid. That is my way to say it, because I fully accept the fact that they are not hypothyroid. They are somewhere in the continuum but not so far from being hypothyroid, which is why I treat at some point third cycle of treatments after months and months of some deceiving outcome with my interventions, I start treating them with a quarter grain, this one a quarter grain, and the brother who was worse, half a grain. It is still small amounts...

...A Little thing, very briefly because I realise I have now forgotten to cover the theme of iodine. It is interesting the theme of iodine, because I have given some iodine to this boy. Iodine, I am perfectly well aware that official guidelines at least in this country, not in Belgium but in this country, I know that iodine is not recommended, and the American Thyroid Association as well does not recommend giving iodine to thyroid patients, which I fully support. I fully agree with that because in fact what happens is that you cannot give iodine to thyroid patients without, in my opinion, without demonstrating that there is an iodine deficiency. The only indication for iodine is that is if iodine is deficient.”

485. The Tribunal noted the joint expert report from Professor Q and Dr S:

"Prof Q and Dr S agreed that this patient was euthyroid.”

486. The Tribunal considered Professor Q’s oral evidence:

"I think there were perhaps some reservations about if you take a patient (who I thought was euthyroid) and giving thyroid hormone to, say, the
children in F and G, it does strike me that there is some concern about possible over-treatment if you carry on with that in a young person. Coming back to your point to me, nobody was at any serious risk here.”

And further:

“Q …as you point out this patient was prescribed with Armour Thyroid and you say most paediatric endocrinologists would not have diagnosed hypothyroidism or treated this child with thyroid hormones. Would any competent medical practitioner have treated this child with Armour Thyroid in these circumstances?

A I do not believe that they would, no.”

487. The Tribunal accepted the joint expert report from Professor Q and Dr S in which they both agreed that Patient G was biochemically euthyroid. The Tribunal noted that Dr Mouton first prescribed Armour Thyroid, which contained both T3 and T4, in January 2015. It further noted the previous two TSH results were both well within the accepted range and had also dropped from the result that had been first obtained in January 2014. The Tribunal determined that this prescription was not an adequate response to the results that had been obtained up to that point in time.

488. The Tribunal therefore finds paragraph 41 of the Allegation proved.

Allegation 42

42. You prescribed thyroid hormone treatment, Armour thyroid, to Patient G which treatment was not:

a. clinically indicated;  
   **Found proved**

b. supported by scientific guidelines;  
   **Found proved**

c. evidence based.  
   **Found proved**

489. The Tribunal accepted the agreed view of Professor Q and Dr S that Patient G was euthyroid. It concluded that it could see no evidence before it in the guidelines to support treating a paediatric patient who is euthyroid with Armour Thyroid. It further noted that the use of combination therapy, a category in which Armour Thyroid falls, is not supported by the guidelines.

490. For these reasons and those set out in paragraph 41 of the Allegation, the Tribunal finds that the treatment of Armour Thyroid in the case of Patient G was not clinically indicated, supported by scientific guidelines, or evidence based.
491. The Tribunal therefore finds paragraph 42a, b and c of the Allegation proved.

**Allegation 43**

43. You failed to communicate appropriately with Patient G’s General Practitioner in that you:

   a. did not include information in your letters to Patient G’s General Practitioner regarding his:

      i. previous and/or new diagnoses; \(\text{Found not proved}\)
      ii. key results; \(\text{Found not proved}\)
      iii. investigations; \(\text{Found not proved}\)
      iv. treatment plans. \(\text{Found not proved}\)

492. The Tribunal noted that as with regard to Patient F, Dr Mouton did send a letter to Patient G’s General Practitioner which included his annotations on the lab results and reference to fungal dysbiosis and leaky gut syndrome amongst other possible diagnoses. It therefore concluded that there is some form of diagnosis indicated in his correspondence to Patient G’s General Practitioner.

493. The Tribunal also noted that Dr Mouton’s letter to Patient G’s General Practitioner did include information about test results, investigations and treatment plans.

494. The Tribunal therefore finds paragraph 43ai, ii, iii and iv of the Allegation not proved.

   b. failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing to which you referred Patient G, namely:

      i. why the tests were carried out; \(\text{Found proved}\)
      ii. the degree of explanation given to Patient G’s parent and/or Patient G prior to taking the test; \(\text{Found proved}\)
      iii. any implications that the General Practitioner should be aware of arising from the results of the genetic testing; \(\text{Found proved}\)
The Tribunal determined that for the same reasons outlined in relation to Patient F Dr Mouton failed to respond adequately or appropriately to the General Practitioner’s questions regarding genetic testing.

The Tribunal therefore finds paragraph 43b i, ii and iii of the Allegation proved.

c. suggested that the results of the genetic testing that you sent to the General Practitioner should not be entered into the medical notes of Patient G.  **Found not proved**

The Tribunal determined that for the same reasons outlined in relation to Patient F Dr Mouton did not fail to communicate appropriately with Patient G’s General Practitioner by suggesting that the results of the genetic testing should not be entered into Patient G’s medical notes.

The Tribunal therefore finds paragraph 43c of the Allegation not proved

**Patient H**

**Allegation 45**

45. The investigations referred to in paragraph 44 were:

a. not clinically indicated;
   **Found proved in relation to Apo E and DIO2 genotype testing**

**Apo E genotype testing**

**Found proved**

499. The Tribunal first considered Dr Mouton’s witness statement:

“I am fond of testing this polymorphism because of its profound implications regarding dietary guidance that I systematically provide to all patients. In this case, E3/E3 genotype shows compatible with low-carb high-fat diet but I always take other factors into account, such as cholesterol that shows perfectly normal here. Therefore, I have also focused on improving the blood glucose stability in order to reduce excessive HbA1c level, knowing that 5.8% corresponds to the threshold for prediabetes.”

500. The Tribunal noted the joint expert report from Professor Q and Dr S:

“Prof Q and Dr S agreed:
The investigations were potentially justified in the context of a full health check and functional medicine context in the private sector.

However, Professor Q and Dr S’s opinions differed in that while Professor Q acknowledged the plausibility that genotyping information might be relevant to treatment decisions, he also considered the evidence that the genotyping information really altered patient management was very limited and therefore he questioned the use of these tests. Nevertheless, Dr S considered that the tests were still justifiable and might enhance clinical treatment decisions. He also considered that the freedom of choice to choose tests and to apply the results to treatments was an important principle.”

501. The Tribunal noted Dr Mouton’s assertion that he was fond of this test and that he systematically provided it for all his patients given the dietary benefits. It also noted his evidence that this was one of the reasons patients would see him rather than a mainstream General Practitioner.

502. The Tribunal had regard to the fact Patient H presented to Dr Mouton on 18 February 2016 with a recurrent cough and chest infection at which point he suggested to her that he would like to run some genotype tests.

503. The Tribunal noted that Patient H’s primary presenting complaint was of a recurrent chest infection, it also noted that from the medical records that Patient H did not appear to be overweight or underweight for her height. It accepted that the consultation would also have been in the context of Dr Mouton considering Patient H’s health in more general terms. However, the details of the medical history Dr Mouton took from Patient H on 18 February 2016 did not reveal any issues with Patient H’s diet. Accordingly, the Tribunal concluded that the Apo E test was not clinically indicated in this case.

504. The Tribunal therefore finds paragraph 45a in relation to paragraph 44a of the Allegation proved.

DIO2 genotype testing
Found proved

505. For the reasons outlined with regard to Patient A, the Tribunal found the investigation DIO2 genotype testing was not clinically indicated.

506. The Tribunal therefore finds paragraph 45a in relation to paragraph 44b of the Allegation proved.

b. conducted without obtaining informed consent from Patient H;
   Found proved in relation to Apo E genotype testing
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Found not proved in relation to DIO2 genotype testing

c. conducted without appropriate counselling of Patient H;

Found proved in relation to Apo E genotype testing

Found not proved in relation to DIO2 genotype testing

Apo E genotype testing

507. The Tribunal considered that Dr Mouton’s rationale for conducting the Apo E test related solely to dietary issues. Whilst the Tribunal noted there is a signed consent form dated 18 February 2016, nowhere in Dr Mouton’s notes does he indicate that he has specifically discussed the potential implications of this test with respect to Patient H’s risk to Alzheimer’s disease or the potential risk to other members of her family. Given that Patient H was 67 years old and given that she complained of poor concentration and short term memory, the Tribunal would have expected Dr Mouton to have made such notes, had such a discussion taken place. Notwithstanding Dr Mouton’s evidence as to his general practice on this issue, in the absence of a specific note, the Tribunal concluded, on the balance of probabilities that the discussion did not take place and accordingly that Dr Mouton did not obtain informed consent from Patient H.

508. The Tribunal noted that in the result of Patient H’s test, there was no increased risk of Alzheimer’s disease. However, there was no entry in Dr Mouton’s records that he had explained the results to Patient H and given her reassurance. Again, the Tribunal considered that if the conversation had taken place Dr Mouton could and should have made a note of it. In the absence of such a note the Tribunal concluded, on the balance of probabilities, that Dr Mouton did not provide appropriate counselling to Patient H.

509. The Tribunal therefore finds paragraphs 45b and c in relation to paragraph 44a of the Allegation proved.

DIO2 genotype testing

510. The Tribunal considered that Dr Mouton had given oral evidence about his general approach regarding consent and counselling for this particular test. There was also evidence of a signed consent form for this patient. Given these circumstances, and for the reasons outlined in respect of Patient C the Tribunal found that the DIO2 genotype testing was carried out with the informed consent and appropriate counselling of Patient H.

511. The Tribunal therefore finds paragraphs 45b and c in relation to paragraph 44b of the Allegation not proved.
Allegation 46

46. You failed to adequately interpret or act upon:
   a. the findings that Patient H was biochemically euthyroid;

   Found proved

512. The Tribunal considered the witness statement of Dr Mouton:

   “This is precisely why I have not advised any prescribed thyroid hormones. I would like to make a point here regarding my GTA “supplementation” (you notice that I did not write my GTA “prescription”). The respected Endocrinologist(s) to whom I typically refer complex thyroid cases have never been bothered by such “supplementation”. Quite logically, they would only worry about prescriptive thyroid treatments, not about thyroid supplements.”

513. The Tribunal also noted Dr Mouton’s comments dated 27 March 2016 following the lab results on the 16 March 2016:

   “... stress clearly represents huge player here through the weakening inflicted to thyroid and adrenal glands as shown by poor conversion from the thyroid prohormones T4 into active hormones T3 on the one hand, and by lack of adrenal prohormone pregnenolone (precursor to ‘stress hormone’ cortisol and to progesterone).

   We can help through supplementing missing items in their natural / bioidentical forms: glandular with a bit of T3 (GTAEN, twice a day given the short T3 half-life) and compound capsules with pregnenolone...”

514. The Tribunal considered the joint expert report from Professor Q and Dr S:

   “We agreed that this patient was biochemically euthyroid. However, we also agree that hypothyroidism was not diagnosed.”

515. The Tribunal considered the joint expert report from Professor Q and Dr J:

   “We both agree that a certain degree of hypothyroidism may exist with thyroid tests within reference range (at borderline low T3, T4, levels, high-normal TSH levels), that there is a place for thyroid treatment of hypothyroidism with T3-T4 treatments (and not on thyroxine), and desiccated thyroid.

   We also agree that thyroid guidelines are partly designed for less experienced practitioners, and that thyroid treatments not included in the guidelines may be prescribed by more experienced physicians, on conditions that safety and
efficacy is adequate or can adequately be shown. We both agree that there is scientific evidence.

Professor Q considered that Patient H was euthyroid, and that symptoms were unlikely to be thyroid related. He attached most importance to the TSH test result of 1.0, which is clearly well in the normal range. Dr J argued the case for her having mild hypothyroidism and at least justifying a trial of thyroid treatment.”

516. The Tribunal accepted the opinions of Professor Q and Dr S that Patient H was biochemically euthyroid. It noted that this is confirmed by the results set out on 16 March 2016 where all of the blood endocrinology results are well within the normal range. Notwithstanding this, Dr Mouton had interpreted the results as showing that “through the weakening inflicted to thyroid and adrenal glands as shown by poor conversion from the thyroid prohormones T4 into active hormones T3 on the one hand”, and had treated Patient H with GTA which contains T3 and T4. The Tribunal concluded therefore that Dr Mouton did not adequately interpret or act upon the findings that Patient H was biochemically euthyroid.

517. The Tribunal therefore finds paragraph 46a of the Allegation proved
   b. Patient H’s steroid blood results.
      Found proved

518. The Tribunal noted Dr Mouton’s response to this Allegation in his witness statement:

“I have identified a lack of pregnenolone and progesterone thanks to blood measurements. Typical Functional Medicine strategy will always consist in restoring physiological balance by natural means, thus prescribing (in Europe) or just supplementing (in the US where it is considered as a food supplement) pregnenolone represents a common practice, including by US Consultants in Internal Medicine. This should not surprise us given that pregnenolone represents the mother prohormone for all steroids. I use the word "prohormone" because no receptor has ever been identified for pregnenolone, thus we cannot really consider it as a classic hormone. The GMC expert uses the word “intermediary product in the synthetic pathway for cortisol and other steroids”, which basically mean the same thing. The problem here also is that I cannot prescribe estradiol alone in the absence of progesterone support for a menopausal woman who has not undergone hysterectomy, which is Patient H’s situation. The expert should have stressed that he recommends progesterone prescription at the side of estradiol prescriptions, rather than just blaming me for prescribing pregnenolone. Alternately, he could have criticized both estradiol and pregnenolone prescriptions, but he did not come to that conclusion either. My pregnenolone
prescriptions usually restore protective levels of progesterone and that should not be surprizing because it takes only one enzymatic reaction.”

519. The Tribunal noted the joint expert report from Professor Q and Dr J:

“These questions have been discussed earlier in the global evaluations. We both agree that in private practise, there is more freedom for different testing and urinary steroid tests have justifications.”

520. The Tribunal went on to consider the expert report from Professor Q:

“...Progesterone is a steroid hormone produced mainly by the ovaries in premenopausal women and with important physiological functions in pregnancy. However, it is uncertain whether post-menopausal progesterone deficiency at the age of 67 is of any clinical significance. While Dr Mouton also mentions that pregnenolone is required for the production of cortisol, because it is an intermediary product in the synthetic pathway for cortisol and other steroids, it is noted that the test result for 17 hydroxysteroids (this is considered to be a test for cortisol and corticosterone production) is elevated, which if anything suggests an excess, rather than any deficiency of cortisol. Thus, regarding pregnenolone supplementation, Dr Mouton seems to be making an argument that is not supported by the results of the test he has requested. In summary, I do not see these results as indicative of poor T4 to T3 conversion or insufficiency of the hormones cortisol and progesterone.

- Dr Mouton justified the use of T3 and pregnenolone on the basis of these test results.
- Dr Mouton advises "bio-identical safe estradiol". Here, Dr Mouton is advising estrogen and progesterone supplementation as an approach to postmenopausal sex hormone replacement aiming to restore mucosal health.
- Dr Mouton advises a series of vitamin, mineral and herbal treatments to enhance thyroid, adrenal and mucosal function…”

And further:

“Steroid hormone measurements

Dr Mouton also obtained measurements of pregnenolone, progesterone and adrenal steroid production (17 hydroxycorticosteroids). However none of these results suggested adrenal insufficiency or progesterone deficiency. Indeed the 17 hydroxy steroids were elevated, which is the reverse of what might be expected with adrenal insufficiency. It is not clear why Dr Mouton
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sees evidence for inadequate cortisol and progesterone production from these results, and endocrinologists would not interpret the results in this way. If Dr Mouton is not using these results to make decisions it is unclear why he is arranging these measurements in the first place. Therefore, it is my opinion Dr Mouton also did not adequately and appropriately interpret or act upon these steroid blood results.”

521. The Tribunal considered the opinion of Professor Q who suggested that Patient H’s blood steroid results were normal and within the usual range, especially in the context that Patient H was a 67 year old post-menopausal woman. Dr Mouton interpreted these results as indicating a “weakening inflicted to thyroid and adrenal glands as shown by poor conversion from the thyroid prohormones T4 into active hormones T3 on the one hand”. The Tribunal considered that Dr Mouton had interpreted Patient H’s test results as showing an “underperforming adrenal gland” and a lack of adrenal prohormone and he prescribed pregnenolone. The Tribunal accepted the expert evidence of Professor Q that this was an incorrect interpretation and that Dr Mouton’s prescriptions as a result were not justified. It therefore concluded that Dr Mouton failed to adequately interpret or act upon Patient H’s steroid blood results.

522. The Tribunal therefore finds paragraphs 46b of the Allegation proved.

Allegation 47

47. You prescribed the treatments referred to in Schedule 6 which were not:

   a. clinically indicated;  
      Found proved  
   b. supported by scientific guidelines;  
      Found proved  
   c. evidence based.  
      Found proved

GTAEN natural thyroid extract

Found proved

523. The Tribunal heard expert evidence in relation to Patient E that GTA contains natural T3 and T4 thyroid hormone. In the light of the Tribunal’s conclusion in relation to paragraph 46 of the Allegation, it concluded that the prescribed treatment of GTAEN natural thyroid extract was not clinically indicated, supported by scientific guidelines or evidence based.

524. The Tribunal therefore finds paragraphs 47a, b and c of the Allegation proved with regard to GTAEN natural thyroid extract referred to in schedule 6.
Pregnenolone  
**Found proved**

525. In light of the Tribunal’s conclusion in respect of pregnenolone as already set out in this determination regarding other patients, the Tribunal concluded that the prescribed treatment of pregnenolone was not clinically indicated, supported by scientific guidelines or evidence based.

526. The Tribunal therefore finds paragraphs 47a, b and c of the Allegation proved with regard to pregnenolone referred to in schedule 6.

**Allegation 48**

48. You failed to make any diagnosis or differential diagnosis that explained Patient H’s presenting complaint, namely, a chronic cough.  
**Found not proved**

527. The Tribunal noted Dr Mouton’s response to this Allegation in his witness statement:

"The NHS Practice, as demonstrated in health records, has explored diagnosis and differential diagnosis of patient G’s (sic) chronic cough. She has passed an X-ray; she has refused the CT-scan (if I understood well, because of radiation risk); she has seen Consultant Respiratory Physician Dr FF on three occasions. First consultation occurred on 18 May 2015, second one 6 July 2015, and third one on 28th July 2015. The suggested solution consists in taking long courses of antibiotics, something patients become nowadays less and less happy to consider. Patient H has come to me to find another more effective route, surely not to repeat what she had already performed with her NHS Practice."

528. The Tribunal noted the comments made by Dr Mouton on 27 March 2016 following the lab test results:

"We find numerous means to boost immune defences from a Functional Medicine approach despite not spotting one isolated issue that would explain recurrent chest infections. In fact, this situation happens very often – not one major trigger but many minor ones – which is why holistic strategies help patients..."

529. The Tribunal noted the joint expert report from Professor Q and Dr J:

"We both agree that chronic cough might result from an impaired immune state, which can result from various factors including hypothyroidism and..."
nutritional deficiencies. Prof Q would have liked more differential diagnosis but understands that when a patient consults a functional medicine specialist he or she will be offered functional medicine treatments."

530. The Tribunal noted the joint expert report from Professor Q and Dr S:

"Dr S did not consider there was any failure to make a diagnosis or consider a differential diagnosis from a functional medicine standpoint.

Professor Q respected this opinion, although considered that the differential diagnosis of cough had probably not been fully considered.

However, we both agreed that seeking a functional medicine approach to improve certain aspects of health is still legitimate"

531. The Tribunal noted Dr Mouton’s comments dated 27 March 2016 in respect of Patient H as set out above. In his comments he refers to a deficient immune system and the possibility that there is no one major trigger but many minor ones which may be the cause of Patient H’s chest infection. The Tribunal also noted that during Dr Mouton’s examination of Patient H he also considered reflux as a cause.

532. The Tribunal determined that in the context of Dr Mouton being aware that Patient H had already had other investigations performed by other medical practitioners, Dr Mouton had made some diagnosis and differential diagnoses in respect of Patient H’s recurrent chronic cough.

533. The Tribunal therefore finds paragraphs 48 of the Allegation not proved.

Allegation 49

49. You failed to adequately or properly communicate to Patient H or her General Practitioner:

   c. your diagnosis;  
      **Found Proved**

   d. why you had prescribed the treatments referred to in Schedule 6.  
      **Found Proved**

534. The Tribunal considered the letter from Dr Mouton to Dr GG (Patient H’s General Practitioner), dated 22 April 2016:

   "I saw your patient today, thus I wish to keep you informed about the evolution within my practice."
I therefore send you a copy of the results as well as the report that I have besides handed over to your patient during today’s appointment. The patient has received prescriptions and dietary recommendations for a period of 4 months and has booked a follow-up appointment in my practice in 3 months.

I am of course available for any further information that you may wish to receive.”

535. The Tribunal considered Dr Mouton’s oral evidence:

“Q Would it not be, apart from anything else, a simple courtesy to say, “Dear Dr GG, I am prescribing a number of functional medicine treatments that are designed to address this patient’s fatty acid deficiencies as follows: Borage, fish oil”, and so on?

A Yes.

Q Would that not make it obvious to the doctor what you were prescribing and why?

A Yes, there is certainly a way to improve that understanding, I fully agree with you, and that is certainly something to consider. However, I will also stress the fact that since I have been sending new reports to new GPs who deal with new patients, Dr GG is the only one who has reacted so negatively. Sometimes the doctors follow what I invite them to do, is to come back to me to get more information, and I am always very diligent to provide that, but your suggestion is certainly something I will take into consideration because even without necessarily spending four hours on a report, there is probably some way to make that a bit more better introduced and increase the acceptance from the GPs, so that is clearly a lesson from this hearing. I am here to learn and to explain and I hope I have explained, but I am certainly here to learn.”

536. The Tribunal noted that Dr Mouton’s letter to Dr GG in respect of Patient H, dated 22 April 2016, which enclosed test results, prescriptions and dietary recommendations made by Dr Mouton was effectively a proforma. The letter itself contained no diagnosis and no explanation for the prescriptions made by Dr Mouton.

537. The Tribunal concluded it would not have been obvious to a General Practitioner what Dr Mouton’s diagnoses were or the rationale for his treatment regimen. It noted that GMP places a duty on Dr Mouton to communicate clearly with other practitioners who are treating the patient; simply enclosing the results and the prescriptions was not adequate communication with Patient H’s General Practitioner.
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538. The Tribunal therefore finds paragraphs 49c and d of the Allegation proved.

The Tribunal’s Overall Determination on the Facts

539. The Tribunal has determined the facts as follows:

**Patient A**

1. On various dates between 10 April 2013 and 18 November 2014, you consulted with Patient A and you:

a. failed to elicit from Patient A adequate information about:

   i. possible drug misuse;  
      **Found proved**

   ii. decline in academic performance;  
       **Found not proved**

   iii. depressed mood;  
        **Found not proved**

   iv. social withdrawal;  
       **Found not proved**

   v. paranoid symptoms;  
      **Found not proved**

b. failed adequately to assess Patient A’s mental state;  
   **Found proved**

c. attributed Patient’s A’s symptoms to:

   i. hypothyroidism;  
      **Admitted and found proved**

   ii. nutritional issues;  
       **Admitted and found proved**

   iii. obsessive compulsive disorder;  
        **Found not proved**

   iv. leaky gut syndrome;  
      **Admitted and found proved**
d. failed to make a differential diagnosis of a psychiatric disorder in light of Patient A’s presenting symptoms.  **Found proved**

2. You referred Patient A for the investigation of:
   a. urinary thyroid hormones;  
      **Admitted and found proved**
   b. steroid hormones;  
      **Admitted and found proved**
   c. insulin;  
      **Admitted and found proved**
   d. DIO2 genotype testing.  
      **Admitted and found proved**

3. The investigations referred to in paragraph 2 were not clinically indicated.

   Urinary thyroid hormones  **Found proved**
   Steroid hormones  **Found not proved**
   Insulin  **Found not proved**
   DIO2 genotype testing  **Found proved**

4. You prescribed dietary treatments for Patient A which were not:
   a. clinically indicated;
      Iodine  **Found proved**
      Selenium  **Found proved**
      Otherwise  found not proved
   b. supported by scientific guidelines;
      Iodine  **Found proved**
      Selenium  **Found proved**
      Otherwise  found not proved
   c. supported by sound scientific evidence (“not evidence-based”).
      Iodine  **Found proved**
      Selenium  **Found proved**
      Otherwise  found not proved
5. You failed to communicate with Patient A’s General Practitioner regarding Patient A’s:
   a. diagnosis of autoimmune thyroid disease with hypothyroidism; **Found not proved**
   b. prescriptions for thyroxine and T3 therapy. **Found not proved**

6. In an email to Patient A’s father dated 15 December 2014, you:
   a. advised him that psychiatrists ‘too often react with a very heavy chemical load which I find dangerous and sometimes definitely destructive’; **Admitted and found proved**
   b. failed to communicate clearly with Patient A’s father, in that you provided contradictory advice regarding referring Patient A to a psychiatrist. **Found proved**

**Patient B**

7. Between 1 September 2014 and 1 September 2015 you consulted with Patient B and you referred Patient B for the following investigations:
   a. CTLA-4 genotype testing; **Admitted and found proved**
   b. VDR genotype testing. **Admitted and found proved**

8. The investigations referred to in paragraph 7 were:
   a. not clinically indicated;
      
      CTLA-4 genotype testing **Found proved**  
      VDR genotype testing **Found not proved**  
   b. conducted without obtaining informed consent from Patient B;
      
      CTLA-4 genotype testing **Found not proved**  
      VDR genotype testing **Found not proved**  
   c. conducted without appropriate counselling of Patient B.
      
      CTLA-4 genotype testing **Found not proved**
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VDR genotype testing    Found not proved

9. You referred Patient B for investigation of:
   a. urinary thyroid hormones;
      Admitted and found proved
   b. steroid hormones;
      Admitted and found proved
   c. insulin.
      Admitted and found proved

10. The investigations referred to in paragraph 9 were not clinically indicated.

Urinary thyroid hormones    Found proved
steroid hormones            Found not proved
insulin                     Found not proved

11. You attributed Patient B’s symptoms of acute lethargy to hypothyroidism when there was no sound clinical basis for doing so.
    Found not proved

12. You prescribed Patient B the treatments set out in Schedule 1, which were not:
   a. clinically indicated;
      Iodine                     Found proved
      Selenium                  Found proved
      Novothyral                Found proved
      Pregnenolone              Found proved
      All other items in schedule 1    Found not proved
   b. supported by scientific guidelines;
      Iodine                     Found proved
      Selenium                  Found proved
      Novothyral                Found proved
      Pregnenolone              Found proved
      All other items in schedule 1 Found not proved
   c. evidence based.
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Iodine                       Found proved
Selenium                     Found proved
Novothyral                   Found proved
Pregnenolone                 Found proved

All other items in schedule 1  Found not proved

13. You failed to communicate adequately with Patient B’s General Practitioner regarding:

a. Patient B’s diagnoses, including:

i. hypothyroidism;            Found proved

ii. iodine deficiency;        Found proved

iii. hypogonadism;            Found proved

b. Patient B’s prescriptions as set out in Schedule 1.  
   Found proved

Patient C

14. Between 15 December 2013 and 22 September 2015, you consulted with Patient C and you failed to record:

a. any clear diagnosis;        Found proved

b. any differential diagnoses. 
   Found not proved

15. You referred Patient C for the investigations set out in Schedule 2. 
   Admitted and found proved

16. The investigations referred to in Schedule 2 were:

a. not clinically indicated;

   DIO2 genotype testing  Found proved
   CTLA-4 genotype test   Found proved
Apo E genotype testing  Found not proved
MTHFR genotype testing  Found not proved
LCT genotype testing  Found not proved
FUT2 genotype testing  Found not proved
VDR genotype testing  Found not proved

b. conducted without obtaining informed consent from Patient C;

Apo E genotype testing  Found proved
MTHFR genotype testing  Found proved
LCT genotype testing  Found not proved
FUT2 genotype testing  Found not proved
VDR genotype testing  Found not proved
DIO2 genotype testing  Found not proved
CTLA-4 genotype test  Found not proved

c. conducted without appropriate counselling of Patient C.

Apo E genotype testing  Found proved
MTHFR genotype testing  Found proved
LCT genotype testing  Found not proved
FUT2 genotype testing  Found not proved
VDR genotype testing  Found not proved
DIO2 genotype testing  Found not proved
CTLA-4 genotype test  Found not proved

17. You referred Patient C for:

   a. investigation of urinary thyroid hormones;
      Admitted and found proved

   b. thyroid ultrasound scan.
      Admitted and found proved

18. The investigations referred to in paragraph 17 were not clinically indicated.

   Investigation of urinary thyroid hormones  Found proved
   Thyroid ultrasound scan  Found proved

19. You prescribed Patient C the treatments set out in Schedule 3, which were not:
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a. clinically indicated;
   Iodine & Tyrosine       Found proved
   Pregnenolone           Found proved
   Selenium and selenomethionine Found proved
   Armour Thyroid and l-thyroxine Found proved
   All other items in schedule 3 Found not proved

b. supported by scientific guidelines;
   Iodine & Tyrosine       Found proved
   Pregnenolone           Found proved
   Selenium and selenomethionine Found proved
   Armour Thyroid and l-thyroxine Found proved
   All other items in schedule 3 Found not proved

c. evidence based.
   Iodine & Tyrosine       Found proved
   Pregnenolone           Found proved
   Selenium and selenomethionine Found proved
   Armour Thyroid and l-thyroxine Found proved
   All other items in schedule 3 Found not proved

20. You failed to communicate adequately with:

   a. Patient C regarding her therapeutic treatment plan;
      Found not proved

   b. Patient C’s General Practitioner regarding the tests referred to in
      Schedule 2 and the implications of them;
      Found proved

   c. Patient C’s treating Gastroenterologist regarding the intestinal
      and dietary treatments referred to in Schedule 3.
      Found proved

Patient D

21. Between 14 January 2014 and 9 July 2015, you consulted with Patient D and you referred Patient D for the following investigations:
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a. investigation of urinary thyroid hormones;  
   **Admitted and found proved**

b. thyroid ultrasound scan.  
   **Admitted and found proved**

22. The investigations referred to in paragraph 21 were not clinically indicated.

   Investigation of urinary thyroid hormones  **Found proved**
   Thyroid ultrasound scan  **Found proved**

23. You referred Patient D for the investigation of DIO2 genotype testing and such investigation was:

   a. not clinically indicated;  
      **Found proved**

   b. conducted without obtaining informed consent from Patient D;  
      **Found not proved**

   c. conducted without appropriate counselling of Patient D.  
      **Found not proved**

24. You failed to adequately interpret or act upon the findings that Patient D was biochemically euthyroid.  
   **Found proved**

25. You prescribed the treatments set out in Schedule 4 which were not:

   a. clinically indicated;

      Pregnenolone  **Found proved**
      Novothyral  **Found proved**
      All other items in schedule 4  **Found not proved**

   b. supported by scientific guidelines;

      Pregnenolone  **Found proved**
      Novothyral  **Found proved**
      All other items in schedule 4  **Found not proved**

   c. evidence based.

      Pregnenolone  **Found proved**
      Novothyral  **Found proved**
All other items in schedule 4 Found not proved

Patient E

26. Between 3 September 2014 and 20 August 2015, you consulted with Patient E and you referred Patient E for the following investigations:

   a. investigation of urinary thyroid hormones;  
      Admitted and found proved
   
   b. thyroid ultrasound scan.  
      Admitted and found proved

27. The investigations referred to in paragraph 26 were not clinically indicated.

   Investigation of urinary thyroid hormones Found proved
   Thyroid ultrasound scan Found proved

28. You referred Patient E for the following investigations:

   a. DIO2 genotype testing;  
      Admitted and found proved
   
   b. Apo E genotype testing;  
      Admitted and found proved
   
   c. CTLA-4 genotype testing.  
      Admitted and found proved

29. The investigations referred to in paragraph 28 were:

   a. not clinically indicated;
      
      DIO2 genotype testing Found proved
      Apo E genotype testing Found not proved
      CTLA-4 genotype testing Found proved
   
   b. conducted without obtaining informed consent from Patient E;
      
      DIO2 genotype testing Found not proved
      Apo E genotype testing Found proved
      CTLA-4 genotype testing Found not proved
   
   c. conducted without appropriate counselling of Patient E.
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DIO2 genotype testing  Found not proved
Apo E genotype testing  Found proved
CTLA-4 genotype testing  Found not proved

30. You failed to adequately interpret or act upon the findings that Patient E was biochemically euthyroid.
Found proved

Found not proved

32. You prescribed the treatments referred to in Schedule 5 which were not:
   
a. clinically indicated;
   
   Thyrocsin  Found proved
   Tirform  Found proved
   Pregnenolone  Found proved
   GTA  Found proved
   Novothyral  Found proved
   L-selenomethionine  Found proved

   All other items in schedule 5  Found not proved

b. supported by scientific guidelines;

   Thyrocsin  Found proved
   Tirform  Found proved
   Pregnenolone  Found proved
   GTA  Found proved
   Novothyral  Found proved
   L-selenomethionine  Found proved

   All other items in schedule 5  Found not proved

c. evidence based.

   Thyrocsin  Found proved
   Tirform  Found proved
   Pregnenolone  Found proved
   GTA  Found proved
   Novothyral  Found proved
   L-selenomethionine  Found proved
All other items in schedule 5  Found not proved

33. You failed to communicate adequately with:
   
a. Patient E’s General Practitioner regarding the tests referred to in paragraph 28 and the implications of them;  
   Found proved
   
b. a Gastroenterologist regarding the intestinal treatments you intended to prescribe.  
   Found not proved

Patient F

34. Between 17 November 2014 and 16 December 2015 you consulted with Patient F and you referred him for the following investigations:
   
a. Apo E genotype testing;  
   Admitted and found proved
   
b. DIO2 genotype testing;  
   Admitted and found proved
   
c. FUT2 genotype testing.  
   Admitted and found proved

35. The investigations referred to in paragraph were:
   
a. not clinically indicated;
      
      Apo E genotype testing  Found not proved
      DIO2 genotype testing  Found proved
      FUT2 genotype testing  Found not proved
   
b. conducted without obtaining informed consent from Patient F and/or from his parent or guardian;  
   Found proved
   
c. conducted without appropriate counselling of Patient F and/or his parent or guardian.  
   Found proved

36. You failed to adequately interpret or act upon the findings that Patient F was biochemically euthyroid.  
   Found proved
37. You prescribed thyroid hormone treatment, Armour thyroid, to Patient F which treatment was not:
   a. clinically indicated;  
      **Found proved**
   b. supported by scientific guidelines;  
      **Found proved**
   c. evidence based.  
      **Found proved**

38. You failed to communicate appropriately with Patient F’s General Practitioner in that you:
   a. did not include information in your letters to Patient F’s General Practitioner regarding his:
      i. previous and/or new diagnoses;  
         **Found not proved**
      ii. key results;  
          **Found not proved**
      iii. investigations;  
          **Found not proved**
      iv. treatment plans;  
          **Found not proved**
   b. failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing to which you referred Patient F, namely:
      i. why the tests were carried out;  
         **Found proved**
      ii. the degree of explanation given to Patient F’s parent and/or Patient F prior to taking the test;  
         **Found proved**
      iii. any implications that the General Practitioner should be aware of arising from the results of the genetic testing;  
         **Found proved**
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c. suggested that the results of the genetic testing that you sent to
the General Practitioner should not be entered into the medical
notes of Patient F.  Found not proved

Patient G

39. Between 17 November 2014 and 16 December 2015 you consulted
with Patient G and you referred him for the following investigations:

a. Apo E genotype testing;
   Admitted and found proved

b. DIO2 genotype testing;
   Admitted and found proved

c. FUT2 genotype testing.
   Found not proved

40. The investigations referred to in paragraph 39 were:

a. not clinically indicated;

   Apo E genotype testing  Found proved
   DIO2 genotype testing  Found proved
   FUT2 genotype testing  Found not proved

b. conducted without obtaining informed consent from Patient G
   and/or from his parent or guardian;

   Apo E genotype testing  Found proved
   DIO2 genotype testing  Found proved
   FUT2 genotype testing  Not applicable

c. conducted without appropriate counselling of Patient G and/or
   his parent or guardian.

   Apo E genotype testing  Found proved
   DIO2 genotype testing  Found proved
   FUT2 genotype testing  Not applicable

41. You failed to adequately interpret or act upon the findings that Patient
   G was biochemically euthyroid.
   Found proved
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42. You prescribed thyroid hormone treatment, Armour thyroid, to Patient G which treatment was not:
   a. clinically indicated;  
      **Found proved**
   b. supported by scientific guidelines;  
      **Found proved**
   c. evidence based.  
      **Found proved**

43. You failed to communicate appropriately with Patient G’s General Practitioner in that you:
   a. did not include information in your letters to Patient G’s General Practitioner regarding his:
      i. previous and/or new diagnoses;  
         **Found not proved**
      ii. key results;  
         **Found not proved**
      iii. investigations;  
         **Found not proved**
      iv. treatment plans;  
         **Found not proved**
   b. failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing to which you referred Patient G, namely:
      i. why the tests were carried out;  
         **Found proved**
      ii. the degree of explanation given to Patient G’s parent and/or Patient G prior to taking the test;  
         **Found proved**
      iii. any implications that the General Practitioner should be aware of arising from the results of the genetic testing;  
         **Found proved**
   c. suggested that the results of the genetic testing that you sent to the General Practitioner should not be entered into the medical notes of Patient G.  
      **Found not proved**
Patient H

44. Between 18 February 2016 and 22 March 2016, you consulted with Patient H and you referred Patient H for the following investigations:
   a. APOE genotype testing;  
      Admitted and found proved
   b. DIO2 genotype testing.  
      Admitted and found proved

45. The investigations referred to in paragraph 44 were:
   a. not clinically indicated;
      APOE genotype testing Found proved
      DIO2 genotype testing Found proved
   b. conducted without obtaining informed consent from Patient H;
      APOE genotype testing Found proved
      DIO2 genotype testing Found not proved
   c. conducted without appropriate counselling of Patient H.
      APOE genotype testing Found proved
      DIO2 genotype testing Found not proved

46. You failed to adequately interpret or act upon:
   a. the findings that Patient H was biochemically euthyroid;  
      Found proved
   b. Patient H’s steroid blood results.  
      Found proved

47. You prescribed the treatments referred to in Schedule 6 which were not:
   a. clinically indicated;
      GTAEN natural thyroid extract Found proved
      Pregnenolone Found proved
   b. supported by scientific guidelines;
GTAEN natural thyroid extract  Found proved
Pregnenolone  Found proved
c. evidence based.

48. You failed to make any diagnosis or differential diagnosis that explained Patient H’s presenting complaint, namely, a chronic cough. Found not proved

49. You failed to adequately or properly communicate to Patient H or her General Practitioner:

a. why you had referred Patient H for the genetic tests referred to in paragraph 44; Admitted and found proved
b. the implications of the results of the genetic tests referred to in paragraph 44; Admitted and found proved
c. your diagnosis; Found proved
d. why you had prescribed the treatments referred to in Schedule 6. Found proved

DETERMINATION ON IMPAIRMENT - 04/07/2019

1. The Tribunal now has to decide in accordance with Rule 17(2)(l) of the Rules whether, on the basis of the facts which it has found proved, Dr Mouton’s fitness to practise is impaired by reason of misconduct.

The Outcome of Applications Made during the Impairment Stage

2. On day 35 of the hearing, Mr Ramasamy on behalf of Dr Mouton told the Tribunal that he wished to submit a testimonial bundle from colleagues and patients relevant to this case. Mr Atherton on behalf of the GMC, objected to its submission at this stage. Mr Ramasamy submitted that when determining whether a doctor’s fitness to practise is impaired, it must consider whether the misconduct is remediable, has been remediated and if there is a risk of repetition. He submitted
that the timespan of the facts in this case (April 2013 to March 2015) occurred more than three years ago and that the testimonials focus on Dr Mouton’s practice since 2016. He submitted the testimonials were directly relevant to whether Dr Mouton has remediated his practice. Mr Atherton submitted that the testimonials amounted to anecdotes and were irrelevant at this stage. He submitted that they may be relevant at stage 3, if at all. The Tribunal determined to admit the testimonial evidence at this stage in order to assess whether there was any material therein that was relevant to the issues outlined by Mr Ramasamy. The Tribunal considered that they would have had to read these testimonials in any event to determine if there was material relevant to stage 2. The Tribunal indicated that it would only consider the testimonials insofar as they are relevant to the issues before it at this stage.

The Evidence

3. The Tribunal has taken into account all the evidence received during the facts stage of the hearing, both oral and documentary. The Tribunal received oral evidence from Dr Mouton at the impairment stage and also received further documentary evidence on behalf of Dr Mouton which included, but was not limited to:

- Written statement by Dr Mouton, dated 27 June 2019;
- Care Quality Commission ('CQC') registrations;
- Clinical Governance Self Assessments, dated 28 April 2017, 15 June 2017 and 3 October 2017;
- Letters from Dr HH, Medical Director and Dr Mouton’s Responsible Officer, dated 2 November 2018; Dr BB, Consultant Physician and Endocrinologist, dated 5 September 2018; and, Dr II, Consultant Physician, Endocrinology and Diabetes, 10 June 2019;
- 360 Feedback - Colleagues and Patients, dated 2014 and 2018;
- Responsible Officer comments, dated 2015 and 2016;
- Continuous Professional Development ('CPD') evidence, dated 2013-2019;
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- Peer group, Schedule for Meetings and list of Attendees, 2017, 2018 and 2019;
- Confirmation of designation of FUNDEVMED Ltd;
- Queen Anne Street Medical Centre (‘QASMC’)/New Clinic Settings;
- QASMC Launch compliments;
- New communication process with General Practitioners for new patients;
- Note on functional medicine;
- Anonymised genetic consent forms;
- An updated CV of Dr Mouton;
- Testimonials from colleagues and patients.

Submissions on behalf of the GMC

4. In summary, Mr Atherton submitted that Dr Mouton had demonstrated a serious and persistent failure to follow Good Medical Practice (2013) (‘GMP’). He submitted that paragraphs 57, 65 and 68 of GMP are relevant.

5. Mr Atherton referred the Tribunal to the relevant legal principles. He submitted that this Tribunal has noted that Dr Mouton was referred to the GMC due to the care he provided. Mr Atherton referred to the test set out in CHRE v NMC and Grant [2011] EWHC 927 (Admin) : do the findings of fact show that Dr Mouton’s fitness to practise is impaired in the sense that: he has acted or is likely to act in the future so as to put patients at unwarranted risk of harm, he has brought the profession into disrepute, or he is likely to bring the profession into disrepute in the future or he has breached one of the fundamental tenets of the medical profession.

6. Mr Atherton submitted that Dr Mouton relies on his compliance with his interim order of conditions to persuade the Tribunal that his fitness to practise is not impaired. He submitted that the conditions were put in place to protect patients until this hearing and that compliance with his conditions does not demonstrate whether his fitness to practise is impaired or remediated.

7. Mr Atherton submitted that the Tribunal’s findings of fact fully reflect the case as it was outlined by Mr Sefton QC. He submitted that this has always been put as a pattern of similar facts with eight patients over a period of time, both individually and collectively as serious misconduct. He submitted that this is a doctor who is registered and licenced with the GMC who has seriously and persistently failed to follow the GMC’s guidance and other relevant guidance.
8. Mr Atherton submitted that patients who consult with practitioners of Functional Medicine are vulnerable patients with chronic illnesses who feel they have been failed by mainstream medicine. Mr Atherton submitted that the facts found proved raise concerns in relation to patient safety and that Dr Mouton’s conduct has placed these vulnerable patients at risk of harm.

9. Mr Atherton submitted that Dr Mouton has either demonstrated inadequate knowledge of the applicable guidelines and standards, or demonstrated a conscious disregard for them and used tests indiscriminately.

10. Mr Atherton submitted that had it not been for the alertness of the practitioners who brought these matters to the attention of the GMC, this practice would have continued and it may yet continue. He submitted that it is difficult to monitor a practitioner in the private sector practising Functional Medicine, supported by others who also have a passionate interest in Functional Medicine. He submitted that it has become clear during this hearing that Dr Mouton has sought to justify the use of his tests and treatments. He submitted that there are many aspects in this case that raise serious concerns about Dr Mouton’s practice.

Submissions on behalf of Dr Mouton

11. Mr Ramasamy submitted that, with regard to Mr Atherton’s submission inviting the Tribunal to consider paragraph 65 of GMP, there is and has been no allegation of dishonesty or lack of integrity. Mr Ramasamy accepted that paragraphs 57 and 68 of GMP were relevant.

12. Mr Ramasamy referred the Tribunal to Dr Mouton’s remediation statement, in which he accepted and respected the decision the Tribunal had reached at the facts stage. He referred the Tribunal to the testimonial evidence from colleagues and patients. He submitted that the picture that emerges is of a caring doctor who has made a difference to many lives where conventional medicine has not worked. He submitted that the testimonials provide evidence from the patients’ point of view and that there is no single way of practising medicine.

13. Mr Ramasamy submitted that the Tribunal criticised parts of Dr Mouton’s care but that patients have not complained. They say he has helped them or members of their families. Mr Ramasamy accepted that patients’ views were not decisive but submitted they were important when considering this case.

14. Mr Ramasamy submitted that the conditions imposed on Dr Mouton’s registration were to maintain a log of his thyroid prescribing practice and communication with General Practitioners. Mr Ramasamy submitted that Dr Mouton had broadened the scope of the logs to include patients within the UK and outside of
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the UK. Mr Ramasamy further submitted that the logs had been forwarded to the GMC. The GMC had not complained about their content.

15. Mr Ramasamy submitted that Dr Mouton had devised a document to explain the ethos and generic approach of Functional Medicine which he now sends to the relevant General Practitioner. He submitted that this innovation is an example of Dr Mouton’s insight.

16. Mr Ramasamy referred the Tribunal to Dr Mouton’s updated patient guide and reference sheet. He submitted that this is a living database which continues to grow and can be accessed by other General Practitioners. Mr Ramasamy submitted that the database explains Dr Mouton’s use of Thyroxine, his reason for prescribing it and his openness about using it. He submitted that the GMC had raised no complaints or concerns about Dr Mouton’s use of Thyroxine in the intervening four years. He submitted that Dr Mouton has demonstrated transparency and shown a willingness to open his practice to scrutiny. Mr Ramasamy submitted that Dr Mouton has voluntarily added a further level of scrutiny to that which was imposed by the interim order of conditions by seeking out supervision, from Dr BB, the final author of the Okosime paper.

17. Mr Ramasamy referred to the experts’ opinions in relation to Dr Mouton’s use of genomic testing and consent forms. He submitted that Dr Mouton’s practice was now better than many other practitioners who did not come before the GMC.

18. Mr Ramasamy referred to Dr Mouton’s new clinic setting at the QASMC. He submitted that this new centre provides Dr Mouton with the opportunity to interact and collaborate with other mainstream doctors and that he is no longer working in isolation. He submitted that Dr Mouton attends and runs peer group meetings, which is an indication of an open practice and provides an opportunity for communication with other practitioners.

19. Mr Ramasamy referred the Tribunal to Dr Mouton’s appraisals for 2016, 2017 and 2018 and his CPD. He submitted that when considering CPD, Dr Mouton has been learning throughout this case and the experience has been one of ongoing CPD and learning. He submitted that it has been four years since the conditions were first imposed and that Dr Mouton has complied with them during that period, read reports from experts, both for the GMC and the defence, and he has read scientific papers that both support and do not support his practice. Mr Ramasamy submitted that the Tribunal can take the appraisals and CPD as evidence of Dr Mouton’s remediation, reflection and insight.

20. Mr Ramasamy referred the Tribunal to the relevant legal principles and submitted that the facts found proved would have to amount to a serious departure from the standard expected in order to amount to misconduct. He submitted that not all the alleged facts had been found proved. Mr Ramasamy submitted this case
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centred on the standard of care and communication Dr Mouton provided for his patients. He invited the Tribunal to consider why Dr Mouton acted as he did and submitted that, like all doctors he aimed to help his patients and that there was no suggestion to the contrary. He submitted that Dr Mouton genuinely believed he was doing the right thing.

21. Mr Ramasamy referred the Tribunal to the relevant legal principles as set out in the case of *Schodlok [2011] EWHC 927 (ADMIN)*, on the ability to aggregate a series of non-serious failures to reach a cumulative finding of serious misconduct. He submitted that whilst this approach was open to the Tribunal, it should proceed with caution as these matters related to eight patients in a three year period starting in 2013. He invited the Tribunal to ask itself whether this was a very unusual case with a large number of unusual facts of the same or similar misconduct. He submitted that it is the defence position that this is not one of those cases.

22. Mr Ramasamy submitted that impairment involves looking at the position today rather than the past. He reminded the Tribunal that some of the events in question dated back as far as six years. He submitted that the conditions that were imposed by the Interim Orders Tribunal remain in place today and that Dr Mouton has been professionally under the spotlight of the GMC for all that time with no further complaints. He submitted that Dr Mouton has looked at the conditions imposed as a means of improving his practice.

23. Mr Ramasamy submitted that in the light of Dr Mouton’s remediation since 2016 his fitness to practise is not impaired today.

Further submissions at stage 2

24. Mr Ramasamy proposed that the Tribunal should hear submissions from both Counsel before Dr Mouton gave evidence. Mr Atherton made no objection to proceeding in this way and the Tribunal acceded to this request. Following oral evidence by Dr Mouton, the parties made additional stage 2 submissions in relation to the evidence provided by Dr Mouton.

Further submissions on behalf of the GMC

25. Mr Atherton submitted that the stage 2 oral evidence of Dr Mouton had been illuminating in revealing the effect the Tribunal’s determination on facts has had on Dr Mouton. He submitted that it seems, in a relatively short time, that Dr Mouton had come to recognise the conduct as set out in the charges cannot be allowed to go on.
Further submissions on behalf of Dr Mouton

26. Mr Ramasamy invited the Tribunal to consider a question put to Dr Mouton by Mr Atherton, ‘What does Dr Mouton add which a mainstream doctor cannot’. He submitted that, for patients who resist or are unsatisfied with a mainstream approach, Dr Mouton may offer an alternative perspective but can achieve the same results as mainstream practice.

27. Mr Ramasamy referred to a submission made by Mr Atherton that if the Tribunal did not make a finding of Dr Mouton’s fitness to practise being impaired, it would be endorsing his conduct. He submitted that is incorrect as the Tribunal are considering current impairment today. He submitted that if the Tribunal were to find Dr Mouton’s fitness to practise not impaired today, it would not be endorsing that conduct, it would be recognising the changes he has made during these proceedings.

28. Mr Ramasamy submitted that Dr Mouton provided oral evidence at stage 2, which he chose to do. He submitted that the Tribunal have heard a man able to answer in a considered and reflective way, having digested much of the Tribunal’s determination on facts, demonstrating his reflection and insight. He submitted that Dr Mouton’s mind-set has changed through this process and he no longer accepts just being in a Functional Medicine mind-set. He submitted that Dr Mouton was grateful for the Tribunal’s detailed decision which he has accepted and will share with Dr II and his colleagues. Mr Ramasamy submitted that Dr Mouton’s communications with General Practitioners are now open. Mr Ramasamy submitted that by being open he takes the risk that General Practitioners could complain about his practice to the GMC. He submitted that Dr Mouton will have a more cautious approach in the future and that this case is an example of this regulatory process working as Dr Mouton has engaged, listened and learned.

The Relevant Legal Principles

29. The Tribunal reminded itself that at this stage of proceedings, there is no burden or standard of proof and the decision of impairment is a matter for the Tribunal’s judgement alone.

30. In approaching the decision, the Tribunal was mindful of the two stage process to be adopted: first whether the facts as found proved amounted to serious misconduct. It must then consider whether Dr Mouton’s fitness to practise is impaired by reason of any serious misconduct.

31. The Tribunal must determine whether Dr Mouton’s fitness to practise is impaired today, taking into account Dr Mouton’s conduct at the time of the events and relevant factors such as whether he has insight, whether the matters are remediable, whether they have been remediated and any likelihood of repetition.
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32. Throughout its consideration of the issue of impairment, the Tribunal has borne in mind the overarching objective as set out in section 1 of the Medical Act (1983)

The Tribunal’s Determination on Impairment

Misconduct

Patient A

1. On various dates between 10 April 2013 and 18 November 2014, you consulted with Patient A and you:

    a. failed to elicit from Patient A adequate information about:
        i. possible drug misuse;
           Found proved

33. The Tribunal determined that on the basis of the letter from Patient A’s father to Dr Mouton about Patient A’s possible drug misuse, Dr Mouton failed to elicit any information about possible drug misuse during the consultation. However, the judgement of the Tribunal was that this omission would not be considered deplorable by fellow practitioners. It therefore amounted to misconduct but not serious misconduct.

    b. failed adequately to assess Patient A’s mental state;
       Found proved

    c. attributed Patient’s A’s symptoms to:
        i. hypothyroidism;
           Admitted and found proved
        ii. nutritional issues;
            Admitted and found proved
        iv. leaky gut syndrome;
            Admitted and found proved

    d. failed to make a differential diagnosis of a psychiatric disorder in light of Patient A’s presenting symptoms.
       Found proved

34. Patient A presented with symptoms of depressed mood, including physical manifestations of depression, on more than one occasion. It was incumbent on Dr
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Mouton to assess Patient A’s mental state to determine the likelihood of self-harm, suicide and harm to others. It is the Tribunal’s opinion that this is a matter of serious professional misconduct.

35. The Tribunal noted that Dr Mouton admitted to attributing Patient A’s symptoms to hypothyroidism, nutritional issues and leaky gut syndrome. He failed to include the possibility of a psychiatric disorder in his differential diagnosis despite Patient A’s presenting symptoms. The failure to adequately assess Patient A’s mental state was inextricably linked to failing to make a differential diagnosis that included a psychiatric disorder. This failure amounted to serious professional misconduct as it left Patient A’s symptoms of depression unaddressed with the potential for further deterioration.

2. You referred Patient A for the investigation of:

   a. urinary thyroid hormones;  
      **Admitted and found proved**

   b. steroid hormones;  
      **Admitted and found proved**

   c. insulin;  
      **Admitted and found proved**

   d. DIO2 genotype testing.  
      **Admitted and found proved**

3. The investigations referred to in paragraph 2 were not clinically indicated.

   Urinary thyroid hormones  
   **Found proved**

36. The Tribunal heard evidence that other practitioners use urinary thyroid hormone testing. It also heard evidence from Professor Q who accepted that there was a greater freedom for testing in the private sector. The Tribunal therefore determined that, taken alone, this amounted to misconduct but not serious misconduct.

   DIO2 genotype testing  
   **Found proved**

37. The Tribunal considered that the scientific evidence is such that this test is not of value for the uses to which Dr Mouton was putting it. However, the Tribunal accepts that Dr Mouton genuinely believed that this test was of some value, notwithstanding the lack of robust scientific evidence to support it. In those
circumstances, although the test was not clinically useful or indicated, the Tribunal did not think that, taken alone, this amounted to serious professional misconduct.

4. You prescribed dietary treatments for Patient A which were not:
   
   a. clinically indicated;
      Iodine Found proved
      Selenium Found proved
   
   b. supported by scientific guidelines;
      Iodine Found proved
      Selenium Found proved
   
   c. supported by sound scientific evidence (“not evidence-based”).
      Iodine Found proved
      Selenium Found proved

38. The Tribunal found that Dr Mouton’s use of selenium in respect of Patient A did not amount to serious professional misconduct as the American guidelines do not suggest that it is potentially dangerous even if its benefit is not established. However, in respect of iodine, the Tribunal noted the guidance set out in the facts determination as well as the evidence of Professor Q who said that iodine ingestion can potentially disturb thyroid function in the direction of over activity or under activity and that it should therefore not be given to patients with thyroid issues. Professor Q expressed a clear view that iodine is an unsafe supplement for patients with hypothyroidism. Accordingly, the Tribunal takes the view that the prescribing of iodine to Patient A amounted to serious misconduct.

6. In an email to Patient A’s father dated 15 December 2014, you:

   a. advised him that psychiatrists ‘too often react with a very heavy chemical load which I find dangerous and sometimes definitely destructive’; Admitted and found proved
   
   b. failed to communicate clearly with Patient A’s father, in that you provided contradictory advice regarding referring Patient A to a psychiatrist. Found proved

39. The Tribunal noted that paragraph 6a of the Allegation was admitted and found proved. It is the Tribunal’s judgement was this was an inappropriate and unprofessional comment to make.

40. The context of the email written by Dr Mouton, on 15 December 2014, was that he was responding to an email from Patient A’s father who indicated that the NHS General Practitioner had recommended that Patient A should see a psychiatrist.
in the light of recent events that had occurred at Wimbledon police station. Patient A’s father requested that as Dr Mouton was the only healthcare professional actively treating Patient A, he might be best placed to make the referral to the psychiatrist taking all of Patient A’s symptoms into account. However, Dr Mouton gave conflicting advice as to whether Patient A would benefit from seeing a psychiatrist and indeed whether he would facilitate the referral. He went on to make comments about psychiatrists in general that would have discouraged Patient A’s father from continuing with the referral.

41. The Tribunal noted paragraphs 15, 16d and e, and 35 of GMP:

“15. You must provide a good standard of practice and care. If you assess, diagnose or treat patients, you must:

a. adequately assess the patient’s conditions, taking account of their history (including the symptoms and psychological, spiritual, social and cultural factors), their views and values; where necessary, examine the patient
b. promptly provide or arrange suitable advice, investigations or treatment where necessary
c. refer a patient to another practitioner when this serves the patient’s needs.”

“16. In providing clinical care you must:
…
d. consult colleagues where appropriate
e. respect the patient’s right to seek a second opinion”

“35. You must work collaboratively with colleagues, respecting their skills and contributions.”

42. The Tribunal is of the view that the comment about psychiatrists and the contradictory advice given by Dr Mouton created a risk that a vulnerable patient would not receive the timely and appropriate intervention and care that he needed at that time. It clearly would have served Patient A’s needs to be assessed by a psychiatrist. Accordingly, the Tribunal took the view that this was a serious departure from GMP and constituted serious professional misconduct.

**Patient B**

7. Between 1 September 2014 and 1 September 2015 you consulted with Patient B and you referred Patient B for the following investigations:

   a. CTLA4 genotype testing;

   Admitted and found proved
43. The Tribunal noted that Dr Mouton’s purpose for doing this test was to obtain information as to whether any autoimmune thyroiditis had a genetic origin. Whilst Dr S supported Dr Mouton’s view that there was an association for this gene with autoimmune diseases, it preferred the view of the experts, Professor P and Dr T, that the test is in fact associated with assessing a risk for ulcerative colitis and that it is not clinically useful because of only an approximate two fold risk modification from the test. Whilst the Tribunal has concluded that this test was not clinically indicated, it did not take the view that Dr Mouton’s use of this test, taken alone, amounted to serious professional misconduct.

10. The investigations referred to in paragraph 9 were not clinically indicated.

Urinary thyroid hormones Found proved

44. In respect of the urinary thyroid hormone investigations, the Tribunal determined that, taken alone, this did not amount to serious misconduct for the same reasons outlined in respect of Patient A.

12. You prescribed Patient B the treatments set out in Schedule 1, which were not:

a. clinically indicated;
   - Iodine Found proved
   - Selenium Found proved
   - Novothyral Found proved
   - Pregnenolone Found proved

b. supported by scientific guidelines;
   - Iodine Found proved
   - Selenium Found proved
   - Novothyral Found proved
   - Pregnenolone Found proved

c. evidence based.
   - Iodine Found proved
   - Selenium Found proved
   - Novothyral Found proved
   - Pregnenolone Found proved

45. The Tribunal did not consider there were any reasons to distinguish this case from those circumstances in relation to Patient A, therefore, for the same reasons
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previously set out, it determined that the prescribing of iodine amounted to serious professional misconduct.

46. In respect of selenium, the Tribunal concluded that this did not amount to serious professional misconduct for the same reasons as outlined in respect of Patient A.

47. Patient B had no more than borderline results for hypothyroidism. Dr Mouton stated in his evidence that Patient B was neither euthyroid nor hypothyroid. Notwithstanding this, Dr Mouton prescribed Novothyral (combination T3 and T4 therapy) from the outset. This clearly went against the guidelines suggesting that combination therapy should not be used in the first instance and that combination therapy should be reserved for use by an accredited consultant endocrinologist. The Tribunal takes the view that Dr Mouton’s use of Novothyral in this way had the potential to cause the patient harm and therefore amounted to serious professional misconduct.

48. The Tribunal noted the evidence of Professor Q that there was a risk that if Pregnenolone was prescribed it was uncertain if it would result in excessive production of active steroids and that there was no evidence, that Pregnenolone was a helpful hormone to prescribe to patients such as Patient B. The Tribunal noted that there was no evidence before it that Dr Mouton had taken into account the psychiatric effects of Pregnenolone and none of the evidence before it supported his use of Pregnenolone. The Tribunal concluded that because of the potential risks of a non-specialist practitioner prescribing Pregnenolone, in these circumstances, Dr Mouton’s use of it amounted to serious professional misconduct.

13. You failed to communicate adequately with Patient B’s General Practitioner regarding:

a. Patient B’s diagnoses, including:

   i. hypothyroidism;  
      **Found proved**

   ii. iodine deficiency;  
       **Found proved**

   iii. hypogonadism;  
       **Found proved**

b. Patient B’s prescriptions as set out in Schedule 1.  
   **Found proved**
49. The Tribunal determined that Dr Mouton’s communication with Patient B’s General Practitioner, Dr K, consisted simply of sending her proformas enclosing his results but without any diagnosis or explanations of the prescriptions he had made. Whilst the Tribunal was of the view that this was not adequate communication with a fellow practitioner, the Tribunal did not consider that his failures in communication in respect of Patient B amounted to serious misconduct.

**Patient C**

14. Between 15 December 2013 and 22 September 2015, you consulted with Patient C and you failed to record:

a. any clear diagnosis; **Found proved**

50. The Tribunal has already determined that Dr Mouton failed to make a clear diagnosis in respect of Patient C. However, the Tribunal noted that Dr Mouton had documented a large number of symptoms in respect of this patient and that Patient C’s gastroenterologist had also not reached a clear and firm conclusion with regard to a diagnosis for Patient C. The Tribunal concluded that Dr Mouton’s failure to record a clear diagnosis did not amount to serious misconduct.

16. The investigations referred to in Schedule 2 were:

b. not clinically indicated;

DIO2 genotype testing  **Found proved**
CTLA-4 genotype test  **Found proved**

51. In respect of the DIO2 genotype test, the Tribunal determined that, taken alone, this did not amount to not serious misconduct for the reasons previously set out in relation to Patient A.

52. In respect of the CTLA-4 genotype test, the Tribunal determined that, taken alone, this did not amount to not serious misconduct for the reasons previously set out in relation to Patient B.

b. conducted without obtaining informed consent from Patient C;

Apo E genotype testing  **Found proved**
MTHFR genotype testing  **Found proved**

c. conducted without appropriate counselling of Patient C.

Apo E genotype testing  **Found proved**
MTHFR genotype testing  **Found proved**
53. In respect of the Apo E genotype test, proper counselling and obtaining of informed consent are particularly important because of the potential implications of the result of this test in relation to the risk of the patient developing Alzheimer’s disease. The Tribunal determined that Dr Mouton was dismissive of the significance of this aspect of the test result. It concluded that Dr Mouton had failed to obtain informed consent or to provide proper counselling both before and after the test was carried out. This was particularly significant in the case of Patient C as she had an adverse result from the Apo E genotype test result indicating her risk of developing Alzheimer’s disease. The Tribunal concluded that these failures, caused by Dr Mouton’s focus on the dietary implications of the result were serious and the failure to have counselled and obtained informed consent for a test with such serious implications amounted to serious professional misconduct.

54. In respect of the MTHFR genotype test, the Tribunal noted that Patient C consulted Dr Mouton expressing concerns in respect of a very serious disease, namely breast cancer. Dr Mouton referred her for the MTHFR genotype test, without explaining to her that there was an alternative, more widely recognised test that could be conducted. The danger of the patient receiving false reassurance without being given that information was significant. The Tribunal concluded therefore that Dr Mouton’s failure to appropriately counsel or obtain Patient C’s informed consent in respect of this test did amount to serious professional misconduct.

18. The investigations referred to in paragraph 17 were not clinically indicated.

Investigation of urinary thyroid hormones: Found proved
Thyroid ultrasound scan: Found proved

55. In respect of the urinary thyroid hormone investigations, the Tribunal determined that, taken alone, this did not amount to serious misconduct for the same reasons as outlined in respect of Patient A.

56. In respect of the thyroid ultrasound scan, the Tribunal noted that the thyroid ultrasound scan is not an invasive or harmful procedure. It noted Dr Mouton’s evidence that such a test is commonplace in Belgium. The Tribunal also noted Professor Q’s evidence that there is more freedom for the conducting of tests within the private sector. Therefore, whilst the Tribunal did not consider that this test was clinically indicated, it did not consider that the carrying out of this test, taken alone, amounted to serious professional misconduct.

19. You prescribed Patient C the treatments set out in Schedule 3, which were not:

a. clinically indicated;
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Iodine & Tyrosine Found proved
Pregnenolone Found proved
Selenium and selenomethionine Found proved
Armour Thyroid and l-thyroxine Found proved

b. supported by scientific guidelines;

Iodine & Tyrosine Found proved
Pregnenolone Found proved
Selenium and selenomethionine Found proved
Armour Thyroid and l-thyroxine Found proved

c. evidence based.

Iodine & Tyrosine Found proved
Pregnenolone Found proved
Selenium and selenomethionine Found proved
Armour Thyroid and l-thyroxine Found proved

57. In respect of the prescribing of Iodine, the Tribunal determined that this amounted to serious professional misconduct, for the same reasons as outlined in respect of Patient A.

58. In respect of the prescribing of Pregnenolone, the Tribunal determined that this amounted to serious professional misconduct, for the same reasons as outlined in respect of Patient B.

59. The Tribunal noted that both Professor Q and Dr S were very clear that Patient C was euthyroid and there was no reason for Patient C to be given any selenium. The prescription of selenium, in these circumstances, meant there was a risk of potential selenium toxicity and therefore, in this case the Tribunal concluded that prescribing selenium to Patient C amounted to serious professional misconduct.

60. The Tribunal went on to consider the prescribing of Armour Thyroid and l-thyroxine. Patient C was euthyroid. Dr Mouton had commenced this patient on what amounted to a course of combination therapy when there was no clinical indication to do so. This was clearly against the guidelines and was a course of treatment that had a potential risk of harm. In the judgement of the Tribunal this amounted to serious professional misconduct.

20. You failed to communicate adequately with:

b. Patient C’s General Practitioner regarding the tests referred to in Schedule 2 and the implications of them; Found proved
c. Patient C’s treating Gastroenterologist regarding the intestinal and dietary treatments referred to in Schedule 3.  

**Found proved**

61. The Tribunal noted that at the beginning of the period during which Patient C consulted Dr Mouton, he failed to communicate with her General Practitioner at all, instead relying on Patient C to deliver the information to the General Practitioner. From about mid July 2015 onwards Dr Mouton did send the General Practitioner copies of the results with a proforma cover letter. The Tribunal determined that this was inadequate communication with the General Practitioner especially having regard to the importance of the General Practitioner as the central holder of a patient’s medical records within the NHS. However, there is no evidence of Dr Mouton refusing to engage with the General Practitioner or being deliberately obstructive. The Tribunal concluded that Dr Mouton’s failure in this respect did not constitute serious professional misconduct.

62. Dr Mouton was aware throughout the period concerned that Patient C was under the care of a Gastroenterologist. It was clearly important that any such consultant was fully aware of the treatments that Dr Mouton was prescribing for the patient, given the potential for there to be interaction between Dr Mouton’s prescriptions and any treatment by the gastroenterologist. Dr Mouton relied on the patient to pass on this information. However, Dr Mouton could not know whether or not the patient was passing on the information of his treatment accurately or indeed at all. The Tribunal concluded that Dr Mouton’s failure to have a proper and professional system for keeping a fellow treating practitioner informed of his treatment and management of Patient C did amount to serious professional misconduct.

**Patient D**

21. Between 14 January 2014 and 9 July 2015, you consulted with Patient D and you referred Patient D for the following investigations:

   a. investigation of urinary thyroid hormones;  
      **Admitted and found proved**

   b. thyroid ultrasound scan.  
      **Admitted and found proved**

22. The investigations referred to in paragraph 21 were not clinically indicated.

   Investigation of urinary thyroid hormones **Found proved**  
   Thyroid ultrasound scan **Found proved**
63. In respect of the urinary thyroid hormone investigations, the Tribunal determined that, taken alone, this did not amount to serious misconduct for the same reasons outlined in respect of Patient A.

64. In respect of the thyroid ultrasound scan, the Tribunal determined that, taken alone, this did not amount to serious misconduct for the same reasons outlined in respect of Patient C.

23. You referred Patient D for the investigation of DIO2 genotype testing and such investigation was:
   a. not clinically indicated;  
      Found proved

65. In respect of the DIO2 genotype test, the Tribunal determined that, taken alone, this did not amount to serious misconduct for the reasons previously set out in relation to Patient A.

24. You failed to adequately interpret or act upon the findings that Patient D was biochemically euthyroid. 
   Found proved

25. You prescribed the treatments set out in Schedule 4 which were not:
   a. clinically indicated;
      Pregnenolone  Found proved
      Novothyral  Found proved
   b. supported by scientific guidelines;
      Pregnenolone  Found proved
      Novothyral  Found proved
   c. evidence based.
      Pregnenolone  Found proved
      Novothyral  Found proved

66. In regards to paragraphs 24 and 25a, b and c of the Allegation in respect of Novothyral, the Tribunal considered these two paragraphs to be inextricably linked. The Tribunal noted the evidence of Professor Q and Dr S that Patient D was biochemically euthyroid. Nonetheless, Dr Mouton prescribed Patient D with Novothyral as a first line treatment. The prescription of Novothyral (combination T3 and T4 therapy) was in contradiction to the scientific guidelines. The Tribunal considered that Dr Z, Patient D’s Consultant Endocrinologist, expressed her concern
that excessive thyroid replacement could cause a long term result of osteoporosis and atrial fibrillation. Dr Z stated “[Patient D] has already had some side effects such as light periods and weight loss”. The Tribunal concluded that the failure to adequately interpret or act upon the fact that Patient D was euthyroid, and the resultant prescription of Novothyral amounted to serious professional misconduct.

67. In regards to paragraph 25a, b and c of the Allegation in relation to Pregnenolone, the Tribunal determined that for the same reasons as outlined in respect of Patient B, this treatment amounted to serious professional misconduct.

**Patient E**

27. The investigations referred to in paragraph 26 were not clinically indicated.
   - Investigation of urinary thyroid hormones *Found proved*
   - Thyroid ultrasound scan *Found proved*

68. In respect of the urinary thyroid hormone investigations, the Tribunal determined that, taken alone, this did not amount to serious misconduct for the same reasons outlined in respect of Patient A.

69. In respect of the thyroid ultrasound scan investigations, the Tribunal determined that, taken alone, this did not amount to serious misconduct for the same reasons outlined in respect of Patient B.

29. The investigations referred to in paragraph 28 were:
   a. not clinically indicated;
      - DIO2 genotype testing *Found proved*
      - CTLA-4 genotype testing *Found proved*

70. In respect of the DIO2 genotype test, the Tribunal determined that, taken alone, this did not amount to not serious misconduct for the reasons previously set out in respect of Patient A.

71. In respect of the CTLA-4 genotype test, the Tribunal determined that, taken alone, this did not amount to not serious misconduct for the reasons previously set out in respect of Patient B.

   b. conducted without obtaining informed consent from Patient E;
      - Apo E genotype testing *Found proved*

   c. conducted without appropriate counselling of Patient E.
Apo E genotype testing  **Found proved**

72. The Tribunal considered paragraphs 29b and c of the Allegation in relation to Apo E genotype testing together. It has already concluded that this test in respect of Patient C amounted to serious professional misconduct. The Tribunal noted, in the case of Patient E, there was no adverse result in respect of the risk of developing Alzheimer’s disease. Nonetheless, the failure to obtain informed consent and conduct appropriate counselling for this test with its potential ramifications amounted to serious professional misconduct.

30. You failed to adequately interpret or act upon the findings that Patient E was biochemically euthyroid. **Found proved**

32. You prescribed the treatments referred to in Schedule 5 which were not:

a. clinically indicated;

   Thyrocsin  **Found proved**
   Tirform  **Found proved**
   Pregnenolone  **Found proved**
   GTA  **Found proved**
   Novothyral  **Found proved**
   L-selenomethionine  **Found proved**

b. evidence based.

   Thyrocsin  **Found proved**
   Tirform  **Found proved**
   Pregnenolone  **Found proved**
   GTA  **Found proved**
   Novothyral  **Found proved**
   L-selenomethionine  **Found proved**
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73. The Tribunal considered that paragraph 30 is inextricably linked with paragraph 32a, b and c of the Allegation. It went on to consider the prescribing of Novothyral and GTA in this regard. With respect to Novothyral and GTA, the Tribunal noted that this was another patient who was biochemically euthyroid but was nonetheless treated with Novothyral (combination T3 and T4 therapy), and also GTA which also contains thyroid concentrate. The Tribunal concluded prescribing Novothyral and GTA in a biochemically euthyroid patient amounted to serious professional misconduct.

74. In respect of Thyrocsin, the Tribunal noted that this contains iodine. For this reason and those set out in relation to Patient A, it considered that prescribing Thyrocsin amounted to serious professional misconduct.

75. In respect of Triform and L-selenomethionine, the Tribunal considered that both of these products contain selenium. Patient E was biochemically euthyroid and therefore, for the reasons set out in relation to Patient C, the Tribunal determined that prescribing these products amounted to serious professional misconduct.

76. In respect of the prescribing of Pregnenolone, the Tribunal determined that this amounted to serious professional misconduct, for the same reasons as outlined in respect of Patient B.

33. You failed to communicate adequately with:
   a. Patient E’s General Practitioner regarding the tests referred to in paragraph 28 and the implications of them;
      Found proved

77. In respect of paragraph 33a of the Allegation, the Tribunal noted the inadequacies of Dr Mouton’s communication with Patient E’s General Practitioner as set out in its determination on the facts. The Tribunal also noted that from July 2015 Dr Mouton did communicate with Patient E’s General Practitioner, albeit with a full list of laboratory results, a proforma covering letter and a few brief comments. There was no evidence of him being obstructive or refusing to communicate with the General Practitioner when asked to do so. In all the circumstances, the Tribunal considered that the inadequacies of his communications with Patient E’s General Practitioner did not amount to serious professional misconduct.

Patient F

35. The investigations referred to in paragraph were:
   a. not clinically indicated;
      DIO2 genotype testing    Found proved
78. In respect of Patient F, there was no clinical indication for carrying out the DOI2 genotype test. Dr Mouton gave evidence that Patient F was biochemically euthyroid. Despite this finding Dr Mouton still proceeded to conduct this genomic test. The evidence provided to the Tribunal was quite clear that genomic testing in children should only be carried out in the case of medical necessity. In the absence of such necessity, genomic testing should be deferred until the age of 18. The Tribunal finds that Dr Mouton’s disregard and deviation from guidelines and acceptable practice in this case amounted to serious professional conduct.

b. conducted without obtaining informed consent from Patient F and/or from his parent or guardian;  
   Found proved

c. conducted without appropriate counselling of Patient F and/or his parent or guardian.  
   Found proved

79. The Tribunal noted that Patient F underwent testing for the Apo E, DIO2 and FUT2 genotypes. For all genotype testing, the Tribunal has previously set out in detail its reasoning why it concluded that Dr Mouton had not obtained informed consent from Patient F and/or his parent, or conducted appropriate counselling of Patient F and/or his parent. In particular, the Tribunal noted the reaction of Professor DD when she became aware of these tests being conducted: “In my view the GMC should be informed that he appears to be offering testing in a way that is not compliant with professional guidelines and this is dangerous. (As it happens, I chaired the Human Genetics Commission committee that wrote the Framework of Principles for the Regulation of Direct to Consumer Genetic tests.)”. The Tribunal also considered the fact that there was no note of any post-test counselling, notwithstanding the fact that Patient F’s result for the Apo E test indicated an increased risk factor for Alzheimer’s disease by a factor of 15. For all these reasons, the Tribunal concluded that these failures did amount to serious professional misconduct.

36. You failed to adequately interpret or act upon the findings that Patient F was biochemically euthyroid.  
   Found proved

37. You prescribed thyroid hormone treatment, Armour thyroid, to Patient F which treatment was not:

a. clinically indicated;  
   Found proved

b. supported by scientific guidelines;
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Found proved
c. evidence based.
Found proved

80. The Tribunal noted paragraph 14 of GMP, which states:

“14. You must recognise and work within the limits of your competence.”

81. The Tribunal has noted that Patient F was euthyroid. Nonetheless, Dr Mouton embarked on a course of treatment for Patient F that included treating him with thyroid hormone which contained both T4 and T3. The Tribunal noted Professor Q’s opinion:

“...there was insufficient evidence to make a diagnosis of hypothyroidism in this child, and that such a diagnosis would not be considered by a paediatric endocrinologist. A diagnosis of hypothyroidism is a significant and life long diagnosis, with major implications for long term treatment, health, monitoring and health costs... While the relative safety of closely monitored thyroid treatment is agreed, there were also reservations about the possibility of overtreating a child.”

82. The guidelines are very clear that prescriptions of thyroid hormone to a child should be carried out by a consultant paediatric endocrinologist. The Tribunal considered that by treating Patient F in the way that he did, Dr Mouton was failing to recognise the limits of his clinical competence. Therefore, the Tribunal considered these failures as a significant departure from GMP and constituted serious professional misconduct.

38. You failed to communicate appropriately with Patient F’s General Practitioner in that you:

b. failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing to which you referred Patient F, namely:

i. why the tests were carried out;
Found proved

ii. the degree of explanation given to Patient F’s parent and/or Patient F prior to taking the test;
Found proved
iii. any implications that the General Practitioner should be aware of arising from the results of the genetic testing;  
*Found proved*

83. The Tribunal noted that Dr Mouton failed to provide as much detail as he should have done in his letter to the General Practitioner with regards to genetic testing which he carried out in respect of Patient F. However, the Tribunal noted that Dr Mouton did reply to Dr M’s request for information and also that Dr Mouton ended his letter of 16 December 2015 by inviting Dr M to let him know if the answers satisfied him and offering to answer any further queries. In all the circumstances the Tribunal determined that these failures did not amount to serious professional misconduct.

**Patient G**

39. Between 17 November 2014 and 16 December 2015 you consulted with Patient G and you referred him for the following investigations:

a. Apo E genotype testing;  
*Admitted and found proved*

b. DIO2 genotype testing;  
*Admitted and found proved*

40. The investigations referred to in paragraph 39 were:

a. not clinically indicated;

   Apo E genotype testing  
   *Found proved*

   DIO2 genotype testing  
   *Found proved*

b. conducted without obtaining informed consent from Patient G and/or from his parent or guardian;

   Apo E genotype testing  
   *Found proved*

   DIO2 genotype testing  
   *Found proved*

c. conducted without appropriate counselling of Patient G and/or his parent or guardian.

   Apo E genotype testing  
   *Found proved*

   DIO2 genotype testing  
   *Found proved*

84. In relation to paragraph 40a, b and c of the Allegation, the Tribunal has already set out its view of these tests with regard to Patient F. In respect of Patient
G, the Apo E genotype test was not clinically indicated as there was no evidence of clinical failure to thrive which had provided a justification for the Apo E test in the case of Patient F.

85. The Tribunal noted that in Patient G’s case there was again an increased risk of Alzheimer’s disease indicated by the results of the Apo E test. For the same reason as indicated in relation to Patient F, the Tribunal determined that the carrying out of this test and the failures in respect of obtaining informed consent and counselling amounted to serious professional misconduct.

41. You failed to adequately interpret or act upon the findings that Patient G was biochemically euthyroid. **Found proved**

42. You prescribed thyroid hormone treatment, Armour thyroid, to Patient G which treatment was not:

   a. clinically indicated; **Found proved**
   b. supported by scientific guidelines; **Found proved**
   c. evidence based. **Found proved**

86. In relation to paragraphs 41 and 42 of the Allegation, for the same reasons as set out in relation to Patient F, the Tribunal determined that these acts amounted to serious professional misconduct.

43. You failed to communicate appropriately with Patient G’s General Practitioner in that you:

   b. failed to respond adequately or appropriately to the questions of the General Practitioner as to the genetic testing to which you referred Patient G, namely:

      i. why the tests were carried out; **Found proved**
      ii. the degree of explanation given to Patient G’s parent and/or Patient G prior to taking the test; **Found proved**
any implications that the General Practitioner should be aware of arising from the results of the genetic testing;

**Found proved**

c. suggested that the results of the genetic testing that you sent to the General Practitioner should not be entered into the medical notes of Patient G. **Found not proved**

87. In relation to paragraphs 43b and c of the Allegation, for the same reasons as set out in relation to Patient F, the Tribunal determined that these failures did not amount to serious professional misconduct.

**Patient H**

45. The investigations referred to in paragraph 44 were:

a. not clinically indicated;

APOE genotype testing **Found proved**  
DIO2 genotype testing **Found proved**

88. The Tribunal determined that in Patient H’s particular circumstances the Apo E genotype test was not clinically indicated. However, in the context of this consultation with Dr Mouton, in the Tribunal’s judgement, this did not amount to serious professional misconduct.

89. In respect of the DIO2 genotype test, the Tribunal determined that, taken alone, this did not amount to not serious professional misconduct for the reasons provided in respect of Patient A.

b. conducted without obtaining informed consent from Patient H;

APOE genotype testing **Found proved**

c. conducted without appropriate counselling of Patient H.

APOE genotype testing **Found proved**

90. In respect of paragraph 45b and c, the Tribunal has already determined that the failure to obtain informed consent and counsel other patients in respect of this test amounted to serious professional misconduct. Patient H was a 67 year old female complaining of certain symptoms that might be consistent with Alzheimer’s disease. Whilst in the event, the results do not show an increased risk of Alzheimer’s disease, the Tribunal determined that it was particularly important that the potential
results of the test should have been discussed properly with Patient H in advance in order to obtain her informed consent and counsel her fully. The failure by Dr Mouton to do so and his failure to provide any post-test counselling to reassure Patient H were, in the view of the Tribunal, serious failures which amounted to serious professional misconduct.

46. You failed to adequately interpret or act upon:
   
   a. the findings that Patient H was biochemically euthyroid;  
       **Found proved**

   b. Patient H’s steroid blood results.  
       **Found proved**

47. You prescribed the treatments referred to in Schedule 6 which were not:
   
   a. clinically indicated;
      GTAEN natural thyroid extract  
      Pregnenolone  
      **Found proved**  
      **Found proved**

   b. supported by scientific guidelines;
      GTAEN natural thyroid extract  
      Pregnenolone  
      **Found proved**  
      **Found proved**

   c. evidence based.
      GTAEN natural thyroid extract  
      Pregnenolone  
      **Found proved**  
      **Found proved**

91. In respect of paragraph 46a of the Allegation, for the reasons previously set out in relation to Patients D and E, the Tribunal determined that the failure to interpret or act upon the findings that Patient H was biochemically euthyroid amounted to serious professional misconduct.

92. In relation to paragraphs 46a and 47a, b and c of the Allegation in respect of GTAEN natural thyroid extract, the Tribunal considered that as this contains natural T3 and T4 thyroid hormone and Patient H was biochemically euthyroid this amounted to serious misconduct.

93. The Tribunal considered paragraphs 46b in respect of paragraph 47a, b and c of the Allegation with regards to Patient H’s steroid blood results in relation to the prescribing of Pregnenolone. The Tribunal has determined that Dr Mouton incorrectly
interpreted the steroid blood results as indicating an “underperforming adrenal gland”. Dr Mouton failed to take into account that Patient H was a 67 year old post-menopausal woman. Dr Mouton again focussed his interpretation on the conversion of thyroid T4 pro-hormones into active T3 hormones and as a result prescribed Pregnenolone. The Tribunal have already set out the risks of prescribing Pregnenolone unnecessarily. In all the circumstances, the judgement of the Tribunal is that this amounts to serious professional misconduct.

49. You failed to adequately or properly communicate to Patient H or her General Practitioner:

   c. your diagnosis;  
   **Found proved**

   d. why you had prescribed the treatments referred to in Schedule 6. **Found proved**

94. Dr Mouton simply sent Patient H’s General Practitioner the test results with a short proforma covering letter. Whilst this form of communication was inadequate, the Tribunal determined that it did not amount to serious misconduct.

**Overall Consideration of Misconduct**

95. Having considered each of its findings of facts individually, the Tribunal noted that in this case there were a number of findings with common features. Dr Mouton used testing for urinary thyroid hormones, thyroid ultrasound scans, and DIO2 and CTLA4 genotype testing for certain patients when not clinically indicated. These tests were carried out by Dr Mouton with a view to prescribing thyroid hormones as he thought appropriate. The Tribunal bore in mind the case of Schodlok and the passage at paragraph 72, in particular:

   “...I recognise that a small number of allegations of misconduct that individually are held not to be serious misconduct should normally not be regarded collectively as serious misconduct. Where, however, there are a large number of findings of non-serious misconduct, particularly where they are of the same or similar misconduct, I consider the position is different. In such a case, it should in principle be open for a Fitness to Practise Panel to find that, cumulatively, they are to be regarded as serious misconduct capable of impairing a doctor’s fitness to practise.”

The Tribunal exercised considerable caution before applying that principle to this case.

96. However, the Tribunal has found in respect of a number of patients that Dr Mouton’s assessment and treatment of them in relation to prescribing thyroid
hormones was serious misconduct. The Tribunal considered that Dr Mouton used the tests set out above to justify his over prescription of thyroid hormones. The Tribunal noted the evidence of Dr S that his concern with urinary thyroid tests was that they tended to over diagnose thyroid disease. Furthermore, Dr Mouton relied on thyroid ultrasound scans even though the evidence before the Tribunal was that such scans did not provide any information about thyroid function. The Tribunal concluded that the use of these tests, although not individually constituting serious misconduct, did cumulatively amount to serious misconduct when considered in the context of the many other findings of serious professional misconduct in this case.

Impairment

97. The Tribunal first of all considered whether or not Dr Mouton’s fitness to practise was impaired as a result of his serious misconduct in failing to adequately assess the mental state of Patient A and make a differential diagnosis which included a psychiatric disorder. The Tribunal noted the evidence of Dr Mouton to the Tribunal at the impairment stage in which he acknowledged that he had failed to assess the wider picture in relation to Patient A and should not have allowed himself to have been constrained by the “narrow nutritional frame” that he adopted. However, the Tribunal also noted Dr Mouton’s evidence that he effectively carries out the role of a General Practitioner, and as such it is inevitable that he will see a sizeable number of patients with mental health conditions.

98. The Tribunal observed that none of Dr Mouton’s CPD activities over the last three years include any training in respect of mental health. Further, there is no mention of reviewing his practice in assessing mental health in his Personal Development Plan over the same period. Accordingly, the Tribunal concluded that Dr Mouton has not remediated this area of his practice.

99. The Tribunal next considered Dr Mouton’s practice in respect of assessment, investigation and treatment of thyroid patients. In both his remediation bundle and his oral evidence at the impairment stage, Dr Mouton demonstrated the extent to which his practice has changed since 2016. The logs of his management of thyroid cases, as required by his interim order of conditions, goes some way to show what is remediable, to what extent he has remediated and which aspects of his practice have continued. The Tribunal noted that Dr Mouton continues to consult with approximately 50 new and existing patients each month. Most of these consultations now entail communication with General Practitioners and specialist colleagues. The Tribunal further noted a shift from prescribed thyroid preparations, whether as T4 alone or as T3 and T4 combination therapy to more “non-prescriptive thyroid support”. In his 2019 audit of thyroid cases, Dr Mouton appears to be more willing to reduce thyroid hormone doses when the TSH level is well below the reference range. Dr Mouton stated that this was a notable change in his approach following discussions of the adverse cardiac side effects of over suppression of the TSH.
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100. Analysis of the remediation bundle, in particular the logs, demonstrate aspects of Dr Mouton’s practice that have remained unchanged. There is evidence of continued systematic and indiscriminate use of certain tests. The Tribunal noted numerous references to low urinary T3 levels, DIO2 genotype testing and use of ultrasound scans apparently indicating that Dr Mouton continues to rely on these investigations to aid his assessment and management of these patients. In his oral evidence Dr Mouton was clear that despite the guidelines and the findings of the Tribunal, he would discuss the use of such investigations and rely upon the opinions of Dr BB and Dr II. In relation to DIO2, he stated: “I can’t promise I will not use it, I will keep an eye on the science”. In respect of his use of urinary thyroid hormones and thyroid ultrasound scans he stated that he would consider stopping using them. He specified that Dr BB and Dr II had not commented on his use of these investigations. He said “if they recommend stopping I will stop. There is no way I will go against [their] guidance”. Notwithstanding his evidence, the Tribunal has no evidence that he has currently changed his practice.

101. In respect of Dr Mouton’s failure to adequately interpret laboratory findings, the logs contain relatively few TSH values. However, the Tribunal noted that as recently as January 2019 he described a TSH level of 3.89 as indicating low thyroid function when it is well within the established reference range and on another occasion described a TSH level of 3.38 as borderline. The Tribunal concluded that Dr Mouton continues to misinterpret laboratory findings. This has the effect of him prescribing unnecessary thyroid hormone treatment, thereby potentially exposing patients to risk of harm.

102. Whilst the Tribunal has already noted an apparent shift towards “non-prescriptive thyroid support” there is clear evidence that Dr Mouton continues, in some cases, to use combination T3 and T4 therapy in new patients as a first line approach. This is another example of his management being at odds with BTA guidelines, which Dr Mouton stated in his appraisals and oral evidence that he now faithfully follows.

103. With regards to supervision of Dr Mouton’s management in thyroid cases, the Tribunal noted that he has been supervised first of all by Dr BB and latterly by Dr II. This is undoubtedly a positive step. However, only a limited number of Dr Mouton’s cases are able to be reviewed in this way and the Tribunal further noted that none of these cases involved the use of “non-prescriptive thyroid support”. The Tribunal also noted that this process has not detected the instances where Dr Mouton’s practice has derogated from the guidelines.

104. In respect of his use of selenium, iodine and pregnenolone, the Tribunal was encouraged by Dr Mouton’s attitude to the Tribunal’s findings during his evidence. With regard to iodine, Dr Mouton indicated that he would not prescribe this to any patient who did not have an iodine deficiency. He also stated that he would discuss the use of these treatments with his supervising consultant endocrinologist.
However, on the evidence currently before the Tribunal, there is no evidence that Dr Mouton has changed his practice in this respect and therefore the Tribunal cannot say that there is no risk of repetition.

105. The Tribunal went on to consider Dr Mouton’s paediatric practice as, in their opinion, this warranted particular attention. The Tribunal noted that Dr Mouton continues to consult and treat paediatric patients for thyroid issues. This is a direct derogation from the guidelines. The Tribunal further noted that Dr Mouton continues to order genomic testing for paediatric patients in the absence of medical necessity. As such, the Tribunal concluded that his practice of treating and conducting genotype testing in paediatric patients remains unchanged.

106. Dr Mouton’s case has highlighted his practice in respect of counselling and obtaining informed consent for a number of genomic tests conducted with both adults and paediatric patients. The Tribunal has been concerned particularly with Dr Mouton’s failure to counsel patients properly with regard to the implications of the results of the Apo E genotype test relating to Alzheimer’s disease. The Tribunal has before it a number of consent forms for this test conducted since these proceedings began. It is clear to the Tribunal that Dr Mouton is now much more mindful of the need to advise patients about the possible implications of the results of this test concerning Alzheimer’s disease and the Tribunal noted that he now specifically records this on the consent form. The Tribunal considered this to be a significant improvement in his practice and considered that Dr Mouton has remediated his misconduct in this regard.

107. In respect of the MTHFR genotype test that was carried out on Patient C, the Tribunal noted that according to his references spreadsheet, this is still a test that Dr Mouton offers. In his evidence to the Tribunal Dr Mouton stated that he does not offer the alternative BRCA genotype test for breast cancer because “it involves too much counselling”. Dr Mouton has not demonstrated to the Tribunal that when he suggests carrying out the MTHFR tests to patients that he advises them of the possibility of having the alternative and more widely recognised BRCA test for assessing their risk for breast cancer. The Tribunal concluded therefore that Dr Mouton had not shown evidence of remediation in respect of this practice.

108. In respect of his obtaining informed consent in paediatric patients, the Tribunal considered that Dr Mouton still does not sufficiently explain and document that consent for genomic testing rests with the patient and not their parent and, as set out before, such testing should be deferred until they are 18 years old unless there is a medical necessity. Dr Mouton in his oral evidence accepted that his practice still needed to improve in this regard. Whilst the Tribunal accepted that Dr Mouton is willing to develop his practice in this area, the Tribunal does not have evidence that he has remediated this at present.
109. Dr Mouton’s serious misconduct in relation to his communication with others related in respect of Patient A, to his email correspondence with Patient A’s father regarding his unwillingness for Patient A to be referred to a psychiatrist. In respect of Patient C, Dr Mouton failed to inform the gastroenterologist of the treatments he was prescribing. The Tribunal can see from the logs that Dr Mouton has provided, that he does now consistently communicate not only with his patients’ General Practitioner but also with other specialists who are treating his patients. The Tribunal noted that since these proceedings began, Dr Mouton has moved to new premises which he shares with multiple practitioners working in different specialties. The Tribunal noted that there is good evidence that Dr Mouton interacts with these other practitioners both as part of his CPD and by referring patients to them. The Tribunal concluded that Dr Mouton is now much more open to the idea of engaging with professionals from other specialties and the Tribunal therefore concluded that he had fully remediated the deficiencies in his practice with regard to communication with others.

110. The significant feature of this case is Dr Mouton’s failure to comply with the relevant professional guidelines. As the Tribunal has already noted, although Dr Mouton asserted in his 2016 and 2017 appraisals and in his oral evidence to the Tribunal, that he now followed the relevant guidelines for the prescription of thyroid hormones, it noted several instances where his treatment in this regard derogated from the guidelines without appropriate explanation. The Tribunal also noted Dr Mouton’s reflective statements included in his remediation bundle. In his 2018 appraisal he remarked: “also, because my practice is not mainstream, it must be guided by experience rather than by guidelines”, and more recently in one of Dr Mouton’s Functional Medicine blogs, dated 11 January 2019, he states: “anyhow, you will have understood that I am strongly against systematic, blind, and massive calcium supplementation for menopausal women is not my thing...The existence of guidelines recommending it? Just guidelines!”.

111. The Tribunal noted paragraph 16b of GMP:

“16. In providing clinical care you must:

b. provide effective treatments based on the best available evidence”

Guidelines are developed by specialist practitioners having considered all of the best scientific evidence available in a particular field. Dr Mouton’s attitude to guidelines would appear to fail to recognise this fact. The Tribunal considered that there is a risk of Dr Mouton continuing to fail to follow relevant guidelines given the attitudes he has repeatedly expressed.

112. In the light of the above, the Tribunal concluded that Dr Mouton has not remediated in full the clinical failings that have been demonstrated and found proved
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in this case. Accordingly, the Tribunal considered that there is a risk of repetition and that a finding of impairment is necessary to safeguard patient safety.

113. The Tribunal also considered that Dr Mouton’s serious professional misconduct and his persistent non-compliance with guidelines has an impact, not only on his practice, but on public confidence in the profession. The Tribunal concluded that for this reason, and to uphold proper standards within the profession, a finding of impairment was necessary in this case.

DETERMINATION ON SANCTION - 26/09/2019

1. Having determined that Dr Mouton’s fitness to practise is impaired by reason of misconduct, the Tribunal now has to decide on the appropriate sanction, if any, to impose.

Submissions

2. The Tribunal heard submissions from Mr Atherton on behalf of the GMC, and from Mr Ramasamy on behalf of Dr Mouton. These submissions are summarised below.

On behalf of the GMC

3. Mr Atherton submitted that, given the Tribunal’s findings in relation to both facts and impairment, conditions would not be appropriate in Dr Mouton’s case. He submitted that the Tribunal had found wide-ranging and serious misconduct, and that Dr Mouton had persistently failed to follow relevant guidance. He further added that, given the nature of Dr Mouton’s practice, it would be difficult to formulate workable and measurable conditions.

4. Mr Atherton took the Tribunal to relevant sections of the Sanctions Guidance (‘SG’). He submitted that Dr Mouton’s insight had come at a ‘very late stage’. He reminded the Tribunal of the importance of insight when assessing whether conditions could be appropriate and workable, and submitted that Dr Mouton’s failure to observe guidelines in the past - as well as his conduct when previously subject to interim conditions - raise doubts about his ability to comply with any conditions imposed.

5. Mr Atherton submitted that conditions would not adequately address the serious misconduct found by this Tribunal, which included a serious breach of the principles set out in GMP. Accordingly, Mr Atherton invited the Tribunal to impose an order of suspension on Dr Mouton’s registration. In making this submission, Mr Atherton reminded the Tribunal that, when considering the appropriateness of
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suspension, it would need to satisfy itself that there was no significant risk of repetition.

On behalf of Dr Mouton

6. Mr Ramasamy submitted that an order of conditions would be the appropriate, proportionate, workable and measurable response to the concerns identified by this Tribunal. In making this submission, Mr Ramasamy invited the Tribunal to consider a number of factors, including the following:

- That Dr Mouton has complied with interim conditions for four years, and that these conditions have been proven to work well, with no further complaints arising during this period. Mr Ramasamy submitted that any shortfall in respect of the efficacy of the logs kept by Dr Mouton were not his fault, but due to the wording of his interim conditions.
- That Dr Mouton has begun to show insight and remediation, has undertaken extensive CPD, and has already begun to modify his practice since 2017.
- That Dr Mouton has shown a willingness to engage with the GMC throughout this process.
- That the concerns in Dr Mouton’s case are purely clinical, and therefore remicable.

7. Mr Ramasamy set out a number of conditions that, in his submission, might be workable in Dr Mouton’s case; these included the requirement for a workplace reporter, the requirement for Dr Mouton to keep logs, and the requirement for Dr Mouton to abide by BTA guidelines. Mr Ramasamy further submitted that additional conditions could be imposed specifically addressing Dr Mouton’s treatment of thyroid and paediatric patients, as well as patients with mental health conditions.

8. Mr Ramasamy submitted that the testimonial evidence before this Tribunal shows that Dr Mouton is held in high regard by his patients, and he invited the Tribunal to consider the principles set out in the case of Kamberova v The Nursing and Midwifery Council [2016] EWHC 2955 Admin, submitting that the Tribunal could take Dr Mouton’s interim order into consideration, particularly with regards to insight and engagement. Mr Ramasamy submitted that interim conditions had been proven to be workable, measurable, appropriate, and proportionate in Dr Mouton’s case, and he invited this Tribunal to impose a similar substantive order of conditions on Dr Mouton’s registration, submitting that such an order would serve to adequately address the concerns identified.
The Tribunal’s Determination on Sanction

9. The decision as to the appropriate sanction to impose, if any, is a matter for this Tribunal exercising its own judgment. In reaching its decision the Tribunal has taken account of the SG and has borne in mind that the purpose of a sanction is not to be punitive, although a sanction may have a punitive effect. The Tribunal has applied the principle of proportionality, weighing Dr Mouton’s interests with the wider public interest.

10. In reaching its decision on sanction the Tribunal bore in mind that its primary responsibility is to the statutory overarching objective, which is as follows:

- To protect, promote, and maintain the health, safety, and well-being of the public;
- To promote and maintain public confidence in the medical profession;
- To promote and maintain proper professional standards and conduct for members of that profession.

11. The Tribunal first considered Dr Mouton’s insight into his misconduct. It accepted that Dr Mouton has improved and remediated his clinical practice in some areas, for example in respect of obtaining consent for some genomic tests, his communication with other healthcare professionals, and information displayed on his website. Dr Mouton informed the Tribunal that he could see the benefit of these improvements to both patients and colleagues, and in this respect, the Tribunal was satisfied that Dr Mouton has demonstrated some insight.

12. However, the Tribunal considered that Dr Mouton does not appear to have developed insight into all areas of his misconduct. For example, the Tribunal has seen evidence that Dr Mouton remains dismissive of guidelines, at times referring to them as ‘just’ guidelines, and indicating that he preferred to rely on his own personal experience. The Tribunal further noted that Dr Mouton’s Personal Development Plan (‘PDP’) does not address a number of the specific concerns identified in his case. For example, it does not address the concerns relating to his treatment of patients with mental health conditions. The Tribunal therefore found that Dr Mouton’s insight is developing, but remains incomplete in respect of a number of areas.

13. Having considered insight, the Tribunal next had regard to the aggravating and mitigating factors in Dr Mouton’s case.

Mitigating

14. In mitigation, the Tribunal identified the following factors:
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- The amount of time that has elapsed since the index incidents occurred (between 2013 and 2016) and the fact that Dr Mouton has been subject to interim conditions for a considerable period of time.

- The fact that no patients came to any actual harm as a result of Dr Mouton’s actions.

- The positive testimonials received from patients and other professionals.

- The (albeit limited) extent to which Dr Mouton has changed his practice in respect of thyroid treatments.

- Dr Mouton has some insight into his misconduct.

Aggravating

15. The Tribunal identified the following aggravating factors:

- Dr Mouton’s persistent disregard of guidelines in respect of managing patients with endocrine concerns.

- Dr Mouton’s continued systematic and indiscriminate use of tests that were not clinically indicated.

- The fact that the Tribunal has found serious professional misconduct in relation to a number of different areas of Dr Mouton’s practice.

- The fact that the index incidents involved eight patients, amounting to a pattern of behaviour.

- Dr Mouton’s limited insight, which appears to have developed quite late into the GMC’s investigation and this hearing process.

16. Having considered these factors in the overall context of Dr Mouton’s case, the Tribunal next went on to consider the sanctions available to it, starting with the least restrictive.

No action

17. In coming to its decision as to the appropriate sanction to impose in Dr Mouton’s case, the Tribunal first considered whether to conclude the case by taking no action. The Tribunal determined that, having found Dr Mouton’s fitness to practise to be impaired by reason of serious misconduct, taking no action would not be sufficient to meet the overarching objective. The Tribunal did not consider that there were any exceptional circumstances that would justify taking no action.
18. The Tribunal next considered whether it would be sufficient to impose conditions on Dr Mouton’s registration. In so doing, it bore in mind that any conditions imposed would need to be appropriate, proportionate, workable and measurable.

19. The Tribunal began by asking itself whether it was satisfied that Dr Mouton would comply with any conditions imposed on his registration. Based on the evidence before it (namely the fact that Dr Mouton has complied with interim conditions for over four years), the Tribunal was satisfied that Dr Mouton would not wilfully breach any conditions imposed on his registration at this hearing. However, whilst Dr Mouton has complied with his interim conditions, the interim conditions themselves had not been sufficient to prevent Dr Mouton derogating from guidelines. The level of external scrutiny that Dr Mouton had in place had not been sufficient to identify those occasions when his practice was in breach of the guidelines. The Tribunal therefore considered that any conditions imposed would need to be significantly more prescriptive and accompanied by a sufficient level of supervision to ensure patient safety.

20. Having reached this decision, the Tribunal next went onto consider whether workable conditions of this nature could be formulated. In so doing, the Tribunal found it helpful to group the concerns identified in Dr Mouton’s case into five broad areas: ‘endocrine’ patients, genomic testing, patients with mental health conditions, paediatric patients, and unnecessary tests.

‘Endocrine’ patients

21. Dr Mouton is currently subject to a number of interim conditions that address his treatment of patients with potential endocrine problems. Whilst these conditions have not stopped Dr Mouton derogating from professional guidelines, he has nonetheless complied with the conditions imposed, and has made some changes to his professional practice. For example, the Tribunal noted a shift towards non-prescriptive thyroid support. The clinical logs Dr Mouton has kept have been thorough and professional.

22. The Tribunal considered whether conditions could be a workable way of addressing the outstanding concerns in respect of Dr Mouton’s treatment of patients with endocrine problems. Mr Ramasamy had suggested amendments to the list of interim conditions, including an explicit requirement to follow the BTA guidance, and to log any derogation with a summary of reasons for the derogation; to not commence thyroid treatment (either T3 alone or combination T3/T4 treatment) without discussion and approval by a consultant endocrinologist, and to cease treating any paediatric patient with thyroid medications.
The Tribunal considered that the conditions suggested by Mr Ramasamy would not sufficiently address the failings in Dr Mouton’s practice. The Tribunal has found that Dr Mouton has persistently over-diagnosed thyroid problems, as evidenced by findings of misconduct in relation to his assessment, diagnosis, and management of patients, as well as his interpretation of laboratory findings. The Tribunal considered that the proposed conditions would not address these failings in Dr Mouton’s practice without a significant level of supervision. Given the nature and circumstances of Dr Mouton’s practice, namely as a single-handed GP in the private sector, the Tribunal determined that such supervision would not be workable or feasible.

Accordingly, the Tribunal determined that workable, proportionate, appropriate, and measurable conditions could not be formulated in respect of Dr Mouton’s treatment of patients with ‘endocrine’ conditions.

**Genomic Testing**

Dr Mouton has continued to undertake genomic testing whilst subject to interim conditions. The Tribunal considered that more prescriptive conditions could sufficiently address the outstanding concerns in this area of his practice. These conditions could stipulate which genomic tests Dr Mouton is allowed to carry out, as well as under what circumstances. Conditions would also allow Dr Mouton to carry out the necessary training to remediate his practice, both in respect of what tests he should carry out, and the consent and counselling that should accompany them. The Tribunal was satisfied that a workplace reporter would be sufficient to monitor any such conditions; a workplace reporter does not have to be based at the doctor’s place of work, and would provide regular feedback to the GMC in relation to Dr Mouton’s compliance with any conditions.

Accordingly, the Tribunal determined that workable, proportionate, appropriate, and measurable conditions could be formulated in respect of Dr Mouton’s genomic testing.

**Paediatric patients**

As set out in its determination on impairment, the Tribunal was concerned by Dr Mouton’s ongoing treatment of paediatric patients with potential endocrine issues. The Tribunal considered that these concerns could be met to a degree with a more stringent set of conditions, for example, the condition that Dr Mouton does not undertake particular testing on paediatric patients (including genomic, ultrasound, and urinary thyroid tests), and the condition that he refer any paediatric patient with potential endocrine issues to a consultant paediatrician prior to prescribing nutraceutical supplements or thyroid preparations. The Tribunal considered that
such conditions could be workable. However it was concerned that they may not be measurable insofar as there being a sufficient level of scrutiny.

28. Dr Mouton’s practice has been the subject of external review, but his current level of scrutiny has not been sufficient to prevent Dr Mouton undertaking tests on, and prescribing supplements to, paediatric patients. The Tribunal therefore considered that any workable conditions would need to be accompanied by a higher level of supervision, namely clinical supervision rather than workplace reporting. The Tribunal had regard to the conditions bank, which sets out that any doctor under clinical supervision must not work as a single-handed practitioner. Mr Ramasamy submitted that the Tribunal could adapt the definitions within the conditions bank. However, the Tribunal concluded that Dr Mouton requires a higher level of supervision than merely having his work logs scrutinised by another practitioner. As Dr Mouton works single-handedly in a private clinic, the Tribunal determined that the level of supervision necessary to protect the public would be unworkable.

29. Accordingly, whilst the Tribunal found that conditions may address the outstanding concerns in this area, any such conditions would need to be accompanied by a more stringent level of supervision in order to protect the public. Given the nature of Dr Mouton’s practice, the Tribunal found that supervision of this nature – even at its lowest level – would not be practicable. The Tribunal therefore found that conditions would not be appropriate in respect of Dr Mouton’s treatment of paediatric patients.

Unnecessary tests

30. Similarly to above, the Tribunal determined that the only way to ensure that Dr Mouton uses tests appropriately (ie only when clinically indicated) would be through clinical supervision. The Tribunal determined that a log would not be sufficient in addressing its concerns. For example, in relation to ultrasound testing of the thyroid, such a test might be clinically indicated when there was an abnormal finding on examination of the thyroid gland. However the Tribunal found that such a test is not clinically indicated if its purpose is to assess thyroid function. Given the Tribunal has already found clinical supervision to be unworkable, the Tribunal determined that conditions would not be appropriate in respect of the outstanding concerns relating to Dr Mouton’s use of unnecessary tests.

Patients with mental health conditions

31. As set out in its determination on impairment, the Tribunal found that Dr Mouton lacked knowledge in respect of patients with actual or potential mental health conditions, with Dr Mouton previously failing to recognise when a patient presented with mental health symptoms. The Tribunal considered that, as a GP, Dr Mouton will continue to encounter patients with mental health conditions as part of
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his usual patient cohort, and therefore this continues to be an ongoing area of concern.

32. Mr Ramasamy submitted that this concern could be met with a condition requiring Dr Mouton to incorporate mental health training into his PDP. Whilst the Tribunal agreed that Dr Mouton would benefit from further training in respect of this area of his practice, it was concerned that, at present, Dr Mouton may not be able to recognise when psychiatric input is needed. Any workable condition would be reliant on Dr Mouton recognising when a patient had a mental health component to their condition, and the Tribunal was not satisfied that Dr Mouton has yet remediated this area of his practice to the extent that he would be able to recognise underlying mental health issues in a patient. The Tribunal therefore determined that, until Dr Mouton has undertaken further training in this area, any conditions imposed would be unworkable and potentially unsafe.

The Tribunal’s overall decision on conditions

33. The Tribunal carefully and thoroughly considered the possibility of conditions, and it had regard to both the SG and the conditions bank. It found that some aspects of Dr Mouton’s case could be met by the imposition of conditions. However it also found that conditions would not be appropriate in respect of the majority of the concerns identified. The level of supervision required in order to ensure Dr Mouton was practising safely would not be feasible or workable given the nature of his practice. In respect of patients with mental health or endocrine conditions, the Tribunal found that Dr Mouton required further training in order to practice safely.

34. Even if conditions were workable in all aspects of Dr Mouton’s case, the Tribunal considered that, given the nature of Dr Mouton’s misconduct, they were not sufficient in order to uphold the statutory overarching objective. In his oral evidence at this hearing Dr Mouton continually failed to show that he appreciated the importance of guidelines, and the Tribunal determined that it was necessary to send a signal to the public and the profession marking the seriousness of Dr Mouton’s public and open disregard for guidelines. In this respect, the Tribunal found that conditions would not be sufficient or appropriate.

35. Bearing all of the above in mind, the Tribunal determined that conditions would not be appropriate, workable, measurable, or proportionate in the overall context of Dr Mouton’s case.

Suspension

36. Having determined that conditions would not be appropriate, the Tribunal next considered whether an order of suspension would be sufficient to uphold the statutory overarching objective. In so doing, it had regard to the SG, particularly to paragraphs 91 and 92, which set out that:
'Suspension has a deterrent effect and can be used to send out a signal to the doctor, the profession and public about what is regarded as behaviour unbefitting a registered doctor... Suspension will be an appropriate response to misconduct that is so serious that action must be taken to protect members of the public and maintain public confidence in the profession. A period of suspension will be appropriate for conduct that is serious but falls short of being fundamentally incompatible with continued registration...’

37. The Tribunal considered that suspension would be an appropriate response to Dr Mouton’s misconduct, in that it would serve to mark the seriousness of his actions as discussed previously, and would also send a message to the public and the profession about the importance of having proper regard to regulatory guidelines. The Tribunal has found that Dr Mouton’s misconduct is remediable, and that he has begun to show insight and to change his practice. In this way, the Tribunal determined that Dr Mouton’s misconduct is not so serious that it is fundamentally incompatible with continued registration.

38. The Tribunal further had regard to paragraph 97 of the SG, which sets out that suspension may be appropriate in cases where the doctor has shown insight and a willingness to engage. As set out above and earlier in the determination, the Tribunal has found that Dr Mouton has begun to develop insight, and has willingly engaged with his interim conditions and with the GMC process.

39. Bearing the above in mind, the Tribunal determined that an order of suspension was the appropriate and proportionate response to Dr Mouton’s misconduct.

**Duration**

40. The Tribunal determined that a period of nine months’ suspension would be appropriate. It considered that nine months would allow Dr Mouton time to remediate the outstanding concerns, particularly in relation to mental health, his prescribing of thyroid hormones in breach of the relevant guidelines, his use of tests that were not clinically indicated, and his practice in relation to paediatric patients and genomic testing. Nine months should also allow Dr Mouton time to demonstrate evidence of remediation and to establish himself in a group practice that is able to provide strong foundations in governance and quality assurance, thereby enabling Dr Mouton to look to returning to work in a more supervised environment.
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Review

41. A Tribunal will convene to review Dr Mouton’s case shortly before the end of the period of suspension. Any future Tribunal reviewing Dr Mouton’s case may be assisted by evidence of the following:

- Training in mental health medicine to improve his practice in assessing, diagnosing, and managing patients with mental health conditions;

- Training in endocrine medicine to improve his practice in assessing, diagnosing, appropriately investigating, accurately interpreting investigation results, and managing patients with endocrine conditions;

- Training in genomic testing to improve his practice in the correct use of such tests, with appropriate informed consent and counselling.

42. In the Tribunal’s view, the best evidence of achieving the above might be by way of clinical attachments and focussed CPD.

DETERMINATION ON IMMEDIATE ORDER - 27/09/2019

1. Having determined to suspend Dr Mouton’s name from the Medical Register for a period of nine months, the Tribunal next considered whether Dr Mouton’s registration should be made subject to an immediate order of suspension.

On behalf of the GMC

2. Mr Atherton submitted that, given the Tribunal’s findings in relation to impairment and sanction, it would not be appropriate for Dr Mouton to continue in unrestricted practice pending the substantive order of suspension taking effect. He therefore submitted that it was necessary to impose an immediate order of suspension on Dr Mouton’s registration.

On behalf of Dr Mouton

3. Mr Ramasamy invited the Tribunal to consider, on the evidence before it, whether the risk identified in Dr Mouton’s case was such that an immediate order was necessary in order to protect patients. Mr Ramasamy submitted that the Tribunal was not faced with a ‘binary’ decision between no restriction and between an immediate order of suspension, submitting that a more proportionate response would be to leave the current interim order of conditions in place on Dr Mouton’s registration pending the substantive order taking effect. Mr Ramasamy took the Tribunal to Rule 17 of the General Medical Council (Fitness to Practise) Rules 2004, as amended (‘the Rules’), submitting that the Rules granted the Tribunal a discretion.
to leave the interim order in place. Mr Ramasamy submitted that the current interim order would guard against any risk posed by Dr Mouton pending the substantive order taking affect, and he invited the Tribunal to consider a number of factors in support of this submission, including the following:

- The Tribunal’s finding that Dr Mouton would not wilfully breach any conditions;
- The fact that the Tribunal’s decision on impairment was handed down in July of this year, and that the Tribunal at that stage chose not to make an interim order of suspension;
- The fact that a recent Interim Orders Tribunal was provided with this Tribunal’s findings on impairment, but chose to leave the current interim order of conditions in place;
- The fact that no patients have come to any harm as a result of Dr Mouton’s actions.

**The Tribunal’s decision**

4. In making its decision the Tribunal has exercised its own judgment, and has taken account of the principle of proportionality. The Tribunal noted that it may impose an immediate order where it is satisfied that it is necessary for protection of members of the public, is in the public interest, or is in the best interests of the practitioner.

5. The Tribunal carefully considered Mr Ramasamy’s submissions, and it accepted that it was not required to revoke the interim order at this stage as a point of law.

6. The Tribunal made certain findings at sanction stage as to what sanction it believed necessary in order to guard against the risks identified. The Tribunal found, at sanction stage, that even under the current interim conditions Dr Mouton continued to pose a risk to patients. The Tribunal accepts that no patients have come to any direct harm as a result of Dr Mouton’s actions, however it has found that patients have been subject to unnecessary tests, suboptimal practice, and have been left in a vulnerable state as a result of gaps in Dr Mouton’s knowledge and his failure to abide by guidelines. The ongoing risks posed by Dr Mouton arise from an absence of skills in assessment, diagnosis, and management of patients, and are evidenced, for example, by his treatment of Patient A, where he failed to identify and act upon mental health concerns.

7. The Tribunal accepts that it did not impose an immediate order of suspension after reaching its decision on impairment. However the Tribunal has now considered the case as a whole, and has specifically addressed the issue of sanction in detail. In its decision on sanction the Tribunal identified a number of key areas in which Dr
Mouton requires further training and remediation in order to protect patients. It found that the current interim order of conditions have not removed the risks posed by Dr Mouton, with Dr Mouton failing to (as an example) provide proper counselling to paediatric patients in respect of genomic tests and continuing to subject patients to tests that were not clinically indicated, even whilst under interim conditions. Dr Mouton has been aware of the Tribunal’s findings on impairment since July of this year, but it was not provided with any evidence to suggest that he has changed his practice in the intervening period such that any ongoing risk has been negated.

8. Given the Tribunal’s findings that the current interim order of conditions (or indeed any order of conditions) would not be sufficient in order to protect the public, the Tribunal similarly determined that, notwithstanding the effect it will have on Dr Mouton’s practice, an immediate order of suspension was necessary in order to provide full protection for members of the public pending the substantive order taking effect.

9. This order means that Dr Mouton’s registration will be suspended from the time when notification of this decision is deemed to have been served on him.

10. The substantive direction for suspension, as already announced, will take effect 28 days from when written notice of this determination has been served upon Dr Mouton, unless an appeal is made in the interim. If an appeal is made, the immediate order of suspension will remain in force until the appeal has concluded.

11. The interim order currently imposed on Dr Mouton’s registration will be revoked when the immediate order takes effect.

12. That concludes Dr Mouton’s case.

Confirmed
Date 27 September 2019

Mr Charles Thomas, Legally Qualified Chair
## Schedule 1 – Patient B

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<td>14 September 2014</td>
<td>Saccharomyces boulardii</td>
</tr>
<tr>
<td>14 September 2014</td>
<td>Curcumin 95 (Tumeric)</td>
</tr>
</tbody>
</table>
Record of Determinations – Medical Practitioners Tribunal

Schedule 2 – Patient C genetic testing

DIO2 genotype testing;
Apo E genotype testing;
MTHFR genotype testing;
LCT genotype testing;
FUT2 genotype testing;
CTLA4 genotype testing;
VDR genotype testing.
<table>
<thead>
<tr>
<th>Date</th>
<th>Details of Prescriptions/Treatments</th>
</tr>
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<tbody>
<tr>
<td>7 December 2014</td>
<td>B-Complex Plus</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>B-Complex including pyridoxine &amp; thiamine</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Methyl-B12</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Riboflavin 5-phosphate (Vitamin B2)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Mediopro vegan (Protein Powder)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Vitamins D &amp; K</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Hemp seed oil</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Ultra GLA (Omega 6 fatty acids)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Original 68</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Curcumasorb</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Iodine 225mg &amp; Tyrosine 500mg</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Armour thyroid</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>PGNLG (pregnenolone 50mg)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Ashwagandha extract</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>LycPe (carotenoid)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Selenium and selenomethionine</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Resveratrol</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Zinc</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Bio AE emulsion</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>B-Complex</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Fer (Iron and Vitamin C)</td>
</tr>
<tr>
<td>Date</td>
<td>Product</td>
</tr>
<tr>
<td>------------</td>
<td>----------------------------------------------</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Vitamin D3</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Vitamin K2</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Calcium/magnesium malate</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Hemp oil seed</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Ultra GLA (Omega 6 fatty acids)</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Armour thyroid</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>l-thyroxine</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Ashwagandha extract</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Rhodiola rosea</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Lycosorb</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Vitamin A</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>NAC</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Original 68</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Curcuma</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Griselfuline</td>
</tr>
<tr>
<td>11 July 2015</td>
<td>Gabbroral</td>
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</table>
**Record of Determinations – Medical Practitioners Tribunal**

**Schedule 4 – Patient D**

<table>
<thead>
<tr>
<th>Date</th>
<th>Details of Prescriptions/Treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td>7 December 2014</td>
<td>Vitamin B-complex</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Methylcobalamin (vitamin B12)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Vitamin-D3 (5000IU/day)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Magnesium powder</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Vitamin K2</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Mega-GLA complex (Omega-6 fatty acids)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>PGNLG (pregnenolone 100mg &amp; magnesium glycinate 100mg)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Novothyral (100ug T4 &amp; 20 ug T3)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Ashwagandha extract</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Zinc</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Copper</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>N-Acetyl Cysteine</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Coenzyme Q10</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Cod liver oil (Vitamin A, EPA &amp; DHA)</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Mega GLA complex</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Havitall</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Curcumasorb</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Allimed</td>
</tr>
<tr>
<td>7 December 2014</td>
<td>Ayu-Neem-Chlorofyl</td>
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### Record of Determinations –
Medical Practitioners Tribunal

#### Schedule 5 – Patient E

<table>
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<tr>
<th>Date</th>
<th>Details of Prescriptions/Treatments</th>
</tr>
</thead>
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<tr>
<td>27 September 2014</td>
<td>B-complex</td>
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<tr>
<td>27 September 2014</td>
<td>Vitamin-D (cholecalciferol 5000IU/day)</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Vitamin D3 &amp; K</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Magnesium Magnum</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Thyrocsin (iodine &amp; aswagandha extract - to support thyroid function)</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Tirform</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Pregnenolone 50mg</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>BTA</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Evening Primrose</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Hemp seed oil</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Zinc</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Gamma E 300</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Bio AE emulsion</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>NAC 900 (N-Acetyl Cysteine)</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Vitamin A</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>L-glutamine</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Curcumin 95 (Turmeric)</td>
</tr>
<tr>
<td>27 September 2014</td>
<td>Eliminex</td>
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<tr>
<td>3 February 2015</td>
<td>Iron &amp; vitamin C</td>
</tr>
<tr>
<td>3 February 2015</td>
<td>Vitamin-D (cholecalciferol 5000IU/day)</td>
</tr>
<tr>
<td>Date</td>
<td>Supplement</td>
</tr>
<tr>
<td>------------</td>
<td>-----------------------------------</td>
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<tr>
<td>3 February</td>
<td>Vitamin K2</td>
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<tr>
<td>3 February</td>
<td>Magnesium glycinate</td>
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<tr>
<td>3 February</td>
<td>Vitamin B-complex Plus</td>
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<tr>
<td>3 February</td>
<td>Flax seed oil</td>
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<td>3 February</td>
<td>Hemp seed oil</td>
</tr>
<tr>
<td>3 February</td>
<td>Novothyral (100ug T4 &amp; 20 ug T3)</td>
</tr>
<tr>
<td>3 February</td>
<td>Pregnenolone 50mg</td>
</tr>
<tr>
<td>3 February</td>
<td>Thyrocsin (iodine &amp; aswagandha extract - to support thyroid function)</td>
</tr>
<tr>
<td>3 February</td>
<td>Lycopene</td>
</tr>
<tr>
<td>3 February</td>
<td>NAC 900 (N-Acetyl Cysteine)</td>
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<tr>
<td>3 February</td>
<td>Zinc</td>
</tr>
<tr>
<td>3 February</td>
<td>Vitamin A</td>
</tr>
<tr>
<td>3 February</td>
<td>Boulardii</td>
</tr>
<tr>
<td>3 February</td>
<td>Curcuma Longa-Extract (Turmeric)</td>
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<tr>
<td>3 February</td>
<td>Berbercap</td>
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<tr>
<td>12 July</td>
<td>Vitamin B-complex Plus</td>
</tr>
<tr>
<td>12 July</td>
<td>Thiamine</td>
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<tr>
<td>12 July</td>
<td>Vitamin-D3 (cholecalciferol 2500IU/day)</td>
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<td>12 July</td>
<td>Cal Mag 80/40</td>
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<tr>
<td>12 July</td>
<td>Huile de Bourrage</td>
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<tr>
<td>12 July</td>
<td>Hemp seed oil</td>
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<td>12 July</td>
<td>Max DHA</td>
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</tbody>
</table>
Record of Determinations –
Medical Practitioners Tribunal

12 July 2015  Novothyral (100ug T4 & 20 ug T3)
12 July 2015  Ashwagandha extract
12 July 2015  Zinc 30
12 July 2015  Vitamin A
12 July 2015  NAC 900 (N-Acetyl Cysteine)
12 July 2015  L-selenomethionine
12 July 2015  Boulardii
12 July 2015  Curcuma
12 July 2015  Oregano Oil
12 July 2015  Allimed
Schedule 6 - Patient H

GTAEN natural thyroid extract

Pregnenolone