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2<sup>nd</sup> January 2007

General Medical Council,  
5th Floor,  
St James's Buildings,  
79 Oxford Street,  
Manchester,  
M1 6FQ

Dr. Gordon Skinner

Dear Madam,

I was diagnosed with thyroid failure [redacted]  
[redacted] I had originally been diagnosed with [redacted] until I  
learned of the family history of thyroid problems, only then was I offered a TSH test-  
reluctantly.  
A TSH of [redacted] confirmed that this was indeed the problem and I was prescribed  
thyroxine. [redacted]

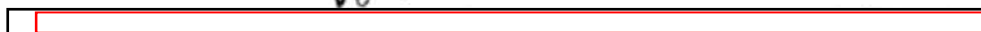
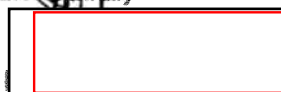


I learned more about thyroid disease from the internet and asked my GP if terroxin  
might help. He did not even bother to answer, but showed me the door. I was  
insulted and decided to consult a doctor in the private sector who might take me  
more seriously.

The difference that a change of medication from thyroxine to Armour natural thyroid  
and Tertroxin made was little short of miraculous. All my symptoms went over the  
next [redacted] weeks, [redacted]



I feel that Dr Gordon Skinner's approach to thyroid disease is essential to my  
continued wellbeing. There is little point in medication that does not relieve  
symptoms, even if blood tests are within the reference interval, it is quality of life that  
is vital to the patient, and I did not get this from my GP.

Yours faithfully







--




  
Ms Patricia Collins  
Investigation Officer  
Fitness to Practice Directorate  
General Medical Council  
5th Floor St James's Buildings  
79 Oxford Street  
Manchester M1 6FQ

Re Dr. Gordon R B Skinner

Dear Ms. Collins,

I would like to submit further evidence in support of Dr. Skinner and many other doctors who adopt a similar approach to patient care.

First a subjective statement. In  just before my first consultation with Dr. Skinner I was profoundly hypothyroid (see my previous letters) and my declining condition led me to believe I would survive less than five years. At the age of  I now have a realistic 30 year horizon, my cholesterol is low, I am fit and healthy.

The problem I encountered was that I was hypothyroid with a normal thyroid profile, that is to say my thyroid hormones (known as T3 and T4) were in the middle of their respective reference intervals. My TSH of  was ideal. I was told I am not hypothyroid, couldn't possibly be, because the numbers said so! I certainly was hypothyroid and it took a good honest doctor to say so.   


Clearly the question of being hypothyroid with normal numbers is the crux of the matter. Many (but not all) endocrinologists have a wholly irrational belief that a patient with a "normal" level of thyroid hormone in their blood cannot be hypothyroid and should not receive treatment; this is the position of the British Thyroid Association. By some magic, thyroid hormones will always act as they should, none of the mechanisms of thyroid hormone action will ever fail.

The situation is further confused by an absolute faith in TSH measurements. This hormone, produced by the pituitary, stimulates the thyroid to produce thyroid hormones. TSH increases exponentially and inversely to the T3 and T4 levels in the blood. Thus TSH is a marker for thyroid hormone activity - **in the pituitary**. Of course a marker for pituitary hormone activity is not a marker for other tissues which have different receptor types and cofactors. An elevated TSH can of course point to a failing thyroid, a normal TSH does not unfailingly indicate an euthyroid patient.

This blind faith in the numbers, along with a little arrogance, harms patients and is demonstrated by the quotes that follow:-

**Toft AD, Beckett GJ. Thyroid function tests and hypothyroidism  
BMJ. 2003 Feb 8;326(7384):295-6**

Some patients seeking an explanation for feeling "below par" are disappointed when thyroid function tests are normal. Unable to accept that there may be psychosocial reasons for their symptoms, a vociferous minority believe that hypothyroidism may exist with normal serum concentrations of both thyroxine (T4) and thyroid stimulating hormone (TSH).

**Weetman AP. Whose thyroid hormone replacement is it anyway?  
Clin Endocrinol (Oxf). 2006 Mar;64(3):231-3.**

Nothing seems more straightforward than treatment of hypothyroidism. We have robust assays to diagnose the condition and an effective replacement in the form of synthetic thyroxine. However, the field appears to be in some turmoil and clinical endocrinologists are under increasing pressure from disaffected patients who believe their symptoms indicate hypothyroidism despite normal thyroid function tests. ....  
The majority of patients who demand thyroid hormone treatment for multiple symptoms, despite normal thyroid function tests, have functional somatoform disorders, which in the postmodern world can understandably be misdiagnosed as hypothyroidism.

**In October 2005 I attended a "Thyroid Update" at the Royal Society Medicine where Dr Graham Beastall made the following comment:-**

"I think there are very few, in fact zero cases of a thyroid disorder with a normal thyroid profile".

**During a question and answer session an endocrinologist asked:-**

"what do I do about all the nutty psychologists who want me to test demented patients who inevitably end up with a normal thyroid profile?"

Clearly patients such as myself not only have to overcome hypothyroidism, but also psychosocial and functional somatoform disorders along with concurrent dementia. It's important to note that these opinions are not based on evidence; other than Dr. Skinner's research there has been only one small research project led by a Glasgow GP:-

**Pollock MA et al. Thyroxine treatment in patients with symptoms of hypothyroidism but thyroid function tests within the reference range: randomised double blind placebo controlled crossover trial.  
BMJ. 2001 Oct 20;323(7318):891-5.**

This trial had a number of limitations, some of which I noted in my rapid response "More Elegant Research Needed" which is available on the BMJ website.



In summary, Dr. Skinner's opponents assert that patients with multiple signs and symptoms of hypothyroidism and a normal thyroid profile should not receive a trial of thyroid hormone supplementation. This is predicated on the assumption there are zero hypothyroid patients with thyroid blood tests within the 95 percent reference interval. This assumption is not based on evidence, these doctors have refused to treat and failed to conduct research into this issue.

I would now like to comment on the way the Fitness to Practice and Interim Orders have proceeded. There was to have been a FTP hearing in January this year, this was subsequently cancelled. We now have a further IOP at short notice. These proceedings cause great anxiety and disruption to Dr. Skinner and his patients. Many patients who would like to attend the 26th February IOP are unable to get time off work at short notice or afford transportation, they are reliant on reduced cost advanced booking. [REDACTED]

[REDACTED] I appreciate the GMC has a very difficult schedule to juggle but request that any further attempts at procrastination are refused. The doctors who have complained about Dr. Skinner have managed to create major disruption for the doctor and his patients with little effort. I ask that this issue is addressed after the FTP hearing.

Thank-you very much for taking the time to read this letter.

Yours faithfully,

[REDACTED]

## TESTIMONY

[Redacted]

To: Patricia Collins [Investigation Officer]  
Fitness to Practice Directorate, General Medical Council  
5<sup>th</sup> Floor, St. James's Buildings, 79 Oxford Street  
Manchester M1 6FQ  
16<sup>th</sup> February 2007

Dear Ms Collins

**Testimony in support of Doctor G R B Skinner MD (Hons) DSc FRCPath FRCOG**

I am writing this testimony to you today in support of the above named doctor. Both my daughters have been treated by Dr. Skinner for hypothyroidism and thanks to him, they have both been returned from serious debilitating ill health to full health. I cannot speak highly enough of him.

I have followed the IOP hearings in relation to this excellent doctor with keen interest and I have written to Professor Sir Graeme Catto on several occasions voicing my dismay at the proceedings thus far, which have now spanned 3 years. I am still strongly of the opinion that Dr. Skinner should not have been brought before the IOP panel or had an interim order made against him. For ease of reference, I am copying to you the letters I have written to Professor Catto, rather than repeat my sentiments here and trust that you will read these through prior to the next IOP which I understand is to take place on Monday the 26<sup>th</sup> of February 2007 [at very short notice] at your Manchester offices.

However to summarise, I am convinced that Dr. Skinner does not present any risk to himself, members of the public, the public interest and certainly not to his patients. I can say this with confidence having met and corresponded with a large number of Dr. Skinner's patients. [Redacted] all of whom have the highest regard, faith and trust in him. Furthermore, from the hearings I have attended and the transcripts I have received of said hearings, the issue here is a slight difference of opinion on how hypothyroid patients should be treated [something which I understand is outside the remit of the GMC] rather than complaints from patients. Indeed the complaints appear to be in the main from endocrinologists. From first hand experience, I know that Dr. Skinner takes a holistic approach with his patients [researching all aspects of the patient's medical history in full, carrying out full clinical examination and blood tests etc] and it is this attention to detail and individual and meticulous approach which makes such a difference to Dr. Skinner's patients and why his treatments are such a success.

Therefore, instead of these endocrinologists expending energy on complaints and all the bureaucracy that goes with them, they might be better employed expending such energy on investigating why Dr. Skinner's patients get well and why his treatments are so successful! Can I also say that Dr. Skinner has never represented himself as an endocrinologist as has been alleged. When my daughters asked to be referred to him, it was because of his reputation as an excellent, caring and humane doctor who made people well [word gets around you know!]. That is precisely what he turned out to be, a caring doctor who listened, who carried out a full clinical examination and who considered presenting symptoms, family and medical history as well as blood tests before prescribing a course of treatment.

One of the things I have found deeply disturbing about this whole business is the lack of concern and compassion [by the GMC] of the plight of the patients in all this. Dr. Skinner has several thousand patients on his books, hundreds of whom have submitted testimonials in support of him. Such large numbers of people can't be wrong about this doctor and their views must count in his favour. I therefore look forward to your urgent confirmation that this testimonial will be used in support of Dr. Skinner at the forthcoming IOP [26<sup>th</sup> of February 2007] and for any subsequent IOPs and any Fitness to Practice hearing should this occur.

Yours sincerely

[Redacted]

Copy



17<sup>th</sup> February, 2007

Ms. P Collins  
Investigation Officer  
Fitness to Practise Directorate,  
General Medical Council,  
5<sup>th</sup> Floor,  
St James's Buildings,  
79 Oxford Street,  
Manchester,  
M1

Dear Ms. Collins,

Testimonial: Interim Orders Panel  
Hearing of Dr. Gordon R.B. Skinner  
Monday 26<sup>th</sup> February, 2007

In the eyes of the GMC, why is it such a crime for Dr. Skinner to make his patients well?

I have written to the GMC on many occasions now and I wish to re-iterate that I believe Dr. Gordon Skinner to be one of the most competent doctors I have ever consulted.

I also believe that what the GMC and members of the medical establishment are trying to do to him is a travesty of justice!

He is NOT a danger to the public as I and thousands of other patients will tell you. It appears that doctors are the ones who wish to see him struck off. To be a fair trial, the GMC should urgently consult with the Crown Prosecution Service, because what I have seen at the previous IOP hearings is nothing short of a sham.

Even when Dr. Skinner asked to speak at the June 06 hearing, he was quite rudely and abruptly put down by the chairman, with a brusque 'no you may not'. To make matters worse, this was left out of the transcript! Apparently though, according to the GMC, he should be allowed to speak if he wishes.

I am absolutely certain a judge would be appalled at the behaviour of the GMC.

With regard to this hearing, I believe that once again that Dr. Skinner has been given short notice for this hearing, thus not giving him the 21 days notice to comply with condition 3. How can this be allowed?

In the past, hypothyroidism was always diagnosed on clinical signs and symptoms - doctors didn't have blood tests to rely on then, so why all these problems now. Is it down to professional jealousy that, in other words, Dr. Skinner has succeeded in making patients well when the specialist endocrinologists have failed?

*A quote from Depression and Your Thyroid by Gary S. Ross, MD and Peter J. Bieling, PHD.  
'Low thyroid can be an overlooked, undiagnosed cause of depression. We know that people*

may suffer for years from depression, moodiness, and sluggish thinking either because their thyroid function is never taken into account or because their standardized thyroid tests are returned as 'normal'. For countless people, an underlying hypothyroid condition remains undiagnosed, untreated, life-limiting, and disabling, often becoming the springboard for further illness. According to Haggerty et al. (1993), patients with subclinical hypothyroidism are reported to have a lifetime prevalence of depression of 56 percent compared to a prevalence of 20 percent among depressed patients who do not have hypothyroidism.

*Until recently, there has been a strict reliance on certain basic blood tests and clinical criteria to evaluate the functioning of the thyroid. However, these tests and criteria do not always demonstrate the whole picture of the body's need for thyroid. Although it's true that not all patients with depression are hypothyroid and not all patients diagnosed as hypothyroid are depressed, it has become apparent that properly diagnosing and treating low thyroid can dramatically change a patient's life for the better.'*

The GMC says 'that patients must be able to trust doctors with their lives and well-being. To justify that trust, we as a profession have a duty to maintain a good standard of practice and care and to show respect for human life'.

**Dr. Skinner follows the above criteria to the letter!**

The doctors, who have written to complain about Dr. Skinner, would do well to listen to their patients and to read the extensive literature that there is on the subject.

I am a lay researcher and have accumulated files of information, so I am sure these doctors could do the same. The same applies to your panellists on the IOP and FTP panels.

I suggest you look at the evidence in Dr. Skinner's favour – after all, the diagnosis of hypothyroidism is now being questioned world-wide.

If I or my two daughters' medication is stopped or changed because of the action of the GMC, I will seek legal advice as to action that can be taken against the GMC.

The GMC is failing in its duty of care to patients by attacking a doctor doing such valuable work.

Our health depends on Dr. Skinner and others like him.

This outrage should be brought to an end and quickly.

'Justice delayed is justice denied'! William Gladstone.

Yours sincerely,







17<sup>th</sup> February, 2007

Ms. P Collins  
Investigation Officer  
Fitness to Practise Directorate,  
General Medical Council,  
5<sup>th</sup> Floor,  
St James's Buildings,  
79 Oxford Street,  
Manchester,  
M1

Dear Ms. Collins,

Dr. Gordon Skinner  
IOP Hearing  
Monday 26<sup>th</sup> February, 9.30am

☐ years ago, if it had not been for Dr. Skinner diagnosing me with hypothyroidism, I don't know what the future would have held for me.

I always had a sunny disposition, but gradually that changed ☐

☐

Today all those symptoms are a thing of the past and I now hold down a very successful job ☐ If it hadn't have been for Dr. Skinner, I would have almost certainly had to live off benefits and be a drain on society.

☐

☐ GP's really should read medical papers that indicate some patients, especially the young, can lose weight when they are suffering from hypothyroidism.

Thanks to Dr. Skinner, the improvement in my health was rapid once on thyroid hormone replacement, but I only regained full health when I changed from Thyroxine to Armour Thyroid.

When my treatment was passed from Dr. Skinner to my GP, I sent a letter to Dr. Skinner thanking him for his diagnosis and treatment and also saying that hopefully, in the not too distant future, the medical profession would realise that reliance on blood tests, was blighting peoples' lives.

Dr. Skinner is a saviour to thousands of patients and I do hope that the GMC will come out in favour of Dr. Skinner and his patients and don't cave in to a few doctors who disagree with his very successful treatment of patients.

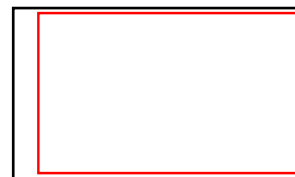
Yours sincerely,

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19th February 2007.

The Adjudicator,  
Interim Orders Panel,  
The General Medical Council,  
St. James Building,  
79 Oxford Street,  
Manchester,  
M1 6FQ

Dear Sir,

I write regarding the G.M.C. calling Dr. Gordon R. B. Skinner, MD (Hons)  
D.Sc., FRCPATH, FRCOG, to a Public Hearing on the 26th February.

I write in support of Dr. Skinner in order that he is allowed to carry on his practice in helping people like me who have struggled for so long to get adequate treatment for Hypothyroidism on the N.H.S.

I have been a patient of Dr. Skinner since  and since that time he has restored me to full health. He has always treated me in a courteous, kindly and respectfully thoughtful manner and his consideration of my condition since my first appointment in  helped me to obtain the health I have now.

I am now  years old but was registered with underactive thyroid in  I had been suffering from the condition for a considerable time before that. After being finally diagnosed I struggled for  long painful years under N.H.S. treatment before a chiropractor advised me to seek better treatment.

I write in support of Dr. Skinner at this Public Hearing.

Yours faithfully,

*Dr. Gordon R. B. Skinner*  
*MD (Hons) D.Sc. FRCPATH FRCOG*



16<sup>th</sup> February, 2007

Ms. P Collins  
Investigation Officer  
Fitness to Practise Directorate,  
General Medical Council,  
5<sup>th</sup> Floor,  
St James's Buildings,  
79 Oxford Street,  
Manchester,  
M1

Dear Ms. Collins,

Testimonial: Interim Orders Panel  
Hearing of Dr. Gordon R.B. Skinner  
Monday 26<sup>th</sup> February, 2007

I am writing in support of Dr. Gordon Skinner as I have done at each of the past IOP hearings.

It is my opinion that the accusations against him are entirely wrong, as I have received nothing but 100% professional treatment from him.

During a recent diagnosis of rheumatoid arthritis, although now under the care of my GP for hypothyroidism, I did write to him to inform him of my condition and he has given me support beyond his duty of care, telephoning to keep up to date with my diagnosis and treatment.

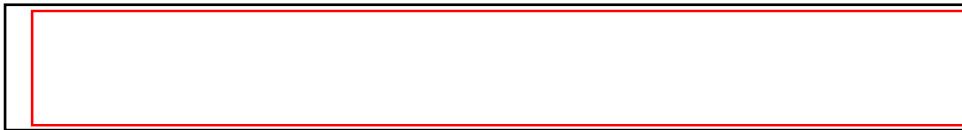
I trust you will take into consideration the many testimonials that you receive from patients.

Yours sincerely,



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Investigation Officer,  
Ms Patricia Collins  
Fitness to Practice Directorate,  
General Medical Council,  
5<sup>th</sup> Floor.  
St James's Buildings  
79 Oxford Street,  
MANCHESTER M1 6FQ

15 February 2007

Dear Ms Collins,

**Re: Dr Gordon Skinner – IOP Hearing 26 February 2007**

I enclose two A4 stapled sheets *which must be included together for this Hearing*. One is a testimonial for Dr Skinner and the other a list of the signs and symptoms.

I would be grateful if you would acknowledge receipt of these documents and your assurance that they will be presented together in material for this Hearing, as requested.

Many thanks.

Yours sincerely,



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**TESTIMONIAL: DR GORDON R B SKINNER – IOP HEARING 26 FEBRUARY 2007**

I would defend Dr Skinner with my own life such is my indebtedness to him for restoring me to health after eighteen years of neglect and indifference from NHS doctors.

I have spent much of my time since my recovery defending his right to practice, and attempting to bring to the notice of the GMC where the real fault lies. I have been greatly assisted in this matter by my MP and by his many colleagues in the House of Commons who are now recognising the dubious state in which thyroid medicine languishes in the NHS and its massive cost in human terms – AND – economically, as a consequence of the widespread misdiagnosis of *hypothyroidism*.

I, like countless thousands of patients in the UK, fell foul of the Reference Interval in the Thyroid Function tests, which in my case, always supposedly showed me to be 'within normal limits'. In line with the majority of his NHS colleagues, my GP's slavish devotion to that blood test meant that I was consistently denied adequate treatment with Thyroxine. He was blind to the very obvious clinical signs and symptoms that pointed conclusively to *hypothyroidism*. [Please see separate sheet]. He was supported in this falsity by the Endocrinologist to whom I was referred, who added insult to injury by diagnosing ME/CFS 'with no further treatment available'. Thus was I condemned to [ ] years of serious ill health and [ ]

What a nightmare!

My refusal to accept this erroneous diagnosis was assisted by my own professional status. [ ]

When things couldn't have been worse it was my great good fortune to find Dr Skinner. Despite his views, my GP referred me. The relief of my husband and myself was intense. Here was a completely professional man of great integrity and experience who clearly had a total grasp of his subject. He listened at length, was thorough in his examination and evaluated thoughtfully the treatment regime which would be required. His prediction that I would begin to feel better quite quickly and be stable within [ ] months was entirely accurate. Having never been examined at all by either my GP or my Consultant, you can imagine the impact Dr Skinner had upon me. After so long being so ill the prospect of recovery was almost beyond belief.

How did he bring my [ ] years of misery and neglect to an end? By some miracle cure? NO! It was all so simple and cost effective with nothing more dramatic than an adequate dosage of Thyroxine. My regime was one of gradually increasing levels of Thyroxine. When stability was achieved and I was WELL, the dose was gradually reduced until a level at which I remained thus, was sustained. It was so very logical, harmless and EFFECTIVE! Dr Skinner should be lauded and honoured for his perspicacity, revered and followed by the medical profession for his SUCCESS. HE restores patients to health where the great majority of doctors are cowed and fearful of the influence of a powerful minority lobby of Endocrinologists and Psychiatrists who prefer to maintain this long held, iniquitous status quo for their own gain. The wellbeing of the patient has no part in this monopoly.

[ ]  
Eternally grateful patient of Dr Skinner  
15 February 2007

**SIGNS AND SYMPTOMS OF HYPOTHYROIDISM EXPERIENCED FOR [ ] YEARS,  
PRIOR TO DIAGNOSIS.**

**ALL THIS HIDEOUS NIGHTMARE, [ ], WAS  
COMPLETELY CURED BY CORRECT AND ADEQUATE DOSES OF THYROXINE,  
PRESCRIBED BY DR GORDON SKINNER, [ ] YEARS LATER.**

**IS IT REALLY REASONABLE FOR HUGE NUMBERS OF PEOPLE TO *SUFFER* LIKE  
THIS - *BECAUSE THEY DO* - JUST BECAUSE DOCTORS ARE IGNORANT OF THE  
BASIC ELEMENTS OF *HYPOTHYROIDISM*??? *THEY ARE TOO READY TO DIAGNOSE  
ME/CFS AND THEN SIT ON THEIR HANDS AND DO NOTHING, THUS TOTALLY  
FAILING TO ADEQUATELY DIAGNOSE, TREAT AND CARE FOR THEIR PATIENTS.  
IT IS DISGRACEFUL, IN THIS DAY AND AGE, THAT SUCH AN EASILY TREATABLE  
CONDITION IS IGNORED. IT IS A SHAMEFUL STATE OF NEGLECT WHICH IS  
CONDONED BY THE PROFESSION AS A WHOLE BY THEIR FAILURE TO ADDRESS  
IT.***

..... Imagine if this was a member of your family. Would you not feel helpless in the face of such blatant indifference to their plight .....?

[ ]  
15 February 2007



16 February 2007

Patricia Collins  
Investigation Officer  
Fitness to Practice Directorate  
General Medical Council  
5<sup>th</sup> Floor, St James's Buildings  
79 Oxford Street  
MANCHESTER M1 6FQ

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Dear Patricia Collins

**Re: Dr Gordon Skinner - IOP 26 February 2007**

My wife suffered with hypothyroidism for  YEARS !

But her Blood Test results indicated 'Normal'.

Despite her displaying a catalogue of symptoms characteristic of the illness, her GP as a consequence refused her appropriate treatment.

This cannot be right, can it?

There has to be something wrong with the present NHS guidelines to GPs if these result in a prime slice of someone's life being ruined - entirely unnecessarily.

***All that was required was adequate doses of Thyroxine!***

***And a doctor who acknowledged the obvious indications of the variety of symptoms which my wife displayed and gave her the appropriate treatment: Dr Skinner.***

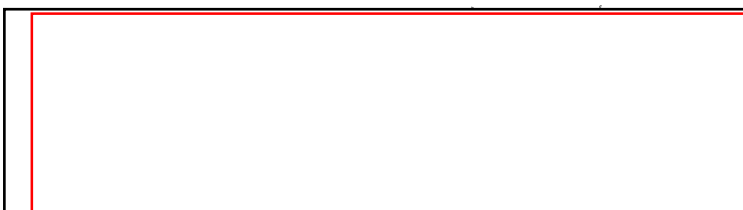
Both she and I are totally indebted to him. But not just us; our daughter as well. And, as we have discovered in recent times, many thousands of others, who have regained healthy lives as a result of his care.

Please acknowledge the skill, judgment and expertise of Dr Skinner. Alter the hypothyroid blood test guidelines now. And let Dr Skinner continue his good work without being repeatedly hounded. I have written to the GMC in London three times in the past eighteen months on this same issue. Now again in Manchester! Is this reasonable behaviour on the part of the GMC?

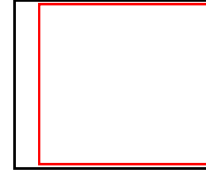
The GMC has a duty not to allow people's lives to be ruined as my wife's was for so many years.

If it were your wife or your daughter you would act on this right now!

Yours sincerely



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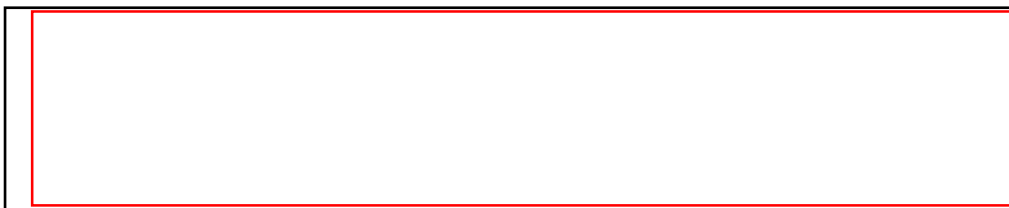
19<sup>th</sup> February 2007

Patricia Collins, Investigating Office  
Fitness to Practice Directorate  
General Medical Council  
5<sup>th</sup> floor, St James Buildings  
79 Oxford Street  
Manchester  
M1 6FQ

Dear Patricia Collins,

**Dr. Gordon R.B. Skinner MD (Hons) DSC, FRCPath, FRCOG.**

I am astounded that [redacted]  
[redacted] there now is a  
similar investigation in progress regarding Dr. Gordon R.B. Skinner MD (Hons) DSC.  
FRCPath, FRCOG. The Doctor who brilliantly maintains my health, and keeps me safely  
out of the hands of the General Practitioners who undoubtedly cannot match his  
brilliance in correctly diagnosing "Thyroid Disease" and who would certainly continue  
to feed me false information about my health, continue to prescribe me toxic drugs for a  
condition that I do not have, and watch me spiral downwards once more into a suicidal  
state of being, insisting the correct diagnoses was made regardless of my protestations.



It took only [redacted] months for me to recover fully and now, [redacted] years later, after the  
correct diagnoses in the year [redacted] the only medication I take is "Armour Thyroid" and

[redacted]  
[redacted]

It may interest you to know that in my [redacted]<sup>th</sup> year [redacted], I have still  
got my life back, [redacted] in

[redacted] I am comparatively normal.



It is quite clear that Dr. Gordon Skinner is a most brilliantly skilled and competent Doctor who has successfully rescued many, many a sick patient from chronic ill health, or mis-diagnosis with God knows what side effects -I can only speak of my self here - and it would be an outrage for his skill and expertise to be dismissed out of hand when people like me will have no place to turn as other Doctors would not be so brave as to stand alone against the G.M.C. for fear of being "Witch Hunted." as the satisfaction of the huge Pharmaceutical Companies is laid in place.

Surely, the Doctors who should be under investigation are the very Medical Practitioners that have a more than unhealthy obsession with there own diagnosis regardless of what suffering rapidly accelerates under there very noses, and who are opposed to the correct approach to Thyroid and Adrenal disorders.

It is their professional conduct that should be investigated, unlike Dr. Skinner, they ignore the Hippocratic Oath. I would also like to point out that a denial of a patients successful treatment is a denial of their human rights, and the G.M.C. should carry out their duty to that effect.

I wonder if you are aware of how many people these two brilliant Doctors help to bring back to a normal state of well being, and my own G.P. quite successfully ( for [redacted] long and painful years) manage to poison, and completely debilitate my body, after

[redacted]

Yours faithfully,

[redacted]

Copies to:- [redacted] Dr Mark Dudley, Ralph Shipway

**To whom it may concern**

**Interim Orders Panel, 26 February 2007, Manchester**

It is hard to imagine whether those on the Panel today have been handed copies of previous testimonials supporting Dr Gordon Skinner for IOPs on 29 June 05, 2 December 2005, my letter of 11 April 2006, 15 June 2006 and 7 August 2006. If you are not privy to such, I shall precis my position.

I first saw Dr Skinner in [redacted] having been diagnosed with thyrotoxicosis in [redacted]. I was diagnosed by my excellent GP who referred me to our local endocrinologist in [redacted]. He confirmed the diagnosis, [redacted]

[redacted]

Having been through the turmoil of the change of state, I remained on thyroxine and all was well until [redacted]

[redacted] I had already seen our local endocrinologist who did not seem remotely interested in the problems I was encountering in my hypothyroid state. [redacted]

[redacted]

When I saw Dr Skinner I realized that at last I was encountering a specialist who earned his reputation as an expert in the field of thyroid problems having been sought out by colleagues who shared with him their concern about patients with ME (some endocrinologists condescendingly describe such as 'somataform' illness in an article in 'Medscape' on 13 March 06). With the knowledge of Dr Skinner's success with ME, an illness that had hitherto led unfortunate patients down many expensive and fruitless medical routes, I increasingly felt confident that here was a doctor who took a full case history, including family history, examined me correctly and thoroughly (unlike the endocrinologist I had seen) and explained why thyroxine alone was not helping me. After a month on the additional drug tertroxin that Dr Skinner recommended and my GP



prescribed, I emerged from the sepiia state I had encountered since irradiation. From [ ]mcg to start with, I increased to [ ]mcg and came back to life in a remarkable way, [ ]

Endocrinologists may assume that patients automatically stick to a regime of medication. Dr Skinner encourages us to adjust our medication according to our needs.

For those of us who have been fortunate enough to be Dr Skinner's patients, we appreciate him for many reasons. Dr Skinner expects us to be intelligent in the way we face up to our thyroid situation. I have, of my own free will, cut back on my tertroxin dosage realizing that I no longer needed its original impact.

Given the choice of the path of anti-depressants and then possibly seeing psychiatrists or the path I took, I unhesitatingly am grateful that I saw Dr Skinner so that with his help I could not only overcome the problems that I encountered [ ]

Members of the IOP Panel may wonder why some of us write in support of Dr Skinner whenever we hear that your Panel is to meet. Part of the reason is that we wish to write tribute to the doctor who has so successfully brought us back to a quality of life denied to family members in the past who have similarly suffered but not achieved the quality of life that I, for one, has. The other part is to recognize that Dr Skinner is having

additional demands on his professional life due to the constrictions of the GMC that are both enormously time consuming and outrageous in the circumstances.

I am aghast that the GMC is spending a great deal of time and therefore money in this case. This is a doctor who should be teaching students the correct treatment of thyroid problems. It is quite extraordinary that he is being subjected to the scrutiny that he is when, from my experience, endocrinologists have much to learn about the treatment of the various forms of thyroid malfunction. I just wish my father, uncle, aunt and cousin had my good fortune and the quality of life I now have and had been similarly privileged to be under the care of Dr Skinner.

[redacted] we have gathered that Dr Skinner is accused of recommending too high a dose of tertroxin. I have done my homework, and discovered that a possible side effect is bone density damage. [redacted]

[redacted]

Perhaps we should all ask the question, is Dr Skinner actually harming patients? Why are so many of us bothering to turn up to support Dr Skinner at IOPs and write in his defence? Before our treatment, we would not have had the energy or inclination.

You may well be aware that a great many of us feel it is vital that the whole question of diagnosis and treatment of thyroid is openly discussed and researched so that future generations, and many living patients, are properly treated without the over-reliance of blood tests but instead proper clinical appraisal of signs, symptoms and medical history. If one is party to a genetic problem such as thyroid and has been successfully treated, one does everything possible to see such treatment is perpetuated. Please listen to Dr Skinner's patients.

[redacted]

20 February 2007

18/02/07

Ms Patricia Collins,  
Fitness to Practise Directorate,  
General Medical Council,  
5th Floor, St James's Buildings,  
79 Oxford Street,  
Manchester M1 6FQ

General Medical Council	
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Dear Ms Collins,

Re: Dr Gordon RB Skinner - Fitness to Practise

I'm informed that the GMC commissioned a report on Dr Skinner and that, when its findings were generally favourable toward Skinner, his FTP hearing was postponed and a second report commissioned. Even more disturbingly, I'm told that the GMC chose Dr [redacted] - a doctor known to take a view opposite to that of Dr Skinner, regarding the treatment of CFS patients. These actions appear grossly unfair and unjust - both for Dr Skinner and for us, his patients.

Dr [redacted]'s published articles on CFS demonstrate an opinion which is not based on clinical evidence and some of his statement I find naïve and insulting. [redacted] alleges that any improvements following treatment with thyroid hormones is due to a placebo effect! Skinner gets 80-85% of patients, some of whom have been ill for decades, returned to full health. I know of no clinical study in which a placebo has achieved any beneficial effects on more than a small proportion of patients, nor do I know of any study in which a placebo restored any patients to permanent full health. [redacted] claims that because CFS patients perceive thyroid hormones as being a 'real' medicine, this allows us to 'give ourselves permission to be well again'. In making this claim, he ignores clinical evidence; When doctors give CFS patients other 'real' medicines (eg. anti-depressants) none have much, if any, beneficial effect on our condition. Contrary to [redacted]'s 'permission' theory are the facts: by the time many of us staggered into Dr Skinner's surgery for our first consultation, we had so little faith left in doctors, that we didn't even believe him when he told us that he could get us well. We'd heard it all before from other doctors and healers. Even when Skinner's patients start to get better, he often has difficulty getting them to acknowledge the obvious progress they are making. This is because, in the early stages of treatment, we do not dare to even begin to believe that thyroid hormones really are working for us. So you see, CFS patients are not gullible patients who can be cured by a placebo of any kind - we are far too cynical about doctors for that to work, regardless of how 'real' the medication is!

I do not understand why the GMC seems not to be *investigating* Dr Skinner, but actively seeking a means to stop him practising. But *why*? *Why* would the GMC want to try to stop one of the few doctors who is restoring so many CFS patients to full health?

Question: Has the fact that Dr Skinner bases his prescribed dosage levels more on our symptoms than on just TSH and T4 test results, had a beneficial effect on most of us?

A: Yes.

Q: Have those of Skinner's patients, who are on what (some) endocrinologists would claim are *dangerously* high levels of thyroid hormone replacement, *actually got* erratic or rapid heartbeats? Do we have *any* of the other symptoms which would certainly occur were we to be taking more thyroid medication than we *needed*?

A: No and no.

Q: So how is Dr Skinner 'endangering patients', if the only thing he is doing is getting us well?

A: ??????????

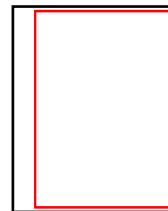
One is tempted to believe that certain endocrinologists are out to 'get' Skinner, because they resent the fact that he gets well patients whom they have failed. So they fabricate complaints about him. And instead of looking at the *evidence* and *why* Skinner is succeeding, they cling to dogma - that TSH levels are *always* a reliable indicator of a hypothyroid condition. To do this is to ignore findings about: resistance to thyroid hormone at cellular level; evidence of poor T4 - T3 conversion and of "reverse" T3; and of all of the other factors which can cause hypothyroid symptoms in people who have what are usually adequate "normal" levels of TSH and T4 in their blood. How *can* these endocrinologists discount such factors? Or the fact that fellow endocrinologists in the US have (finally!) accepted the need for doctors to lower what is regarded as a 'normal TSH level?

I am just one of *thousands* of CFS patients Dr Skinner (and others like him) have returned to health. What better evidence is needed, that a treatment works, than if it gets patients well? Yet we, and the evidence we keep sending the GMC, just seems to be ignored, regarded as mere anecdotal statements, rather than evidence - despite the fact that doctors can often only know if their treatment is working by what their patients *tell* them! The GMC appears *determined* to get rid of Dr Skinner. For us, his patients, this prospect is terrifying - *for what happens* to us then? What doctor would dare, for example, then give me the high levels of thyroid hormones I need, if that would expose him to the risk of being struck off? Do the GMC really *want* me and all the others to go back to suffering CFS hell?

I appreciate that you cannot discuss the specific 'complaints' made against Dr Skinner. But, hopefully, you might be able to assure me: that the GMC is *not* involved in a witch-hunt against Dr Skinner; that your investigation is impartial; that you are taking fully into account our testimony; that you will explain to me *why* the first report on Dr Skinner was rejected and his Fitness to Practise hearing postponed; and (finally!) why the GMC chose a specialist with known partiality against Dr Skinner to compile another report on him.

Yours sincerely,





Dear Ms Collins,

I see that my thyroid doctor ,Dr Skinner ,has been asked yet again to an IOP hearing, ,this time at the GMC in Manchester.

The fact that Dr Skinner is being pursued in this way is extremely distressing to his thousands of grateful patients . Without his skill and care, many of us would no longer be here, or our quality of life would be grossly impaired.

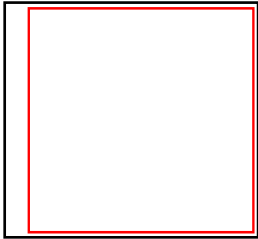
Please give Dr Skinner the respect he deserves and leave him to do his valuable work in peace.

Yours faithfully,

[Redacted signature]

[Redacted name]

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21. 02. 07

Dear Ms Collins,

Although one is aware that the GMC is under pressure to investigate complaints against doctors, especially in the light of the Shipman case, it does seem absurd that enquiries and restrictions are still ongoing in the case of Dr Skinner. A vast amount of time and money have been wasted and there is still no finding of fact against him.

A large number of patients and former patients have given up their time to be present at his hearings and a large number of testimonials have been sent to the GMC supporting him. Dr Skinner is an exceptionally caring, hardworking and experienced doctor whose work is being restricted by your organisation; and other doctors with lesser courage are under pressure not to prescribe adequately because they know of his treatment.

I trust that on Monday at least he will get a fairer hearing than he has in the past and that there will be some awareness of the massive support and gratitude from his patients.

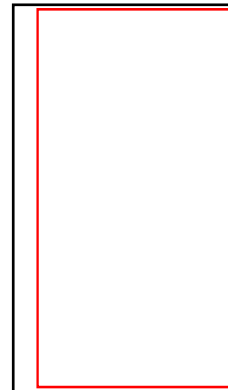
Yours sincerely,



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**Private & Confidential**

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22 February 2007

Ms C Floyd,  
Investigation Officer,  
Fitness to Practise Directorate,  
General Medical Council,  
5<sup>th</sup> Floor,  
St James's Buildings,  
79 Oxford Street,  
Manchester,  
M1 6FQ

Dear Ms Floyd,

**Re: Our Support for Dr G R B Skinner**

We have enclosed a copy of our recent correspondence to Ms Patricia Collins (Investigation Officer) and all members of the Interim Orders Panel and all members of the Fitness to Practise Panel at the General Medical Council – Manchester.

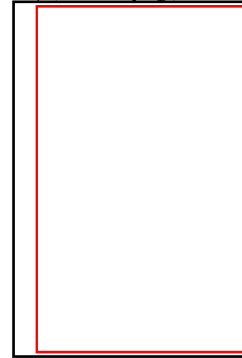
As already stated, we would be most grateful if you could confirm that you have personally received and read the file that we sent to you in December 2006 in support of Dr Skinner. We also require urgent confirmation that the aforementioned file in support of Dr Skinner will be given full consideration at any forthcoming Interim Orders Panel and the Fitness to Practise Panel. Thank you for your help in this matter. We look forward to your reply as a matter of urgency.

Yours sincerely,





Private & Confidential



Ms Patricia Collins (Investigation Officer)  
& all Members of the Fitness to Practise (FTP) Panel  
& all Members of the Interim Orders Panel (IOP),  
Fitness to Practise Directorate, General Medical Council (GMC),  
5<sup>th</sup> Floor, St James's Buildings,  
79 Oxford Street,  
Manchester, M1 6FQ

22 February 2007

Dear Ms Collins,

**Re: Our testimonial in support of Dr G R B Skinner**

In the foreword of the White Paper entitled 'Trust, Assurance and Safety – the Regulation of Health Professionals in the 21<sup>st</sup> Century' the Secretary of State for Health, the Rt. Hon. Patricia Hewitt MP states that 'professional regulation needs to sustain the confidence of both the public and the professions through demonstrable impartiality'.

In the GMC statement issued in response to the Government White Paper, Sir Graeme Catto says 'In November 2006, we argued for an independent and accountable system of medical regulation that would:.... Be objective, fair, accessible and transparent so as to command the confidence and support of those receiving and providing healthcare'.

On the GMC's own website, it already states that the Law (under the Medical Act) gives the GMC the function of 'dealing firmly and fairly with doctors whose fitness to practise is in doubt'.

Our interpretation of 'dealing fairly' would be that the GMC would take positive evidence and supportive testimonials into account in their proceedings.

We have provided a detailed file of information, which shows a consensus of opinion between Dr Skinner and the NHS endocrinologists Professor [redacted] and Professor [redacted] on the way that we should be treated. Our detailed information and supportive testimonials demonstrate that Dr Skinner is an excellent doctor.

However, although we have had confirmation that our correspondence has arrived with the employees of the GMC to whom it has been sent, we have not had individual confirmation (even though we requested this) that you and the aforementioned employees have read our file.

Instead you stated in your latest letter that the GMC is responsible for 'investigating and presenting the case against Dr Skinner at a Fitness to Practise Panel'. Surely the GMC should also consider positive evidence in favour of Dr Skinner and the numerous patient testimonials that have been sent in support of Dr Skinner. Your reply indicates that Dr Skinner is not being treated fairly by the GMC. We have attached a copy of your letter and have highlighted the sentences, which indicate that this excellent doctor is being treated unfairly by the GMC. Surely this contravenes article 6 of the Human Rights Act 1998.

Please could you and the other members of the Interim Orders Panel and the Fitness to Practise Panel start to take your position seriously and start listening to the many voices of the patients in support of Dr Skinner, remembering that if the GMC prevents Dr Skinner from providing the treatment that his patients need, you and others at the GMC would be responsible for the disability and risk of death that his patients would face. One wonders whether the GMC/ individuals concerned would be legally liable in such a situation. Surely such a circumstance could be interpreted as a contravention of article 2 and article 3 of the Human Rights Act 1998.

Please could you send us the names and contact details of the members of any forthcoming IOP and FTP Panel involving Dr Skinner. In addition, please could you send us an application form for requesting information via the Freedom of Information Act 2000.

Please could you also let us know what the procedure would be for initiating a formal complaint via the Charities Commission (since the GMC is a registered charity) and/or the Rt. Hon. Patricia Hewitt MP since the Government will be debating the aforementioned White Paper and will be discussing the future of the GMC.

Yours sincerely,

c.c. Dr G R B Skinner & Afshan Ahmad  
c.c. Mr R Shipway, RadcliffesLeBrasseur  
c.c. Dr M Dudley, Medical Protection Society  
c.c. Professor G Catto, President of the GMC  
c.c. Ms C Henesy, Assistant Registrar at the GMC, London  
c.c. Ms A Thompson, Adjudication Manager at the GMC, London  
c.c. Mr Andrew Wood, Assistant Registrar at the GMC, London  
c.c. Mr A Elliott, of the Interim Orders Panel at the GMC, London  
c.c. Ms A Dewhurst, Performance Assessment Officer at the GMC, London  
c.c. Mr P Swain, Head of Case Presentation at the GMC, London  
c.c. Ms R Goldsach of the Adjudication Section at the GMC, London  
c.c. Ms C Floyd, Investigation Officer at the GMC, Manchester  
c.c. Ms J Oliver, Claimant Solicitor at the GMC, London  
c.c. Ms T Sawtell of GMC Legal at the GMC, London  
c.c. Mr J Hiscock, Legal Assistant at the GMC, London

**Recorded Delivery**

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(For envelopes only - not for parcels, etc.)



Ms P. Collins  
Investigation Officer  
Fitness to Practice Directorate  
General Medical Council  
St James Buildings  
79 Oxford St  
Manchester  
M1 6FQ

**General Medical Council**

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RE: DR GORDON SKINNER

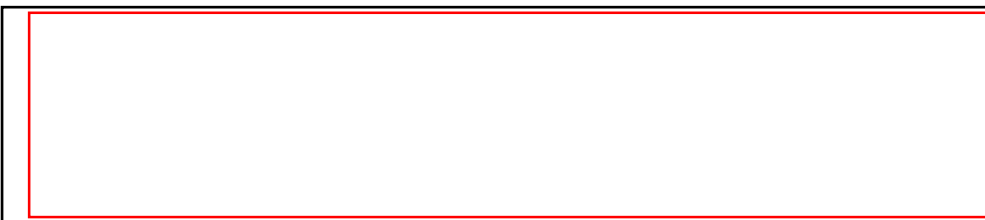
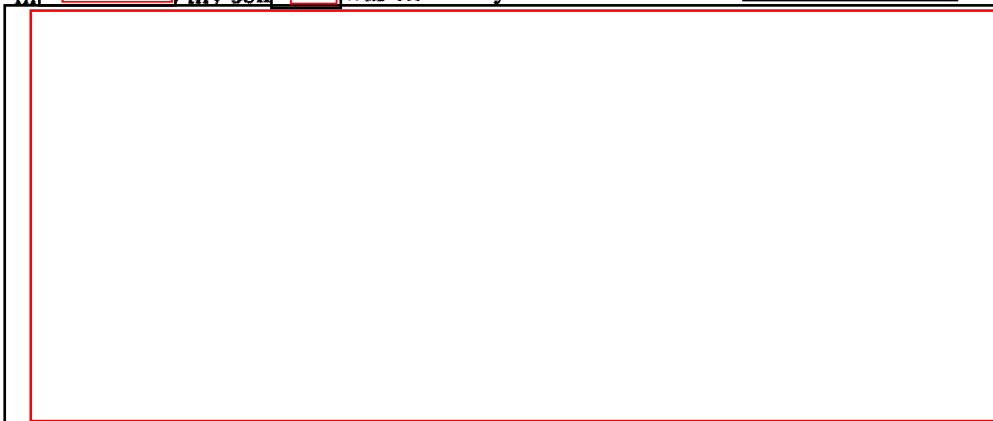
21 February 2007

Dear Ms Collins

I understand that Dr Gordon Skinner is to attend a hearing on Monday 26 February.

I am compelled to write to explain the huge debt of gratitude I and my family owe Dr Skinner.

In [redacted] my son [redacted] was extremely ill. He was suffering [redacted]



My own research led me to Dr Skinner in [redacted]. At this point we had effectively been abandoned by the medical profession who had offered nothing but pain killers, antibiotics, sleeping tablets and anti-depressants, CBT and graded exercise which of course he could not do. To be honest it seemed nothing short of child abuse that a seriously ill child could be treated in this way. He was himself fully aware that Doctors simply did not believe what he was saying. [redacted]

[redacted]. I remember that I had to persuade [redacted] to make the journey to see Dr Skinner as he did not believe this doctor would be any better than all the others. He is eternally grateful that he agreed to go.

Dr Skinner recognised that [redacted] did in fact have an under-active thyroid. There is a strong family history on both sides. There can be no doubting Dr Skinner's diagnosis. In his care being given the appropriate thyroid treatment [redacted] is now fit and well. [redacted]

[redacted]

[redacted] I dare not even imagine what [redacted] would be doing now if it were not for Dr Skinner. He tells me now that he could see no point in carrying on as he was with no hope of getting better and life passing him by.

There are very many sufferers of undiagnosed and therefore untreated hypothyroidism like [redacted]. It is essential for these people, not only middle aged women but often young people like my son, that Dr Skinner is recognised for the brilliant work he is doing.

Yours sincerely

[redacted]

**Adjudication Section**

**26 FEB 2007**



Mr. Adam Elliott,  
Interim Orders Panel,  
Regents Park,  
350 Euston Rd.,  
London,  
NW1 3JN.

21<sup>st</sup> February 2007

**Subject: G.R.B. Skinner MD (Hons), DSc, FRCPath, FRCOG,**



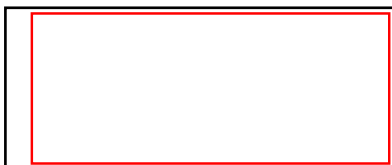
Dear Mr. Elliott,

Enclosed please find a copy of a letter addressed to Ms. Patricia Collins of the Fitness to Practise Directorate.

I addressed a letter with a very similar text to you in June 2005 when Dr. Skinner was obliged to appear before the Interim Orders Panel. As a patient of Dr. Skinner, I would herewith like to state how deeply angered I am that the GMC is continuing to allow itself to be used as a tool in what is very clearly a personal vendetta being orchestrated against Dr. Skinner.

It is time for the GMC to cease letting itself be manipulated in this underhand way and to put an end to the outrageous, Kafkaesque harassment to which Dr. Skinner has now been subjected for several years.

Yours sincerely,



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[Redacted]

Ms. Patricia Collins,  
Investigation Officer,  
Fitness to Practise Directorate,  
General Medical Council,  
5<sup>th</sup> Floor,  
St. James's Buildings,  
79 Oxford Street,  
Manchester,  
M1 6FQ.

21<sup>st</sup> February 2007

**Subject:** **G.R.B. Skinner MD (Hons), DSc, FRCPath, FRCOG,**

Dear Ms. Collins,

I understand that Dr. G.R.B. Skinner is to have a "Fitness to Practise Hearing" on 26<sup>th</sup> February 2007.


I strongly believe that Dr. Skinner must be permitted to continue treating thyroid patients. This belief is based on my own thyroid history, which I would ask you and your fellow Hearing panellists to read and consider most carefully:

- [Redacted]. In my last year there I was diagnosed as hypothyroid and told that I would require lifelong treatment.

- In [Redacted] and sought to have my thyroid treatment continued by an NHS GP. This GP refused to continue prescribing thyroxine for me without an NHS blood test. This NHS test showed my thyroid values to be "normal", and further treatment was therefore refused. [Redacted]

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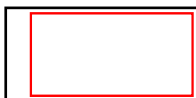
• By contrast, Dr. Skinner's exemplary treatment of my thyroid condition since  has restored me to good health.

There is an old Jewish saying that "a man who saves one life saves the world". Dr. Skinner has saved not only my life but, I know, that of many other thyroid patients who were also failed by the NHS.

If the Fitness to Practise Directorate is genuinely interested in patient care, it should

- recommend that Dr. Skinner be allowed to continue treating thyroid patients,
- call a halt to the increasingly Kafkaesque harassment of this exemplary doctor, and
- instigate an urgent investigation into the inept and callous state of thyroid care in the NHS.

Yours sincerely,







7<sup>th</sup> March 2007

Ms P Collins  
Investigation Officer  
General Medical Council  
5<sup>th</sup> floor  
St James's Buildings  
79 Oxford Street  
Manchester M1 6FQ

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Dear Ms Collins

**Dr Gordon RB Skinner**

I wanted to write to you to let you know of my overwhelmingly positive experience of being treated by Dr Gordon RB Skinner.

I have suffered from [REDACTED]

[REDACTED]  
[REDACTED]. On the basis of these assorted symptoms, I was diagnosed by my GP as having [REDACTED]. Although very sympathetic, he made it clear that there was nothing he could offer me in terms of treatment. I pushed to have the full range of blood tests, including T3 and T4 after which he simply reiterated his diagnosis, and said that I was in a better position than he to keep up to date with any new developments and treatments.

Given this bleak outlook, when a colleague whose friend had been restored to full health via the use of thyroid replacement recommended Dr Skinner, I swiftly followed up. After being referred by my GP in [REDACTED], and after a rigorous discussion of my symptoms, Dr Skinner put me on a course of thyroxine. Although not yet completely better, I have noted a huge improvement in my symptoms which I can only attribute to the treatment; [REDACTED]

[REDACTED]. It is though someone has switched the lights back on for me, and I now have regular glimpses

of a life without fatigue and pain with the expectation of a full recovery within the next few months. During the whole course of my illness, Dr Skinner is the only one who has properly listened to my symptoms and made a real effort to get to the root of my illness. I attribute to the huge improvement in my physical health entirely to his brilliance and the use of thyroxine.

I understand that Dr Skinner is due to go before the GMC in the summer of 2007. I wanted to write to you to let you know of the improvement of my symptoms and to request that you do not take any negative action against Dr Skinner given that he has assisted me and hundreds of others in returning to health.

Yours faithfully

A red rectangular box, likely intended for a signature or stamp.

Cc Prof G Catto, GMC  
Mr A Elliott, GMC  
Ms T Sawtell, GMC



00075351

Ms. Patricia Collins,  
Investigation Officer  
Fitness to Practise Directorate  
General Medical Council  
5<sup>th</sup> Floor  
St. James's Buildings  
79 Oxford Street  
Manchester  
M1 6FQ

28<sup>th</sup> March, 2007

TESTIMONIAL:

Dr. Gordon R.B. Skinner, MD.(Hons), DSc., FRCOG, FRCPath

Fitness to Practise Hearing 2<sup>nd</sup> July, 2007

Dear Ms. Collins,

I first saw Dr. Gordon Skinner around ☐ years ago and I found him extremely thorough and professional during my consultation.

He gave me a very thorough health check, including blood pressure, pulse, temperature, checking skin, etc, and also asked many questions. I believe the consultation lasted around an hour.

Dr. Skinner did not prescribe thyroid medication for me, but referred me to a Cardiologist. I believe this is contrary to your claims that he prescribes thyroid medications for every patient that he sees.

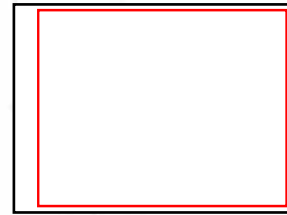
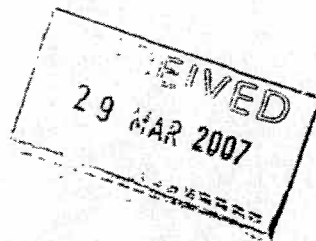
I would have no hesitation whatsoever in suggesting that patients ask to be referred to Dr. Skinner by their GP's.

I had total confidence in him and therefore support him and believe he should not be before the GMC for the charges against him.

Yours Sincerely,



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Dear Mr. Shipway,

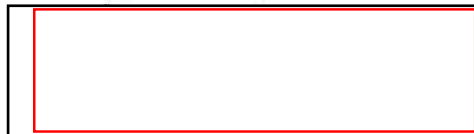
As one of Dr. Skinner's many grateful thyroid patients, I wish to add my name to the list of his supporters. How can someone with Dr. Skinner's years of experience, who listens to his patients, treats them with compassion, and diagnoses by signs and symptoms possibly be a danger to the general public?

I, like so many of Dr. Skinner's patients had been let down by the NHS, because a 'normal' TSH blood test resulted in my GP refusing to treat my hypothyroidism, even though he admitted that I had many of the signs and symptoms.

At my request, and with careful monitoring by Dr. Skinner, I am on thyroid hormone, and after years of ill health, I am now on the road to recovery after a few months.

With hypothyroidism on the increase we need experts like Dr. Skinner who do not diagnose patients with a blood test result decided by a laboratory technician who has never seen the patient.

Yours faithfully,



Ms Patricia Collins  
Investigation Officer  
Fitness to Practise Directorate  
General Medical Council  
5<sup>th</sup> Floor, St. James Buildings  
79 Oxford Street  
Manchester M1 6FQ

Testimonial in support of Doctor Gordon Skinner MD (Hons), DSc, FRCPath, FRCOG

Dear Ms Collins,

My wife and daughters will have already written to you and other members of the GMC in respect of the above doctor. Now that it seems that Dr. Skinner is to appear before the FTP panel in July, I felt that I must also write a letter of support in respect of this capable doctor and endorse everything my wife and daughters have had to say about him to date. In short, he is an excellent and caring as well as a most competent physician. He has been totally responsible for returning my daughters to full health from a state of extreme and debilitating illness, quite frankly he has given them their lives back.

I also wish to say as a [redacted], I have been appalled at the way that the IOP hearings have been conducted. I have read the transcripts and discussed these with my wife and have been most concerned by the disrespect afforded this man by the chairpersons concerned. On at least two occasions, he has not been allowed to speak in his own defence and the continual changes in panel members have been unfair to Dr. Skinner because even when allegations had been dropped, they have continued to be brought to the attention of each subsequent new panel and have caused confusion to all concerned.

As far as I am concerned, Dr Skinner should not have been seen by the IOP in the first place as in the main, the allegations are from other members of the medical profession who have never spoken to or examined the patients concerned and so are not in a position to pass any sort of comment either of diagnosis or treatment. I cannot believe that a doctor has been brought before the GMC for making his patients well. This case should be dropped at once and Dr Skinner and his patients permitted to get on with their lives. This case has caused my family and all concerned, prolonged and unnecessary worry for two years and this should stop now. Would you please ensure that this letter is added to the case in support of Dr. Skinner.

Yours sincerely

[redacted]  
[redacted]

Cc

[redacted] (Dr Skinner)  
Dr Mark Dudley MPS  
Mr Ralph Shipway (RadcliffesLeBrasseur)

General Medical Council	
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or scan	11 APR 2007
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Ms. Patricia Collins,  
Investigation Officer  
Fitness to Practise Directorate  
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M1 6FQ

General Medical Council	
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3<sup>rd</sup> April, 2007

Dear Ms. Collins,

Dr. Gordon R.B. Skinner, MD.(Hons), DSc., FRCOG, FRCPath

I know that you cannot make any comments on the case in hand and I would not expect you to do so, but as my future health and wellbeing, and that of my two daughters' is dependent on the outcome of the GMC's case against Dr. Skinner, I felt I had to write to you on a few points.

I feel incredibly sad and at the same time furious that Dr. Skinner has had to put his unblemished career on the line to help me, my two daughters and others like us.

Scientists have to have doubts otherwise there would be no new treatments, operations etc and if one of those scientists were wrong, would that mean it had to stay wrong forever because of actions taken by the GMC?

I must add, we have written testimonials for each IOP hearing and the three of us will do so for the Fitness to Practise hearing starting 2<sup>nd</sup> July, 2007.

The patients must always come first, something in this case, your organization chooses to ignore. I seriously hope you ask the views of Dr. Skinner's patients', as you may be surprised at the reactions of these people. The GMC appear to be basing this case on the complaints and views of a few doctors and not on the health and welfare of the patients. We as a family would be absolutely furious if our GP complained to the GMC about Dr. Skinner; after all, it was GP's like these who have totally failed us over the years.

My first point is, why do the GMC have different panel members at each hearing as they cannot possible read all the 'evidence' in such a short space of time and therefore it must always go against Dr. Skinner. Thank goodness trials are not run in this deplorable way.

My second point is, if Dr. Skinner is SUCH a danger to the public, why have all the IOP hearings only ever put paperwork conditions on him. If he is that much of a problem with his prescribing, surely he

should have been suspended. Maybe it was because the GMC were incredibly worried about the hundreds of patients who support Dr. Skinner. It would be a travesty of justice if Dr. Skinner were to be struck off the medical register and indeed I for one will be seeking legal advice if this were to be the outcome.

My third point is the level of medication Dr. Skinner prescribes and his so called non-reliance on blood tests. There is plenty of medical evidence that Dr. Skinner is correct in his assumption, also that of the wellbeing of his patients during treatment, that (1) We sometimes need higher doses than has been prescribed over the last few decades. In the days before blood tests, 200 - 400mcg of Thyroxine was the normal level of medication and (2) before that Armour Thyroid was used which contains both T4 and T3! Although 2 grains was generally the norm, 4 grains or higher was not an unusual amount.

The term biochemically thyrotoxic, is that, just a term as I am not a blood test (nor are my daughters.) I am a person who has had [ ] years of ill-health and my chiropractor said for many years I had an underactive thyroid. I had numerous blood tests over the years with doctors saying I looked hypothyroid and then telling me 'no I wasn't because the blood tests said I wasn't'. It had to be something else ([ ]) totally ignored, apart from all the other symptoms.). In my [ ] I read an article about how the blood tests don't always tell the truth and are often misinterpreted. I saw a private GP in [ ] who said she knew what was wrong with me the moment she saw me. I started on Thyroxine and symptoms that I had had for decades started to disappear.

Treatments at Great Ormond Street hospital are often cutting edge, untried and experimental. Do these doctors get brought before the GMC because other doctors complain about them making their patients well?

This brings me to point four. It appears to me that there has only ever been one complaint from a patient and the rest from doctors and yet as Mr. Jenkins says there have been hundreds of testimonials from patients. Does this not seem odd? In the transcript of the hearing dated 26<sup>th</sup> February, 2007, Mr. Jenkins reminded the GMC (transcript page 5 G) that there had only ever been one complaint from a patient (although reading the first transcript of 29<sup>th</sup> June, 2005 I believe Mr. Jenkins (at 26<sup>th</sup> Feb hearing) has put the wrong initial HR, instead of KW). (In the first transcript the GMC barrister refers to KW page 71 of the bundle.) I have printed out my copy of the transcript in smaller type, so I am afraid I cannot give you the page number. Looking at the 05 transcript it appears that it was HR's GP who complained to the GMC. Has anyone from the GMC asked how HR feels about this complaint?

Regardless of this, in the transcript of the determination of 26<sup>th</sup> February, 2007, the Chairman states "The panel has noted the circumstances surrounding the complaints made against you by PATIENTS and other professional colleagues". Why are you pursuing this as patients - plural?

With regard to Dr. Cundy and HR blood tests - 3-4 pages in first transcript, GMC barrister points to blood test dated 10<sup>th</sup> March, result T4 = 11.6 in a range of 9.0 - 20 and TSH 2.2 in a range of 0.4 - 5.5 and states the results are in the normal range, but as more questions are being asked about ranges, the T4 result is very low in the range (much lower than government guidelines as they should be in the upper quartile) and the TSH at 2.2 is suggesting subclinical hypothyroidism and if you look at T. Bjoro's survey, then the TSH should be a lot lower! The later test of August '04 is TSH below 0.1 (often normal in patients on thyroid replacement especially when on T3 as this is the active hormone). Dr. A. Toft treats over the FT4 range and below TSH range in certain patients who still have symptoms. Will the GMC be requesting Dr. Toft to attend and IOP hearing regarding his prescribing?

I find it surprising that Dr. Cundy 'request assistance' from a colleague at Mayday Healthcare NHS Trust. This colleague, a Dr. P (transcript) obviously never saw HR, so was diagnosing from afar. I would not wish that to happen to me or my family. Dr. P finally states that as a former secretary to the BTF it was trying to counteract private practioners who called themselves endocrinologists.

Not once did Dr. Skinner say to me or to my daughters that he was an endocrinologist, only that he was a virologist. We consulted him with our eyes wide open.

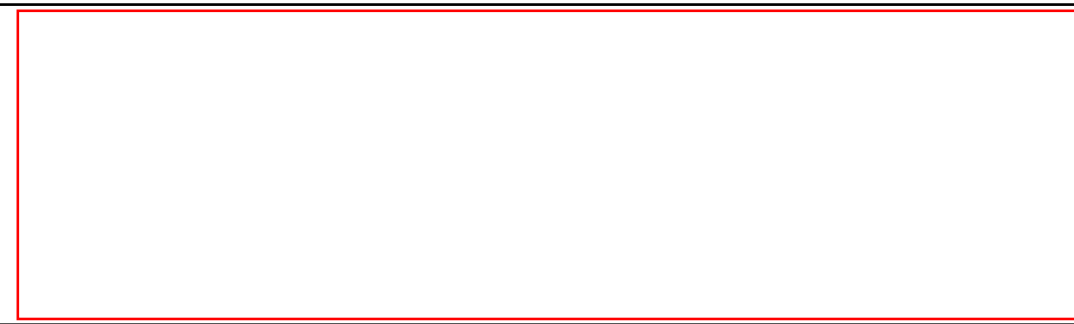
Interestingly, at the beginning of Mr. Brassington's case for the GMC (transcript, of 29<sup>th</sup> June, 05), it appears that a Dr. Cundy wrote to the GMC reporting Dr. Skinner on 23<sup>rd</sup> February, 04 and yet it appears that HR had only been fully registered with his surgery from 1<sup>st</sup> September, 2004.

If this were a court of law, the case I am sure would be thrown out. This is the most dreadful behaviour. Not only is a doctor's profession and reputation on the line, his patients may well have to return to illness because a few doctors don't believe in what he is doing. Would doctors like Dr. Christian Barnard have ever been able to perform the first heart transplant for fear of their reputation?

Dr. Skinner does his best for his patients and is not as stated in the Determination (page 11 F) 'In all the circumstances, the Panel considers that if your registration were to remain unrestricted you may pose a risk to patients and the confidence that the public are entitled to place in the medical profession and its practitioners would be undermined'. He has been this family's saviour and he doesn't pose a risk to me or to my daughters, unless that is, making us well is a risk.

I am extremely concerned at the doctors who are making these allegations against him as they don't seem to have any respect for their patients and how they feel.

Doctors didn't believe bacteria was the cause of stomach ulcers and the doctor who discovered this had to go to great lengths to get the medical profession to believe him and yet eventually he won the Nobel Prize for his work!



Need I say more apart from the fact those children had various illnesses all ignored by one of your main expert witness.

In the words of Donna Anthony, 'this man didn't even meet me and yet he labelled me a murderer'!

What if the experts are talking rubbish is how it was so bluntly written in one newspaper?!

Could the above apply in Dr. Skinner's case? I really believe there certainly has to be an urgent rethink regarding the testing and treatment of hypothyroidism.

Maybe, the patients are the experts and their views should be considered and respected. Others like me and my family had been totally failed by the medical profession until we found doctors like Dr. Skinner who had more of an understanding of hypothyroidism than any other GP or endocrinologist we had consulted. This is what is so hard to understand, why won't the GMC listen to patients. Patients like me, have gone to the public hearings to support Dr. Skinner and the GMC said they had never seen anything like it before. A doctor who was supposed to be such a danger to the public would not normally attract the support of so many.



In the transcript of the hearing of 7<sup>th</sup> August 06, the GMC barrister says 'the panel will see the opening sentence (of a letter), an expression of concern which the Panel will be familiar with'. These are obviously expressions of concerns from the doctors and not from the patients.

Prof Lazarus is quoted in the transcript and also frequently in the press stating that if the blood tests are normal then the patient does not have hypothyroidism. These doctors should see the suffering they heap on patients like me and my daughters. I do sometimes wonder what would be the outcome if

members of these doctors' families were showing all the symptoms of hypothyroidism but with normal blood test results, if they would inflict the same hell on them?!

If Dr. Skinner prescribes at dangerous levels, I find it quite interesting that in the September 06 issue of Nature Clinical Practice, Endocrinology and Metabolism, JV Hennessey, Associate Professor of Medicine at the Brown Medical School, Rhode Island Hospital, Providence, RI, USA, 'Levothyroxine Dosage and the Limitations of Current Bioequivalence Standards', that they used trial doses of 600mcg Thyroxine on healthy (euthyroid) volunteers. What would the GMC say to this?

Research done in Norway in 2000 by T Bjoro proved beyond doubt that the bell curve for TSH as we know it, is not a bell curve at all! See attached.

I have now been a lay researcher in hypothyroidism for eight years and so if I can find the evidence to **support the views of Dr. Skinner, then your panels certainly can, if of course they want to!**

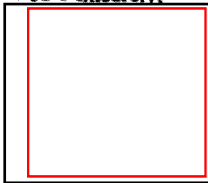
Interestingly, in the attachment dated 22<sup>nd</sup> February, 2004, 'We're listening', it is basically about treatment (or not) in the latter stages of life, but I assume this applies to everyone at any stage in life, Ruth Evans, the then Chairman of your Standards Committee, said, quote 'The General Medical Council's guidance makes clear that patients must be fully involved in decisions about all aspects of their care. It is for patients – not doctors – to decide how much weight to give to the benefits, burdens and risks of a treatment and to decide its overall acceptability. Doctors must also seek patients' views as to how they would like to be treated if their condition worsens'.

In the GMC's own words, 'Our legal authority is the Medical Act, which gives us powers to protect, promote and maintain the health and safety of the public.

**Dr. Skinner does protect, promote and maintain the health and safety of my family and according to Mr. Jenkins hundreds of other patients who wrote testimonials, but the GMC are failing us all by their proceedings.**

**By their own actions, the GMC is putting people's health in jeopardy, three of those in this family!**

Yours sincerely,



Copied to: Mr. Ralph Shipway, RadcliffesLeBrasseur, MPS & Dr. G. Skinner

Attached:

BMJ editorials 29<sup>th</sup> September

Immune Support

Journal of Clinical Endocrinology and Metabolism: Narrow individual variations in Serum T4 and T3

Ditto: The evidence for a narrower thyrotrophin reference range is compelling

Fibromyalgia and Fatigue Centers: Thyroid Resistance

BMJ: Thyroid functions-time for a reassessment

Dr. John Lowe (a chiropractor). Evidence that it's the rarest exception when the ECG rhythm of a patient is of concern.

T.Bjoro et al

TELEGRAPH



BMJ 2001;323:705-706 ( 29 September )

## Editorials

### Wrong biochemistry results

*Interference in immunoassays is insidious and could adversely affect patient care*

The success of analytical methods in clinical chemistry has led to a sense of security in the value of laboratory results. This is largely justified, as evidenced by the quality of laboratory performance assessed by external assurance schemes. Nevertheless, it is not widely recognised among clinicians that some biochemical tests are more prone to interference from unusual serum constituents than others—and that quality assurance schemes can do little about this.

An important example of this is tests carried out by immunoassays based on the recognition of molecules by antibodies. The antibodies are largely derived from animal sources and are typically used for measuring hormones, tumour markers, cardiac troponins, and therapeutic drugs and for viral serology. The design of assays has evolved enormously since the discovery of immunoassay by Berson et al in 1956,<sup>1</sup> and it is now a major analytical tool in clinical laboratories worldwide, allowing relatively minute (picomole ( $10^{-12}$ )) amounts of analytes to be measured with unrivalled precision.

Interference in immunoassays by antibodies is a recognised phenomenon. For example, endogenous antithyroglobulin antibodies invalidate thyroglobulin measurements, and exogenously administered antibodies used to treat digoxin toxicity prevent measurement of plasma digoxin. However, there is more insidious interference due to the presence of unsuspected abnormal binding protein(s) in the patient.<sup>2,4</sup> These mainly include heterophilic antibodies such as rheumatoid factor, anti-animal antibodies (anti-mouse, anti-rabbit, anti-sheep, etc), and anti-idiotypic antibodies (antibodies elicited by an idiotope on another antibody molecule during the course of an immune response). In some cases these antibodies in patients' sera may interfere with the analytical reaction between the analyte being measured and the antibodies used in the immunoassay's cocktail. The exact effect of such interference will depend on the site where they interfere with the reaction, leading to falsely raised or lowered measurements. These interferences are specific to each patient, so only that patient's data will be affected, while quality assurance criteria for the assay will have been passed.

Examples of this type of interference that has had serious clinical consequences include human chorionic gonadotrophin assays.<sup>5</sup> As a result of wrongly interpreted results six young non-pregnant women were aggressively treated with chemotherapy and surgery for non-existent "occult" trophoblastic disease.<sup>6</sup> In our experience at a national reference steroid laboratory, samples with consistent and substantial increases in steroids using routine direct immunoassays have raised the possibility of disease but have subsequently been found to be normal after reassay using more robust techniques involving extraction procedures. In one case a raised oestradiol value led to a patient having a hysterectomy and bilateral oophorectomy, and only when no fall in oestradiol was seen postoperatively was the sample further analysed and the original result found to be wrong because of immunoassay interference. Similar problems are also noted in other steroid assays, such as testosterone in women.<sup>7</sup> False positive interference in troponin assays in patients with chest pain due to acute coronary syndrome has led to prolonged inpatient stays and invasive investigation.<sup>8,9</sup> False negative results are equally important in that they lead to underinvestigation.<sup>10</sup>

The presence of interfering antibodies is surprisingly common, affecting 30-40% of the population.<sup>4</sup> They probably arise from mundane activities such as keeping pets, ingesting animal antigens, vaccination, infection, or even blood transfusion. Most analytical assays currently in use can neutralise and block low concentrations of these interfering proteins ( $\mu\text{g}$  to  $\text{mg/l}$ ) with no or minimum impact on analytical accuracy. Larger amounts of interfering proteins, which may be as high as grams per litre, or proteins with high binding affinity can, however, overwhelm the analytical system, leading to assay interference and erroneous results. The number of these extreme cases is not known, though specific types of interference, such as heterophilic and anti-murine antibodies, in the order of 0.05% have been reported.<sup>4,11</sup> Our experience suggests that interfering antibodies of various types affect about 0.5% immunoassays (A Ismail, J Barth, unpublished data), though others have reported higher percentages.<sup>12</sup> Even the lowest prevalence quoted should be



seen in the context of the total number of immunoassays—many millions a year in British hospital laboratories alone. Thus many thousands of patients in the United Kingdom might be affected. Furthermore, this problem is likely to worsen owing to the wider use of biotechnologies such as monoclonal antibodies and T cells for diagnostic and therapeutic purposes.<sup>12</sup>

Since these interferences are relatively uncommon, clinicians need to be aware of them and alert to the mismatch of clinical and biochemical data. They should not discard clinical evidence in favour of a numerical value. Moreover, this form of interference is not specific to a single analyte and may affect other immunoassays performed on the same patient in a different clinical setting. Thus patients who have such interference detected should have this fact documented in their clinical records, so that the results of future immunoassays can be viewed with caution.

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gmc

## ImmuneSupport.com Treatment & Research Information

### Tip of the Day

ImmuneSupport.com

08-18-2006 According to Kent Holtorf, M.D., "CFS and FM patients will often have a number of thyroid abnormalities including a low free T3, a high reverse T3, and a low TSH. These abnormal ratios are not usually discovered using the standard laboratory interpretation of hypothyroidism.

When CFS and FM patients are treated with thyroid, they are almost always under-dosed because their pituitary dysfunction results in their TSH becoming quickly suppressed, which normally indicates too much thyroid. Because these patients have pituitary dysfunction, one must forget about the TSH and not treat based on this parameter.

These patients can also have a thyroid resistance syndrome. This has not been a well-accepted concept by general mainstream medicine and many refuse to believe it exists because the exact mechanism has not been elucidated, but this is a real phenomenon.

In fact, in a recent issue of *International Journal of Medical Research*, a major peer reviewed medical journal, a patient was described that required 10 times the normal dose of thyroid intravenously before her symptoms would resolve. This resistance usually improves as the patient gets better and they subsequently need less thyroid."

(Source: [www.immunesupport.com](http://www.immunesupport.com). Read complete article at <http://www.immunesupport.com/library/showarticle.cfm?id/4532>)

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28/03/2007

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## Endocrine Care

# Narrow Individual Variations in Serum T<sub>4</sub> and T<sub>3</sub> in Normal Subjects: A Clue to the Understanding of Subclinical Thyroid Disease

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## Abstract

High individuality causes laboratory reference ranges to be insensitive to changes in test results that are significant for the individual. We undertook a longitudinal study of variation in thyroid function tests in 16 healthy men with monthly sampling for 12 months using standard procedures. We measured serum T<sub>4</sub>, T<sub>3</sub>, free T<sub>4</sub> index, and TSH. All individuals had different variations of thyroid function tests ( $P < 0.001$  for all variables) around individual mean values (set points) ( $P < 0.001$  for all variables). The width of the individual 95% confidence intervals were approximately half that of the group for all variables. Accordingly, the index of individuality was low: T<sub>4</sub> = 0.58; T<sub>3</sub> = 0.54; free T<sub>4</sub> index = 0.59; TSH = 0.49. One test result described the individual set point with a precision of plus or minus 25% for T<sub>4</sub>, T<sub>3</sub>, free T<sub>4</sub> index, and plus or minus 50% for TSH. The differences required to be 95% confident of significant changes in repeated testing were (average, range): T<sub>4</sub> = 28, 11–62 nmol/liter; T<sub>3</sub> = 0.55, 0.3–0.9 nmol/liter; free T<sub>4</sub> index = 33, 15–61 nmol/liter; TSH = 0.75, 0.2–1.6 mU/liter. Our data indicate that each individual had a unique thyroid function. The individual reference ranges for test results were narrow, compared with group reference ranges used to develop laboratory reference ranges. Accordingly, a test result within laboratory reference limits is not necessarily normal for an individual. Because serum TSH responds with logarithmically amplified variation to minor changes in serum T<sub>4</sub> and T<sub>3</sub>, abnormal serum TSH may indicate that serum T<sub>4</sub> and T<sub>3</sub> are not normal for an individual. A condition with abnormal serum TSH but with serum T<sub>4</sub> and T<sub>3</sub> within

laboratory reference ranges is labeled subclinical thyroid disease. Our data indicate that the distinction between subclinical and overt thyroid disease (abnormal serum TSH and abnormal T<sub>4</sub> and/or T<sub>3</sub>) is somewhat arbitrary. For the same degree of thyroid function abnormality, the diagnosis depends to a considerable extent on the position of the patient's normal set point for T<sub>4</sub> and T<sub>3</sub> within the laboratory reference range.

**OVERT ABNORMALITIES IN thyroid function** are common endocrine disorders affecting 5–10% of individuals over a lifespan (1). Clinical symptoms and signs are often nonspecific, and the diagnosis and monitoring of therapy depends crucially on measurements of thyroid hormones and TSH in blood (2, 3).

Minor abnormalities in thyroid function with subclinical hypothyroidism or hyperthyroidism are even more common (1, 4, 5). Both subclinical hypothyroidism and hyperthyroidism are associated with an increase in risk of disease (4, 5, 6) as well as abnormalities in biochemical and physiologic measures that are often abnormal in patients with overt thyroid disease (6, 7, 8, 9). Still it is debated to what extent subclinical thyroid disease should be treated (6, 10, 11, 12).

Subclinical thyroid disease is defined by high- or low-serum TSH with T<sub>4</sub> and T<sub>3</sub> within laboratory reference ranges. An important issue is whether serum concentrations of T<sub>4</sub> and T<sub>3</sub> are normal for the individual in subclinical thyroid disease.

Population-based reference ranges for serum T<sub>4</sub> and T<sub>3</sub> are quite wide because of large differences in thyroid function tests among normal subjects. These differences are caused by analytical and biological variation (13). Biological variation consists of between-individual and within-individual variation, the latter being characterized by rhythmic aberrations of multiple frequencies ranging from 30 min to 365 d (14, 15).

In general, population-based reference ranges are of limited value for interpretation of measurements in the individual if variation within individuals is small, compared with variation between individuals (16, 17). This may cause test results that are abnormal for the individual to be unnoticed within the wide group reference range.

Previous studies of the biological variation in thyroid function tests were performed under standardized conditions over shorter periods of time. Hence, the clinical importance of variation in thyroid function tests remains to be clarified (18, 19, 20).

We estimated biological [*i.e.* within (intra-) and between (inter-) individual] variation in thyroid function tests over a 12-month period in a group of healthy men in a routine laboratory setting. This was used to assess the utility of population-based reference ranges for serum TSH, total T<sub>3</sub>,

laboratory reference ranges is labeled subclinical thyroid disease. Our data indicate that the distinction between subclinical and overt thyroid disease (abnormal serum TSH and abnormal T<sub>4</sub> and/or T<sub>3</sub>) is somewhat arbitrary. For the same degree of thyroid function abnormality, the diagnosis depends to a considerable extent on the position of the patient's normal set point for T<sub>4</sub> and T<sub>3</sub> within the laboratory reference range.

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Minor abnormalities in thyroid function with subclinical hypothyroidism or hyperthyroidism are even more common (1, 4, 5). Both subclinical hypothyroidism and hyperthyroidism are associated with an increase in risk of disease (4, 5, 6) as well as abnormalities in biochemical and physiologic measures that are often abnormal in patients with overt thyroid disease (6, 7, 8, 9). Still it is debated to what extent subclinical thyroid disease should be treated (6, 10, 11, 12).

Subclinical thyroid disease is defined by high- or low-serum TSH with T<sub>4</sub> and T<sub>3</sub> within laboratory reference ranges. An important issue is whether serum concentrations of T<sub>4</sub> and T<sub>3</sub> are normal for the individual in subclinical thyroid disease.

Population-based reference ranges for serum T<sub>4</sub> and T<sub>3</sub> are quite wide because of large differences in thyroid function tests among normal subjects. These differences are caused by analytical and biological variation (13). Biological variation consists of between-individual and within-individual variation, the latter being characterized by rhythmic aberrations of multiple frequencies ranging from 30 min to 365 d (14, 15).

In general, population-based reference ranges are of limited value for interpretation of measurements in the individual if variation within individuals is small, compared with variation between individuals (16, 17). This may cause test results that are abnormal for the individual to be unnoticed within the wide group reference range.

Previous studies of the biological variation in thyroid function tests were performed under standardized conditions over shorter periods of time. Hence, the clinical importance of variation in thyroid function tests remains to be clarified (18, 19, 20).

We estimated biological [*i.e.* within (intra-) and between (inter-) individual] variation in thyroid function tests over a 12-month period in a group of healthy men in a routine laboratory setting. This was used to assess the utility of population-based reference ranges for serum TSH, total T<sub>3</sub>,



total T<sub>4</sub> and free T<sub>4</sub> index (FTI). In addition, we calculated the number of tests needed to estimate the individual set point with certain levels of confidence and the difference required between two hormone values for significance in repeated testing of an individual.

## Subjects and Methods

### *Subjects and protocol*

Sixteen healthy Caucasian men participated. Median age was 38 yr, range 24–52 yr. Five were nonsmokers and 11 were smokers (5–25 cigarettes/d). They had an average body mass index of 25.4 kg/m<sup>2</sup>, range 21.3–30.9. Participant number 7 turned out to be subclinical hyperthyroid with permanently suppressed serum TSH and normal T<sub>4</sub>, T<sub>3</sub>, and FTI in serum. He was excluded from calculations. Of the remaining 15, none had clinical goiter or known previous or present thyroid disorders. None took any medication. They lived in Jutland, Denmark, where the iodine intake is moderately low (21), and their median urinary iodine excretion in spot urine samples, collected monthly for 12 months, was 50 µg/liter (22). We made no restrictions to their daily or yearly routines. Approval by the Regional Ethics Committee was obtained before the commencement of this study.

Blood samples were collected monthly for 12 months. Venepuncture was done between 0900 and 1200 h using standard procedures. Whole blood was allowed to clot and serum was separated and stored at -20 °C until analysis.

### *Assays*

Serum TSH was determined using immunochemiluminometric technology and a third-generation assay (LUMitest, Brahms, Berlin, Germany) with an intraassay coefficient of variation (CV) in our laboratory of 2.3% (TSH measured in duplicate in sera from 87 healthy subjects, level 1.5 mIU/liter). Serum total T<sub>3</sub> (CV 2.2% in duplicate measurements of 25 sera from healthy subjects, level 2.3 nmol/liter) and serum total T<sub>4</sub> (CV 2.4% in duplicate measurements of 108 sera at 109 nmol/liter) were determined by radioimmunoassays (Amerlex-M T<sub>3</sub> RIA kit and Amerlex-M T<sub>4</sub> RIA kit, Johnson & Johnson, Cardiff, UK). T<sub>3</sub> uptake for calculation of FTI (CV 4.0% in 91 normal samples) was measured using an assay from Farnos Diagnostica (Oulunsalo, Finland). Single determinations were performed, and all samples from a subject were analyzed in random order in the same assay to eliminate interassay variation. Analytical error was determined for intraassay variation as described. Also, no attempt was made to detect or exclude outliers. Procedures were designed to picture the standard procedures at our laboratory.

### *Statistical analysis*

Serum TSH, T<sub>3</sub>, T<sub>4</sub>, and FTI were normally distributed in all individuals (in the individual participants  $P = 0.3-1.0$  for TSH;  $0.2-1.0$  for T<sub>3</sub> and T<sub>4</sub>;  $0.6-1.0$  for FTI) and mild to moderately positively skewed in the group ( $P$  for normally distributed = 0.08, 0.24, 0.26, 0.02, respectively) as evaluated by the Shapiro-Wilk test and normality plots. As recommended, none of the variables were transformed (17). Samples were assumed independent and the results were compared using Bartlett's test for homogeneity of variance and Kruskal-Wallis test. A  $P$  value of less than 0.05 was considered significant.

The individuality of reference ranges was estimated from the individuality index (16, 17). This index of individuality was calculated from  $SD_{\text{analytical}} + \text{intraindividual}$  (17) where the average  $SD_{\text{analytical}} + \text{intraindividual}$  was computed as the square root of the mean of the individual variances and the  $SD_{\text{interindividual}}$  as the estimated variance between means (17).

The number of tests needed to determine the homeostatic set point for the individual was estimated from  $n = (Z \times CV_{\text{analytical}} + \text{intraindividual}/D)^2$  where  $n$  was the number of specimens obtained,  $Z$  was 1.96 (*i.e.* 95% confidence interval), and  $D$  was the percentage closeness to the homeostatic set point (17).

The difference required for significance in serial testing was calculated from  $1.96 \sqrt{n \times \sqrt{SD_{\text{analytical}}^2} + SD_{\text{intraindividual}}^2}$ .

Statistical analyses were performed using the statistical package for the social sciences version 10.0 and Corel QuattroPro8 (SPSS, Inc., Chicago, IL).

## Results

Figure 1E illustrates within-individual and between-individual differences in serum TSH, serum T<sub>3</sub>, serum T<sub>4</sub>, and serum FTI over 1 yr. For each variable large variations were seen between individuals and within individuals. Also, considerable differences in the variations within individuals were seen. This was independent of position of mean serum TSH, serum T<sub>3</sub>, serum T<sub>4</sub>, and serum FTI in the reference range. When testing for this, homogeneity of variance was not present between individuals ( $P < 0.001$  for all variables) indicating an unpredictable difference in variation among individuals. Also, the participants had different set points ( $P < 0.001$  for all variables). Hence, each subject's thyroid function was unique, as evaluated from hormone concentrations in serum.



**Figure 1.** Serum TSH, total T<sub>3</sub>, total T<sub>4</sub>, and FTI in 16 normal subjects taken monthly for 12 months. Each *dot* represents a monthly measurement and *horizontal bars* indicate individual parametric means. Participants are sorted by increasing mean. Laboratory reference ranges are: TSH, 0.3–5.0; T<sub>3</sub>, 1.2–2.7; T<sub>4</sub>, 60–140; and FTI, 70–140. Large differences were seen between individual set points, and unpredictable differences were seen in variations within individuals for all thyroid function tests.

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The relationship between serum T<sub>3</sub>, T<sub>4</sub>, and FTI levels and serum TSH was examined in each individual. The associations were marginally positive (mean Spearman's rho with 95% confidence intervals: TSH vs. T<sub>3</sub> 0.14 (-0.43 to 0.72), TSH vs. T<sub>4</sub> 0.13 (-0.56 to 0.81), and TSH vs. FTI 0.18 (-0.47 to 0.83)).

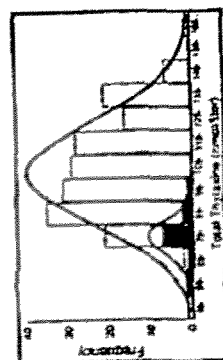
Participant number 7 turned out to be subclinically hyperthyroid with permanently suppressed TSH. He was excluded from calculations. However, the measured values were included in Table 1.

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Table 1. Hormone levels and individual interval of variation in 16 subjects (mean  $\pm$  2 SD). Means and individual 95% reference ranges for each of the 16 participants are listed in Table 1. Also, means and individual reference ranges for an average participant as well as means and group reference ranges for participants 1 through 6 and 8 through 16, and the standard laboratory

reference ranges are presented in Table 1. In addition, the individuality index is shown. The width of the individual participants reference ranges was about half of that of the group for all thyroid function tests, indicating that test results for an individual varied within 50% of the distribution for the group. Accordingly, the individuality index was below the critical limit of 0.6 (16, 23) for all thyroid function tests demonstrating that reference ranges based on the group are insensitive to significant changes in the individual.

Figure 2 illustrates the distribution of serum T<sub>4</sub> in one individual, compared with the distribution of measurements of T<sub>4</sub> in the group. In this individual serum T<sub>4</sub> may be substantially above the individual reference range and still lie well within the group reference range.



**Figure 2.** The distribution of 12 monthly measurements of total T<sub>4</sub> in 15 healthy men (□) and in one individual, number 11 (■). The distribution in one individual is about half the width of the distribution in the group.

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Table 2 shows the number of tests required to obtain a 95% confident estimate of the homeostatic set point for TSH, T<sub>3</sub>, T<sub>4</sub>, and FTI in an individual within plus or minus 5%, plus or minus 10%, and plus or minus 25% variation. Intraindividual variations caused a relatively high number of tests needed to describe the individual set points.

**View this table:** [Table 2. Number of tests required to describe the homeostatic set point in an individual](#)  
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Table 3 shows the difference required in serum TSH, T<sub>3</sub>, T<sub>4</sub>, and FTI to be 95% and 99% confident of real changes in repeated testing. These changes are significantly larger than the normal spontaneous variation observed in the individual. Because of differences in variations among individuals, it is difficult to make general recommendations for evaluation of significant changes in repeated testing.

**View this table:** Table 3. Difference required for significance at 5% and 1% level in repeated testing: variations larger than the spontaneous variations within the individual  
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## Discussion

Population-based reference ranges include both within- and between-individual variation. The value of reference ranges diminishes if between-individual variation exceeds within-individual variation. Harris (16, 23) explored this relationship computing the individuality index (16) and stated that population-based reference intervals are insensitive to real changes in individuals when the index of individuality for the test is less than 0.6. An index greater than 1.4 indicates that observed values can be evaluated usefully against reference values (16, 17, 23).

Our data indicate that within an individual thyroid hormone concentrations are maintained within relatively narrow limits. In addition, we found large variations among individuals and thus a low index of individuality for all thyroid function tests. This demonstrates that conventional population-based reference intervals for thyroid function tests may be unable to detect abnormal test results that are considerably outside the normal range for the individual being tested. These findings are consistent with previous observations (18, 19, 20). Browning *et al.* (19) studied 12 subjects over a period of 5 wk and found ratios of intra- to interindividual variance about 0.6 for all thyroid function tests. Rasmussen *et al.* (18) found ratios of intra- to interindividual variances quite variable (0.27–2.7) because of large differences in variance between individuals. Nagayama *et al.* (20) collected five specimens from each of 47 subjects and confirmed a marked degree of individuality over a period of 2 wk. These studies were performed over shorter periods of time using highly standardized preanalytical conditions. Less standardized preanalytical conditions and rhythmic aberrations of lower frequencies in thyroid function tests (14, 15) may have a different impact on within- vs. between-individual variance and thus change the ratios of intra- to interindividual variance, the individuality index. However, our data indicate that the index of individuality is low in routine laboratory testing of thyroid function over a 1-yr period also.

In our study each individual had a unique thyroid function. Individual set points could be determined by a single blood test within 25% variation



for  $T_4$ ,  $T_3$ , and FTI and within 50% variation for TSH. The difference in test results required for significant changes from the individual set point was relatively large. Previous studies have shown that the problem of unique thyroid function in the individual when testing against a laboratory reference range cannot be solved by stratification (24, 25) or by computation of multivariate reference regions (23, 26). Hence, separate reference ranges for serum TSH,  $T_3$ ,  $T_4$ , and FTI, depending on, for example, sex and age or evaluation of TSH for different levels of serum  $T_4$ , will not improve clinical interpretation.

The study was done in men. Variation in thyroid hormones may be different in menstruating women, although hormone levels are unchanged throughout the normal menstrual cycle (14, 27) and circadian variation showed no difference between men and women (28).

Participant number 7 had no clinical symptoms or signs of thyrotoxicosis, but he had a permanently low TSH, which is common in our area (29). Over 1 yr he had one measurement of elevated serum  $T_3$  compatible with overt hyperthyroidism but 11 measurements of  $T_3$  within the population-based reference range, indicating subclinical hyperthyroidism. If it is hypothesized that participant number 7 would normally have the same set point for serum  $T_3$  as participant number 8, then all 12 measurements of  $T_3$  in participant number 7 would be outside the 95% confidence interval for the individual, indicating overt hyperthyroidism. This illustrates how subclinical thyroid disease could be overt hypo- or hyperthyroidism hidden behind vague symptoms and insensitive reference ranges for serum  $T_3$  and  $T_4$ .

The pituitary gland is sensitive to minor changes in serum  $T_3$  and  $T_4$ , and serum TSH responds heavily to such changes (30). When thyroid function is abnormal, the association between serum TSH and both  $T_3$  and  $T_4$  is log linear (25, 31). This amplified response of serum TSH to changes in serum  $T_3$  and  $T_4$  may cause serum TSH to leave the population-based reference range when serum  $T_3$  and  $T_4$  are outside the individual reference range, even when they are still within the population-based reference range. This is labeled subclinical thyroid disease. The view that individuals with subclinical thyroid disease have abnormal thyroid function is supported by increasing amounts of data on the biological importance of subclinical thyroid disease for a number of organs (4, 5, 6, 7, 8, 9).

Our data indicate that the distinction between subclinical and overt thyroid disease is somewhat arbitrary because it depends to a considerable extent on the position of the patient's normal set point for  $T_3$  and  $T_4$  within the laboratory reference range. For example, serum FTI had to decrease by 14 nmol/liter below the set point in participant 8 to be reported subnormal by the laboratory. In participant 10, on the other hand, FTI had to decrease by 66 nmol/liter below the individual set point. Hence, participant 10 had to be considerably more hypothyroid to have a diagnosis of overt hypothyroidism. The opposite relation existed to have a diagnosis of overt hyperthyroidism based on an increase in FTI. This

would happen much earlier in participant 10 (increase of 6 nmol/liter) than in participant 8 (increase of 58 nmol/liter required). These differences emphasize the importance of abnormalities in serum TSH, more or less independent of serum T<sub>3</sub> and T<sub>4</sub>.

In conclusion, we found that individual reference ranges for serum T<sub>3</sub> and T<sub>4</sub> are about half the width of population-based reference ranges. Hence, a test result within the laboratory reference limits is not necessarily normal for the individual. Serum TSH outside the population-based reference range indicates that serum T<sub>3</sub> and serum T<sub>4</sub> are not normal for the individual.

### Acknowledgments

### Footnotes

Abbreviations: CV, Coefficient of variation; FTI, free T<sub>4</sub> index.

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Table 1. Hormone levels and individual interval of variation in 16 subjects (mean  $\pm$  2 SD)

Participant	TSH, mU/liter, mean ( $\pm$ 2 SD)	Total T <sub>3</sub> nmol/liter, mean ( $\pm$ 2 SD)	Total T <sub>4</sub> nmol/liter, mean ( $\pm$ 2 SD)	Free T <sub>4</sub> index, nmol/liter, mean ( $\pm$ 2 SD)
1	1.71 (0.54-2.89)	1.65 (1.09-2.21)	108 (63-152)	107 (68-146)
2	1.54 (0.72-2.35)	1.67 (1.12-2.23)	98 (84-113)	87 (67-107)
3	1.61 (0.89-2.32)	1.58 (1.04-2.12)	130 (99-162)	123 (91-154)
4	0.95 (0.52-1.38)	1.57 (1.13-2.00)	88 (75-102)	87 (71-104)
5	1.49 (0.96-2.01)	1.34 (0.85-1.83)	95 (80-110)	93 (80-105)
6	0.88 (0.32-1.43)	1.64 (1.12-2.15)	99 (74-124)	84 (65-102)
7 <sup>1</sup>	0.00 (0.00-0.01)	2.36 (1.69-3.03)	129 (109-149)	122 (104-141)
8	1.68 (1.04-2.31)	1.03 (0.44-1.61)	85 (79-91)	83 (75-90)
9	1.11 (0.79-1.42)	2.01 (1.62-2.41)	110 (92-128)	109 (90-128)
10	0.67 (0.34-1.00)	2.10 (1.86-2.35)	137 (130-144)	135 (121-148)
11	2.42 (1.60-3.24)	1.72 (1.53-1.92)	81 (63-99)	84 (65-104)
12	0.48 (0.32-0.64)	2.05 (1.72-2.37)	136 (119-153)	127 (114-141)
13	1.07 (0.50-1.65)	1.72 (1.52-1.91)	122 (88-157)	116 (73-159)
14	1.12 (0.45-1.79)	1.54 (1.31-1.76)	108 (86-129)	105 (79-131)

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15	1.15 (0.70–1.59)	1.51 (1.35–1.68)	89 (72–106)	86 (70–102)
16	1.25 (0.91–1.60)	1.47 (1.30–1.64)	109 (100–118)	111 (75–146)
Average <sup>2</sup>	1.27 (0.66–1.89)	1.64 (1.24–2.04)	106 (87–126)	102 (80–125)
Overall group <sup>3</sup>	1.27 (0.16–2.39)	1.64 (0.97–2.31)	106 (65–148)	102 (61–144)
Reference ranges <sup>4</sup>	– (0.3–5.0)	– (1.2–2.7)	– (60–140)	– (70–140)
Individuality index <sup>5</sup>	0.49	0.58	0.54	0.59

<sup>1</sup> Participant number 7 was excluded from calculations because he was subclinically hyperthyroid.

<sup>2</sup> Calculated values for an average participant; reference intervals calculated as mean  $\pm$  2 SD; average SD is the square root of the average variance.

<sup>3</sup> Calculated values for the group, excluding participant 7.

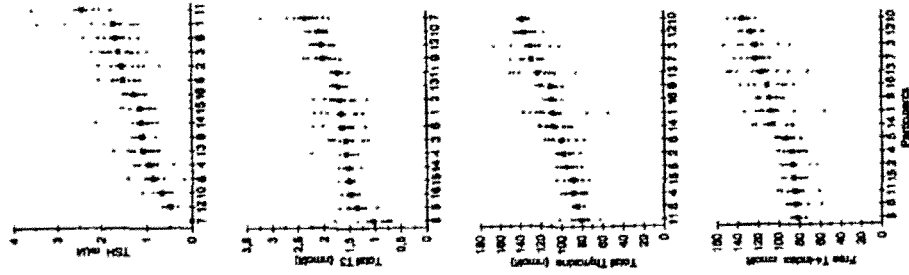
<sup>4</sup> The conventional population-based reference limits used in our routine laboratory.

<sup>5</sup> Individuality index was calculated from  $SD_{analytical+intraindividual}/SD_{interindividual}$ . The individuality index describes the sensitivity of population-based reference ranges to abnormal test results in the individual ( $<0.6$  insensitive;  $>1.4$  sensitive).

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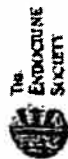
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Debate and controversy currently surround the recommendations of a recent consensus conference that considered issues related to the management of early, mild, or so-called subclinical hypothyroidism and hyperthyroidism. Intimately related to the controversy is the definition of the normal reference range for TSH. It has become clear that previously accepted reference ranges are no longer valid as a result of both the development of more highly sensitive TSH assays and the appreciation that reference populations previously considered normal were contaminated with individuals with various degrees of thyroid dysfunction that served to increase mean TSH levels for the group. Recent laboratory guidelines from the National Academy of Clinical Biochemistry indicate that more than 95% of normal individuals have TSH levels below 2.5 mIU/liter. The remainder with higher values are outliers, most of whom are likely to have underlying Hashimoto thyroiditis or other causes of elevated TSH. Importantly, data indicating that

► Neuroendocrinology and Pituitary  
► Thyroid

African-Americans with very low incidence of Hashimoto thyroiditis have a mean TSH level of 1.18 mU/liter strongly suggest that this value is the true normal mean for a normal population. Recognition and establishment of a more precise and true normal range for TSH have important implications for both screening and treatment of thyroid disease in general and subclinical thyroid disease in particular.

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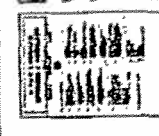
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J. Clin. Endocrinol. Metab., July 1, 2006; 91(7): 2624 - 2630.  
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THE JOURNAL OF CLINICAL ENDOCRINOLOGY & METABOLISM



W. Teng, Z. Shan, X. Teng, H. Guan, Y. Li, D. Teng, Y. Jin, X. Yu, C. Fan, W. Chong, F. Yang, H. Dal, Y. Yu, J. Li, Y. Chen, D. Zhao, X. Shi, F. Hu, J. Mao, X. Gu, R. Yang, Y. Tong, W. Wang, T. Gao, and C. Li  
**Effect of iodine intake on thyroid diseases in China.**  
N. Engl. J. Med., June 29, 2006; 354(26): 2783 - 2793.  
[Abstract] [Full Text] [PDF]

gmc



### Thyroid Resistance

The studies below demonstrate that thyroid resistance is a significant problem in Chronic Fatigue Syndrome and Fibromyalgia. Many doctors don't believe it is a true dysfunction even though it is becoming well documented. The Los Angeles Times even did a story on the problem of a fire retardant, which is band in every other country except the United States, building up in people's bodies and blocking the effect of thyroid. This is a major problem for most Chronic Fatigue Syndrome and Fibromyalgia patients as well as being a problem for the population in general. It is, however, much worse in CFIDS and Fibromyalgia.

Thyroid resistance is basically a condition in which the thyroid in the blood has less of an effect than is normal. Documented causes of thyroid resistance includes viruses, bacteria, yeast, toxins, plastics, fire retardants, pesticides and reverse T3 to name a few. When a doctor "checks your thyroid" he or she is actually checking thyroid hormone levels. What is really the goal is not to know how much thyroid is in the blood, but instead, what the thyroid effect a person is getting. The problem is that there could be normal thyroid levels, but because there is thyroid resistance, there is poor thyroid effect.

There is no standard blood test, but those very familiar with this condition can usually recognize it with extensive thyroid panels. Treatment can be done by eliminating the cause, which can infection or toxin, or overcoming the resistance by giving higher doses of thyroid and watching the effect. High dose inositol can sometimes be beneficial, but it is best to remove the cause of the resistance. A possible screening question to check if your potential doctor is an expert in Chronic Fatigue Syndrome and Fibromyalgia is to ask if he or she treats thyroid resistance.

A metabolic basis for Fibromyalgia and its related disorders: The possible role of resistance to thyroid hormone. Med Hypotheses 2003-7-31 61(2) 182-9

*Effectiveness and safety of T3 (triiodothyronine) therapy for euthyroid Fibromyalgia: a double-blind placebo-controlled response-driven crossover study. Clinical Bulletin of Myofascial Therapy, 2(2/3):31-58, 1997*

Ms. Patricia Collins.

BMJ 2000;320:1332-1334 (13 May)

## Education and debate

### Thyroid function tests—time for a reassessment

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In 1999, 890 000 measurements of thyroid stimulating hormone were performed by Scottish hospital laboratories—approximately one test for every six of Scotland's 5.1 million people.<sup>1</sup> This number does not include tests performed in the non-NHS laboratories or as part of the screening programme for congenital hypothyroidism. Although laboratory statistics are not collected nationally in England and Wales, the market in the United Kingdom (population 59 million) for thyroid stimulating hormone diagnostic tests is currently estimated at 9-10 million each year.

A remarkable downgrading of the clinical aspects of hypothyroidism and hyperthyroidism has paralleled the inexorable increase in the number of thyroid function tests performed over the past 20 years. This has led to chaos in the diagnosis of hypothyroidism. It has been stated that a diagnosis of clinical hypothyroidism can be made on the basis of biochemical measurements alone and that signs and symptoms are unnecessary.<sup>2</sup> Other authors protest, and maintain that biochemical tests can be misleading and that the diagnosis can be made on clinical grounds alone.<sup>3</sup> In hyperthyroidism, a suppressed thyroid stimulating hormone concentration is currently the cornerstone of biochemical diagnosis. No numerical value has been assigned to the serum concentration of thyroid stimulating hormone below which suppression is considered to occur. This value varies from centre to centre depending on the sensitivity of the local assay. Thus, to many non-specialists the diagnosis of hyperthyroidism is also confusing.

### Summary points

- There are no data on the relative importance of biochemical thyroid function tests and clinical symptoms and signs in assessing thyroid dysfunction
- Secretion of thyroid stimulating hormone is influenced by many factors other than the negative feedback inhibition by thyroxine or triiodothyronine
- Changes in thyroid stimulating hormone, thyroxine, and triiodothyronine concentrations during systemic illness are poorly understood
- Thyroid function tests cannot be interpreted in patients with systemic illness
- Since thyroid stimulating hormone concentrations are distributed logarithmically in the population, minor changes are unlikely to be clinically important
- The possibility of false positive and false negative results should be considered in interpreting thyroid stimulating hormone concentrations

### Methods

This review is based on my 20 years' postgraduate experience in providing biochemical thyroid function tests and treating patients with thyroid disorders. I have selected and highlighted some of the publications that have influenced my practice and call into question the increasing reliance on biochemical thyroid function tests in making a diagnosis.

### Historical setting

The treatments currently used for hyperthyroidism and hypothyroidism were established by the beginning of the 1970s. Though the symptoms and signs of these disorders had been analysed and clinical scoring indices had been developed and validated in the 1960s, clinical diagnosis remained problematic.<sup>4-6</sup> The clinical diagnostic schemes for hypothyroidism were similar,<sup>4-6</sup> but there were considerable differences between diagnostic schemes for hyperthyroidism. For example, atrial fibrillation was considered by Wayne and Crooks to be one of the most powerful discriminating signs,<sup>6</sup> but it was not included by Gurney *et al.*<sup>5</sup> Age, on the other hand, was a major diagnostic factor according to Gurney *et al.*,<sup>5</sup> but was not mentioned by Wayne or Crooks.<sup>6</sup> From knowledge of the pathophysiology of the hypothalamic-pituitary-thyroid axis available at that time, it was believed that measuring the concentration of serum thyroid stimulating hormone would simplify the diagnosis.

### Hypothyroidism

The publication of a reliable and practical assay for thyroid stimulating hormone was a landmark.<sup>8</sup> A normal range of 0.5-4.2 mIU/l was established, based on measurements from 29 control subjects. One of the first applications of the assay was in patients who had undergone subtotal thyroidectomy for Graves' disease.<sup>10</sup> In 28 "unequivocally euthyroid" patients followed for three to 21 years, the mean concentration was 8.2 mIU/l (range 1.3-34.0 mIU/l). In four patients followed up for four to 12 years and in whom a therapeutic trial of thyroxine had shown no benefit, the thyroid stimulating hormone concentration range was 10.5-21.5 mIU/l. These patients were considered to be unequivocally euthyroid by a group who had validated clinical indices for the diagnosis of hypoparathyroidism and hyperthyroidism.<sup>5</sup> They were used to show the superiority of thyroid stimulating hormone measurements in detecting hypothyroidism, and no suggestion was made that the normal range could be widened.

In 1973, the data on which the concept of subclinical hypothyroidism was based were published.<sup>11</sup> The reference range for thyroid stimulating hormone, established from measurement in 29 subjects,<sup>10</sup> was used to classify 22 euthyroid subjects as having subclinical hypothyroidism. In six of the 22 subjects given a therapeutic trial of thyroxine, treatment showed no benefit, and 10 had originally been recruited as normal controls.

Whickham survey



The Whickham survey was a further landmark.<sup>12</sup> All Whickham residents with a serum thyroid hormone concentration  $>6$  mU/l were diagnosed as being hypothyroid, irrespective of their clinical status. This reinforced the view that the serum thyroid stimulating hormone concentration defined hypothyroidism.

The 20 year follow up study of the Whickham survey has yielded invaluable data on the natural history of thyroid disorders.<sup>13</sup> A main conclusion of the study, disseminated to most non-specialists in a review published in the *BMJ*, was that "thyroid stimulating hormone concentrations above 2 mU/l are associated with an increased risk of hypothyroidism."<sup>2</sup> Half of the population (male and female) fall into this category.<sup>12</sup> This conclusion was based on the change in the slope of the line obtained when the log of the serum thyroid stimulating hormone concentration was related to the logit probability of developing hypothyroidism over a 20 year period in women (see box).<sup>13</sup> The probability of a 40 year old woman with a thyroid stimulating hormone of 2.1 mU/l developing hypothyroidism is low—at 1 in 50 over 20 years. In men, the probability is so low that an equivalent equation could not be derived.<sup>13</sup>

#### Relation between concentration and risk

The equation to describe the relation between the probability of developing hypothyroidism and the serum thyroid stimulating hormone concentration is:<sup>13</sup>  $\ln [P/(1-P)] = b_0 + b_1$  in thyroid stimulating hormone  $+0.027$  age  $+1.79$  if antibody positive).

$b_0 = -5.02$ ,  $b_1 = 0.30$  if thyroid stimulating hormone  $<2$  mU/l

$b_0 = -6.38$ ,  $b_1 = 1.97$  if thyroid stimulating hormone  $\geq 2$  mU/l

#### Clinical features ignored

The review also highlighted the fact that in making a diagnosis of clinical or overt hypothyroidism "symptoms are not considered a criterion by some authorities."<sup>2</sup> The review claimed great authority. It was pointed out that some of the data on which it was based had been collected for the consensus statement for good practice and audit measures in the management of hypothyroidism and hyperthyroidism published on behalf of the Royal College of Physicians of London and the Society for Endocrinology.<sup>14</sup> This publication makes no reference to the clinical manifestations or clinical diagnosis of hypothyroidism. Thus, the clinical features of hypothyroidism seem to have been relegated to the status of historical curiosities.

### ► Hyperthyroidism

Assays capable of defining the lower end of the statistically derived reference range became available in the early 1980s. One evaluation of such an assay reported that all of 110 hyperthyroid patients studied had a thyroid stimulating hormone concentration  $<0.07$  mU/l, and all 82 euthyroid control subjects had concentrations  $>0.07$  mU/l.<sup>15</sup> However, some clinically euthyroid subjects with abnormally low thyroid stimulating hormone concentrations were classified as having subclinical hyperthyroidism.<sup>15</sup> Assays can now detect thyroid stimulating hormone in serum at concentrations of 0.005 mU/l.<sup>16</sup> At this low concentration, hyperthyroid patients were not distinguished from some euthyroid, though ill, patients.<sup>16</sup> The range of thyroid stimulating hormone concentrations in patients whose condition stabilised on thyroxine replacement treatment was  $<0.005$  to  $>10.00$  mU/l.<sup>16</sup> It is therefore clear that measurement of the thyroid stimulating hormone concentration has failed to deliver what was expected of it.

#### Clinical aspects

During this period the clinical aspects of hyperthyroidism have also been downgraded. Most current undergraduate textbooks treat the clinical diagnosis of thyroid dysfunction by referring the student to *Esq*. In the current edition of the *Oxford Textbook of Medicine*, this matter is dismissed in less than a line, and the reader is referred to unweighted lists of the symptoms and signs.<sup>17</sup> In the popular postgraduate textbook of *Clinical Endocrinology*, the biochemical diagnosis and assessment of hyperthyroidism are given before the clinical features.<sup>18</sup> Medical journals are now effectively devoid of references to the clinical features of hyperthyroidism. Though a symptom rating scale for the diagnosis of hyperthyroidism was described in 1988,<sup>19</sup> the clinical scoring systems for assessing hypothyroidism and hyperthyroidism are now rarely cited (table).

Citation frequency (in BIOS) of published papers on the clinical assessment of hypothyroidism and hyperthyroidism in relation to UK groups and worldwide, 1987-97

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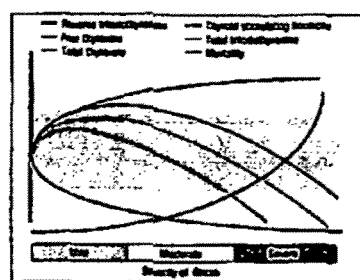
### ► Non-thyroidal illness syndrome

We have recently become aware of the complexity of the effects of non-thyroidal illness on the hypothalamic-pituitary-thyroid axis and thyroid hormone metabolism. Figures like the one shown (taken from a recent review<sup>20</sup>) are frequently used to illustrate the nature of the changes that occur in serum thyroid hormone concentrations in the non-thyroidal illness syndrome. These figures have never been published with a numerical scale or error bars. The problem of interpreting free thyroxine was summarised by the author: "It is common to find that a sample obtained from a patient with non-thyroidal illness syndrome may have a raised free thyroxine by one method but a normal or low free thyroxine by another."<sup>20</sup> The equilibrium dialysis reference method used to profile free thyroxine in the figure is technically demanding and currently not established in the United Kingdom. As the original legend to the figure explains:

The profile for free thyroxine is that obtained using equilibrium dialysis and low sample dilution. The level of free thyroxine found using commercial methods will be heavily method dependent. A profile of free triiodothyronine is not included as some ultrafiltration methods suggest that normal or raised free triiodothyronine may be found in illness whilst equilibrium dialysis methods usually show diminished or normal concentrations.<sup>20</sup>

What free thyroxine and free triiodothyronine assays actually measure is controversial.<sup>21</sup> However, what is clear is that we cannot interpret thyroid function tests in systemically ill patients.

The effects of illness on the concentrations of thyroid hormones: the shaded area represents



the reference range for each method (reproduced with permission from Beckatt and Widdison<sup>20</sup>)

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### ► Current status of thyroid function tests

Our understanding of the complexity of the cerebral-hypothalamic-pituitary-thyroid axis and the mechanism of thyroid hormone action has grown enormously. Current knowledge indicates that the cardiac effects of thyroid hormones, which are clinically very important, are mediated via the  $T_{4}$  thyroid hormone receptor independent of the  $\beta$  receptors, which are the dominant regulators of thyroid stimulating hormone secretion.<sup>22</sup>

#### False positive and negative results

Overlap between the statistically derived normal and abnormal ranges is accepted in diagnostic tests, giving rise to false positive and false negative results. These concepts have not been applied to measurements of thyroid stimulating hormone. Rather than accepting that the test can be fallible, we transfer the problem to the patient. In patients with systemic disease, the non-thyroidal illness syndrome is invoked to explain the anomalous results, and healthy subjects are diagnosed as having subclinical hypothyroidism or hyperthyroidism.<sup>11-15</sup> The distribution of the serum thyroid stimulating hormone concentration in the population is logarithmic.<sup>13</sup> Thus, minor deviations from the statistically derived reference range are unlikely to be clinically meaningful.<sup>11</sup>

#### Confusion

Studies in 1580 inpatients<sup>23</sup> and in 630 patients admitted as medical emergencies<sup>24</sup> found that thyroid function tests performed as screening tests yielded abnormal results in 33% and 20% of patients respectively. In both studies, the biochemical tests suggested thyroid disease incorrectly (that is, they gave false positive results) in nine cases out of 10. Thus, indiscriminate use of thyroid function tests is more likely to confuse than to help.

We do not know how important the thyroid function tests are for making a diagnosis of thyroid dysfunction. It is a matter of personal judgment. Experience has shown that thyroid function tests, like all the signs and symptoms associated with hypothyroidism and hyperthyroidism, are not totally reliable. As it becomes clear that biochemical assessments cannot deliver the diagnostic accuracy expected of them, the fact that the clinical aspects of assessing thyroid dysfunction are being sidelined is a cause for concern. Doing more biochemical tests will lead to further confusion, not the hoped for clarity. The information obtained from thyroid function tests, despite its quantitative numerical appearances, is "soft." How soft has yet to be established.

### ► Acknowledgments

I thank Dr David Lyon for mathematical help, Dr Ann Wales for obtaining the citation data given in the table, and Drs G H Beasall and H G Gray for constructive comments and discussion.

### ► Footnotes

Competing interests: None declared.

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#### Related Article

##### Thyroid function tests

P Kendall-Taylor, Alun Price, A P Westman, A D Toft, G J Beckett, Sudha Bulusu, Martin Eales, and Denis O'Reilly  
 BMJ 2000 321: 1060. [Extract] [Full Text]

**December 12, 2005**

**Question:** I'm a physician in North Carolina who uses your book *The Metabolic Treatment of Fibromyalgia* as a manual for treating my fibromyalgia patients. I have now gotten many of these patients well by using doses of Armour or Cytomel that my partners consider excessive. Their main concern is that the doses I use may cause heart arrhythmias. A local endocrinologist recently told one of my partners that sooner or later, I'm going to cause some of these patients to have heart attacks. Because of the documentation you give in the book, I'm comfortable using doses of thyroid hormone that are obviously necessary for the patients to get well. I am curious, however, what your current position is on the issue of thyroid treatment and arrhythmias.

**Dr. Lowe:** Hardly a week passes that I don't receive an email from a physician asking the same question you have. My answer here is typical of what I send to them.

The major concern of our research and treatment team over the years has been the safety of our patients. Out of that concern, I've given the subject of thyroid hormone therapy and heart arrhythmias intense focus. I have studied the entire research literature on the subject. In addition, our research and treatment team may have run more ECGs (EKGs) and ordered more advanced cardiac testing than any other clinic not specializing in cardiology.

We ran so many in years past that the results forced us to a conclusion: it's the rarest exception when the ECG rhythm of a patient is of concern. We've referred for cardiac consults the few patients who had rhythms we were concerned about. Only once did a cardiologist advise that the patient undergo cardiac rehab before beginning the use of thyroid hormone. In every other case, the cardiologist said that thyroid hormone therapy was safe for the patient. Some cardiologists said that the therapy would most likely improve the patient's cardiac health.

My years of focus on this issue boil down to a few evidence-based beliefs. Arrhythmias occur in some patients with hypothyroidism and thyroid hormone resistance. They occur when the patients' doctors deny them thyroid hormone therapy, or when their doctors under-treat them with thyroid hormone. We usually say these patients' arrhythmias result from "hypothyroid heart."

Arrhythmias also occur in some patients with suppressed TSH levels. It's not clear at all, however, that excessive thyroid hormone stimulation causes these patients' arrhythmias. Unfortunately, the endocrinology specialty has concluded from these studies that anyone taking TSH-suppressive doses of thyroid hormone is likely to have heart arrhythmias. But this conclusion is simply illogical, and it is self-contradictory for the specialty, as I explain below.

Some researchers have reported an association of suppressed TSH levels with heart arrhythmias. These studies, however, included only elderly, sedentary individuals, some of whom were bedridden in nursing homes. None of the researchers controlled for the influence of most of the important heart-protective lifestyle factors. Because of this, the arrhythmias may have been more strongly or wholly associated with unwholesome diet, nutritional deficiencies, or low levels of cardiovascular fitness.

Also, the suppressed TSH levels of people in the studies weren't caused by thyroid hormone therapy; researchers only found the low TSH levels upon reviewing medical records of the people. This raises the possibility that

factors other than too much thyroid hormone were responsible for the people's suppressed TSH levels.

For example, some of the people in the studies may have had pituitary hypothyroidism. This is a condition in which the pituitary gland doesn't produce a normal amount of TSH; as a result, patients have abnormally low TSH levels. It's important to note that these patients have *deficiencies* of thyroid hormone, so thyroid hormone overstimulation couldn't be the cause of their arrhythmias. Other patients in the studies may have had some degree of Graves' diseases. If so, then it's possible that their arrhythmias were caused by some sort of cardiac cross-reaction with thyroid stimulation antibodies.

For real clarity on this issue of arrhythmias, we must compare patients in the studies I just mentioned (elderly, sedentary, often bedridden people) to thyroid cancer patients. The comparison reveals an outrageous double standard of therapy by the endocrinology specialty.

Nearly all thyroid cancer patients use TSH-suppressive dosages of thyroid hormone. Through meta-analyses of many studies, researchers looked at the heart condition of these patients—some of whom have suppressed their TSH levels with thyroid hormone for decades. The researchers found that the suppression has *not* compromised the health of the patients' hearts.

Talking out of both sides of its collective mouth, the endocrinology specialty continues to treat thyroid cancer patients with TSH-suppressive dosages of thyroid hormone, while authoritatively warning hypothyroid patients and other doctors that TSH-suppressive doses are likely to cause arrhythmias and heart attacks. The specialty's self-contradicting inconsistency is so glaring that it unveils conflicts of interest that honorable people would be ashamed to be caught in.

From my study and clinical experience of this issue over the years, I've derived a distinct impression that I justify in my forthcoming book *Tyranny of the TSH*: The endocrinology specialty is unyielding in its endorsement of T4-replacement, and this appears to me to be a commitment to stabilize the financial market for the associated products of T4 replacement. Those products in the U.S. include Synthroid, and importantly, they also include the TSH, free T4, and free T3 tests provided by labs.

It's my belief that the specialty uses the theoretical possibility of cardiac arrhythmias as a scare tactic to intimidate other doctors into ordering more-and-more of these lab tests, especially TSH tests. The intimidation ensures the continuing huge sales of these tests. The sales please the corporations that market the tests, and as a *quid pro quo*, the corporations generously share their profits with the specialty. (As others have noted, the sharing comes as huge financial grants, speaking fees, sponsoring of speaking appearances, research funds, free drug samples and patient literature, and logo gift items.)

The endocrinology specialty's obvious financial conflicts of interest are a devastating blow to its credibility. In my mind, it has none left. I hold suspect anything and everything that flows from the mouths or pens of the specialty. And that certainly includes its scientifically-indefensible claim that TSH-suppressive doses of thyroid hormone are likely to cause cardiac arrhythmias.

**For documentation, see:** Unrealistic Worries About Thyroid Hormone Therapy and Heart Problems: The Source and AskDrLowe: Thyroid Hormone and the Heart.

**November 9, 2005**

**Question:** I am a 41-year-old woman who lives on the east coast. About a year ago, my doctor tested me by your protocol and lab tests. When he and I did a telephone consultation with you, he agreed to put me on Cytomel. I now take 100 mcg per day. I'm doing yours and Dr. Honeyman-Lowe's protocol as you describe it in *Your Guide to Metabolic Health*. I take vitamin supplements daily and exercise at least three times a week. Since I started the protocol and Cytomel, I've regained my life. I have no more pain, no migraines, no swelling, no tingling, no insomnia, and I'm no longer cold all the time. The list of improvements goes on and on. For example, I've lost 65 lbs. I feel great. I suffered for 10 years of my life without a correct diagnosis, so needless to say, I don't want to go back.

The problem I'm facing is that my TSH is very low and my T3 is high. On occasion, I feel that my heart is pounding or I feel anxious. Other than these symptoms every once in a while, I don't feel overstimulated. But because of these symptoms and the lab results, my doctor wants to take me totally off T3 and send me to a local endocrinologist. I've inquired at the endocrinologist's office and learned that he doesn't believe in using Cytomel or your protocol. What can I do? There must be other options than just taking the Cytomel away completely. I feel good now and live an active lifestyle. I don't want that taken away. Please help. I'm desperate not to go back to the way I was before.

**Dr. Lowe:** The improvements you describe are typical of what we hear from patients using high-enough doses of Cytomel. Because of your improvements, and because your symptoms of possible overstimulation are occasional, taking you completely off Cytomel seems to me radically improper.

For someone taking 100 mcg of T3, we expect your pattern of lab results—a low TSH and high T3. However, your TSH and T3 levels are irrelevant to whether you're overstimulated or not. Two studies we just completed confirm other researchers' findings: these tests are *not* reliable gauges of a patient's metabolic status. Many patients taking T3 have TSH and T3 levels like yours but still have severely low metabolic rates. Their metabolic rates become normal only when they increase their dosages further. Their metabolic rates become normal and they have no detectable overstimulation.

In some cases such as yours, the patient's Cytomel dose may need to be reduced. But symptoms such as occasional heart pounding and anxiety are usually not due to a patient's Cytomel dose. I say this because when Cytomel is solely responsible, symptoms of overstimulation are consistent, not occasional.

However, it's important to consider whether a patient's Cytomel dose is high enough to sensitize her to other stimulating chemicals. (Examples are caffeine in coffee, theobromine and theophylline in chocolate, and ephedrine in cold medicines.) If the Cytomel has excessively sensitized her to such chemicals, then when she consumes them in foods or medicines, she'll experience transient symptoms of overstimulation. She'll be overstimulated for a few hours, but then the symptoms will disappear. The Cytomel will have also excessively sensitized her to her own adrenaline and noradrenaline. Because of this, emotional arousal or intense exercise might also cause temporary symptoms of overstimulation.

The proper solution to occasional symptoms of overstimulation is to find the causes and correct them. The patient's may have to reduce her Cytomel



dosage low enough to relieve excess sensitivity to stimulating chemicals. And she may have to reduce her intake of such chemicals. In general, though, the proper approach is not to take the patient completely off Cytomel—not when it has relieved her troubling and disabling symptoms.

Most endocrinologists subscribe to the practice guidelines of the American Association of Clinical Endocrinologists. When a patient such as you sees one of these endocrinologists, he's likely to take her off T3 and switch her to T4-replacement. As many patients have told us, when an endocrinologist switched them to T4-replacement, they became ill and dysfunctional again. These reports are consistent with studies that show the ineffectiveness and potential harm of T4-replacement. The studies show that T4-replacement leaves many patients suffering chronically from hypothyroid symptoms<sup>[1][2][3][4][5][6][7]</sup> and gaining weight they can't lose through dieting and exercise.<sup>[8]</sup> The patients are also likely to use more drugs and develop one or more of several potentially-fatal diseases.<sup>[9]</sup>

Potential harm from T4-replacement has thus been scientifically documented. In view of the risks, you must consider for yourself whether you'll permit your therapy to be changed from Cytomel to T4-replacement. If you decide not to permit it, you can seek out an alternative doctor who understands how ineffective and harmful T4-replacement is for many patients. Alternative doctors are generally more cooperative than conventional doctors, and most of them take the time to learn the cause of troubling symptoms. Because of this, you should be able to find one who'll help you ferret out and correct what's causing your occasional symptoms of overstimulation.

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☐ 1: [Eur J Endocrinol. 2000 Nov;143\(5\):639-47.](#)[Links](#) BioScientifica

FREE FULL TEXT

Prevalence of thyroid disease, thyroid dysfunction and thyroid peroxidase antibodies in a large, unselected population. The Health Study of Nord-Trøndelag (HUNT).

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**OBJECTIVE:** To examine the prevalence of thyroid disease and dysfunction including thyroid autoimmunity in Norway. **MATERIALS AND METHODS:** All inhabitants 20 years and older (94009) in Nord-Trøndelag were invited to participate in a health survey with a questionnaire and blood samples. **RESULTS:** The prevalence of former diagnosed hyperthyroidism was 2.5% in females and 0.6% in males, hypothyroidism 4.8% and 0.9%, and goitre 2.9% and 0.4% respectively. In both sexes the prevalence increased with age. In individuals without a history of thyroid disease the median, 2.5 and 97.5 percentiles for TSH (mU/l) were 1.80 and 0.49-5.70 for females and 1.50 and 0.56-4.60 for males. The TSH values increased with age. When excluding individuals with positive thyroid peroxidase antibodies (TPOAb) (>200U/ml), the 97.5 percentiles dropped to 3.60 mU/l and 3.40 mU/l respectively. The prevalence of pathological TSH values in females and males were TSH >10mU/l 0.90% and 0.37%; TSH 4.1-9.9mU/l 5.1% and 3.7%; and TSH <0.05mU/l 0.45% and 0.20% respectively. The prevalence of positive TPOAb (>200U/ml) was 13.9% in females and 2.8% in males. In females the lowest percentage (7.9%) of positive TPOAb was seen with TSH 0.2-1.9mU/l and increased both with lower and higher levels of TSH. The percentage of males with positive TPOAb was lower than in females in all TSH groups except for those with TSH >10mU/l (85% TPOAb positive). **CONCLUSIONS:** In spite of a high prevalence of recognised thyroid disease in the population a considerable number of inhabitants have undiagnosed thyroid dysfunction and also positive TPOAb.

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## 'Normal' TSH

ALUN STEVENS MSc FLAA

COMMENT

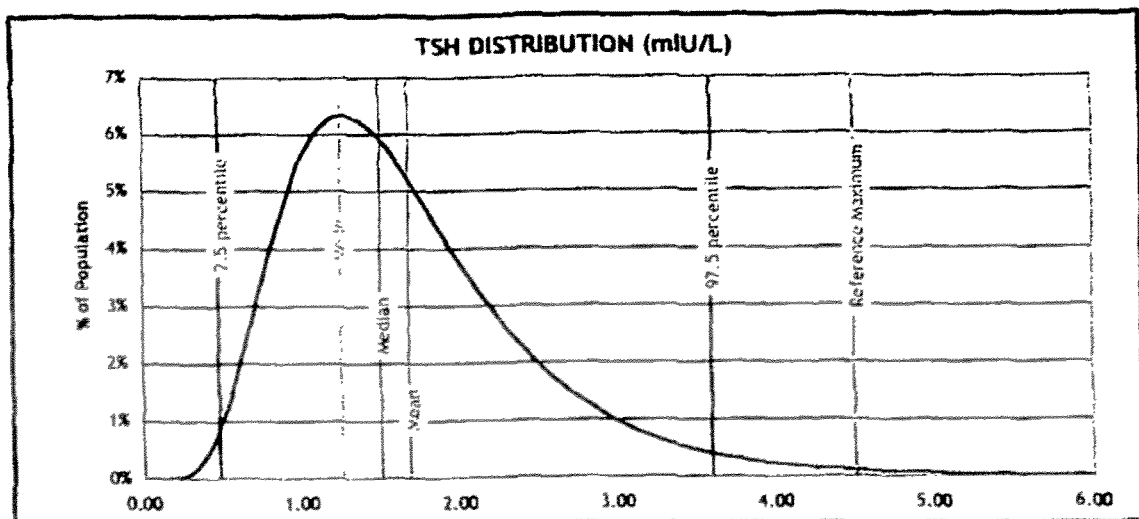
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The most common questions that Thyroid Australia is asked involve the interpretation of Thyroid Function Tests (TFT's). Many people have been told that their TFT results are 'normal'. So what is 'normal'? In this article we will focus on the test for Thyroid Stimulating Hormone (TSH) which is the most common test ordered.

The 'normal' Reference Range for the test is intended to represent the range of values which can be expected in the healthy population – ie those without any thyroid ailment. The Reference Range is found by taking a sample population of healthy individuals and determining their TSH levels. The lowest and highest 2.5% of readings are excluded so that the Reference Range covers 95% of the healthy population. There are a number of different tests for TSH with different levels of sensitivity. They each have their own Reference Range. The most common tests generally have lower limits to their Reference Ranges around 0.2 to 0.5 mIU/L and upper limits from 3.5 to 5.0 mIU/L.

A recent study in Norway provides a good example of the use of the TSH test in practice.<sup>1</sup> The study involved 65,000 people. They were asked questions about their thyroid status and those with a history of thyroid illness were excluded. The blood samples were tested for Thyroid Peroxidase Antibodies (which are an indicator of likely thyroid illness) if they produced a TSH reading greater than 4. Samples with positive antibody results were also excluded. The survey, therefore, attempted to exclude people with any indication of thyroid illness, but still included those with Thyroid Peroxidase antibodies whose TSH reading was 4 or less. The TSH test kit used for the study had a nominated Reference Range of 0.2 to 4.5 mIU/L.

The results for women are shown in the chart. The results for men were only slightly different.



The features of this result are:

- The distribution of TSH readings in the healthy population is skew. It is not the common bell

shaped curve centred in the middle of the reference range.

- The most common value, or Mode, is at 1.25.
- The Median value is at 1.50. This means that half the population (50%) have a TSH reading below 1.50.
- The average, or Mean, value is at 1.68. Over 60% of the population have a TSH reading below this value.
- The centre of the Reference Range for the test kit used in the study is 2.35. Almost 85% of the healthy population have a TSH reading below this value.
- The 2.5 percentile point (ie the point which excludes the bottom 2.5% of the population) is at 0.48. The 97.5 percentile point (ie the point which excludes the top 2.5% of the population) is at 3.6. The range between the 2.5 and 97.5 percentile points (0.48 to 3.6) is much narrower than the test kit's Reference Range (0.2 TO 4.5).
- This narrowing of the range would suggest that the reference group used to calibrate the test kit possibly included people with some level of thyroid illness.
- This narrowing of the range between the 2.5 and 97.5 percentile points would potentially have been even more pronounced if all samples had been tested for Thyroid Peroxidase Antibodies.

The conclusions which can be drawn from this survey are:

- TSH results in the upper half of the Reference Range have a low probability of being 'normal'. This does not mean that they are not 'normal'. It means that they are unlikely to be 'normal'.
- The Reference Ranges for TSH tests are potentially too wide, especially at the upper end. This suggests that 'high normal' TSH readings should possibly be treated with more suspicion than they currently appear to be.
- The centre of the Reference Range is clearly not a good target point because very few of the healthy population have TSH readings around this point.
- A much better target point would be around 1.0 to 1.5. But some people will feel better at higher levels or lower levels. This supports Prof Jim Stockigt's view that the target should be a TSH reading around 1.0.<sup>2</sup>

Another important point which needs to be borne in mind when interpreting statistics like these is that it is the population which has a range of values with probabilities for each reading. Each healthy individual is only at one of the points. They are 'normal' when they are at that point. For those on thyroxine replacement, being in the Reference Range is not good enough in itself. You need to be at your own set point. This will probably be near the lower end of the Reference Range.

This analysis of the distribution of TSH readings in the healthy population supports our recommendations to thyroid patients:

- Obtain a photocopy of all your Thyroid Function Tests. Also get copies of the ones you have had done in the past. These copies will show both the readings and the Reference Ranges.
- When you are going for a new test, make a note of how you feel (especially make a note of any of the major symptoms of thyroid overactivity or underactivity), your weight and your dose. When you obtain your copy of the test result, write this information on the copy. Over time, this process will allow you to make an informed judgement in consultation with your doctor of what the correct set point is for you.
- Do not accept that a Thyroid Function Test is 'normal' just because the result is within the Reference Range if you are still feeling unwell.

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Top

# The Sunday Telegraph

Letters to the editor

Re: We're listening  
Date: 22 February 2004

Elizabeth Day's interview with Leslie Burke, the man who is to go to the High Court to challenge medical guidelines (News, February 15), demonstrates the complex and sensitive issues which are raised by medical care, especially towards the end of a person's life.

We fully support Mr Burke's plea "You need to ask me what I want. No one can know what quality of life I enjoy apart from me". The General Medical Council's guidance makes clear that patients must be fully involved in decisions about all aspects of their care. It is for patients - not doctors - to decide how much weight to give to the benefits, burdens and risks of a treatment and to decide its overall acceptability. Doctors must also seek patients' views as to how they would like to be treated if their condition worsens.

If patients are no longer able to make decisions for themselves or communicate their views, doctors must do their best to judge what the patient would want, based on earlier discussions with the patient, any views or preferences set out in a written "advance statement", and the advice of the patient's family or others close to them. We warn doctors that they must not simply use their own values to decide what treatment is in the best interests of patients whose wishes are not known.

Of course, there are other issues raised by our guidance, including whether doctors should be compelled to provide treatment which they believe to be of no benefit to a patient or not in a patient's best interests, and the circumstances in which decisions about ending treatment, including the provision of nutrition and hydration by tube or drip, should be made by the courts.

This is an exceptionally difficult area of decision-making, and one in which the law is still developing. For this reason, we welcome the forthcoming court case as an opportunity for further clarification of the law.

From:  
Ruth Evans, Chairman, Standards Committee, General Medical Council, London W1