

S.I.L.A. NEWSLETTER 21 SUMMER/AUTUMN 2007

Dear SILA Supporter,

I hope that anyone seeing the Summer/Autumn 2007 above will not think that autumn has come a bit early this year. The SILA newsletter will now be produced and sent out twice yearly, and as you will notice has now considerably more pages than the editions sent out before Winter/Spring 2007.

I am very heartened by all the information and news sent in by SILA members. I have been sent a copy of the British Lung Foundation's magazine *Breathing Space*, Summer 2007. Therein is an article, *Focus...on Sarcoidosis*, and also a feature on Henry Shelford. And herein there is more about Henry, on page twenty-three, where he briefly introduces the exciting new Social Network group that he has started up on the SILA website — just click on “Start discussion” at the, www.sila.org.uk website. But back to *Breathing Space*. In the next issue there will be an article on pulmonary fibrosis. The BLF has a leaflet on sarcoidosis; their new address, for orders of their many leaflets, is BLF Orders, Newnorth Ltd., Kempston, Bedford MK42 8NA, or go to their website at www.lunguk.org

I have also been sent by a SILA member details of a research project by the University of Edinburgh aimed at gaining an understanding of the support patients receive from the professionals. If you want to participate contact Tara Kielmann, Division of Community Health Sciences: GP Section University of Edinburgh, 20 West Richmond Street, Edinburgh EH8 9DX Tel. 0131 650 9238, email tara.keilmann@ed.ac.uk Your contact details will not be used for any other purpose.

The SILA AGM will be held on Thursday October 4th 2007 at King's College Hospital, Denmark Hill, London SE5 at 7 pm. Afterwards there will be the usual support meeting. All fully paid up members of SILA are able to vote at the AGM. I hope members will come along and raise any topics they feel may be relevant.

I have already been told of the daughter of a SILA member who hopes to run in the 2008 London Marathon to raise funds for SILA. I hope perhaps some more members may be interested in running also.

In the next issue of the SILA newsletter it is hoped to deal with aspects of the Marshall Protocol, which regulars will know has been discussed in previous newsletters.

I hope that the Patients' Stories will continue to be sent in for publication, these stories are one of the most popular features of the newsletter.

*Heather Walker, Editor, email heather@sil.org.uk
Andrew Ferguson, Assistant Editor.*



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We start with an illuminating Patient's Story from Di. What she relates typifies the general experience that unless you happen to respond to CMO (as fortunately I have done, see p.6), then with *chronic* pulmonary sarcoidosis the use of a corticosteroid becomes almost unavoidable (although there are some other medical drugs, they are normally used only when people cannot tolerate steroids). Yet steroids are almost certain to cause some ill effects alongside improving breathing, Di's experience of her skin becoming extremely thin and dry and suffering some weight gain being fairly typical.

Di's breathing was measured at regular intervals. She gave me some details. Noteworthy is that her FVC (Forced Vital Capacity) went up from a low point in 2005 of 2.48 litres on 6 May, to 2.76 litres on 18 April 2007. The FVC measurement, that is the amount of air you can force out of your lungs when you really try, is probably the most useful of the clinical measurements. 2.48 to 2.76 is an 11% increase in capacity. But what does that mean in terms of the ability to do work? With my measurement of steps per respiration cycle, I was able to answer that type of question. For instance, I found that with a measured 10% improvement in my FVC, my steps per respiration cycle went up from 5 to 9, that is an 80% improvement in ability to do work. Thus the steps per respiration cycle measurement appears to be a more sensitive measurement. Moreover it is to be preferred because it measures more than just lung capacity. It measures lung function including oxygen exchange. Anyhow it measures what really matters to the owner of the lungs!

Although Di was not making such measurements, her observations are still of interest. She told me that, for a normal person, it is a five minute walk downhill to her library. In 2005, when the lowest of her three FVC measurements was 2.48 (6 May), she could not manage that five minute downhill walk. Her latest FVC measurement is 2.76 (18 April 2007). That is only an 11% improvement over 2.48 litres, yet she says that she "can now, slowly, walk the third of a mile uphill to the nearest shops, although I get out of breath and cough before I reach the crown of the hill (but it is pretty steep). It takes me about 15 minutes to get there." That works out at an average speed of 1.2 miles per hour, which is not too bad for a pretty steep hill, and it is certainly a remarkable change from not being able to do a five minute downhill walk. So once again we see that the clinical FVC measurements tell the right story, albeit in a fairly insensitive way.

Next is *Andrew's Story Part 2*. This is an extension of my account of the experiment in using CMO to treat my chronic sarcoidosis. It is now 61 weeks into the trial, but it looks like being a good deal longer before the story is ended. However there is enough there to encourage others. I have found two other people who are suitable for a similar experiment. So far the signs have been encouraging with both of them, but it is early days yet.

Next I tackle the question of how useful is the evidence when it comes from just a few cases. With the help of Martin Desvaux, who is a PhD in physics, I come up with some answers to that in *Why the Medics are Mistaken about Small Scale Trials*. For sarcoidosis sufferers this is important, because the disease is rare, and chronic cases are very rare.

Last, but not least, is a page on what is an important development on our SILA website.

Di's Story

I was diagnosed with pulmonary sarcoidosis in January 2002, having spent the previous 18 months trying to persuade my GP that, while I may be a hysterical middle-aged woman, there was actually something wrong with me.

The doctor had decided my persistent cough was due to 'an allergy', and had been prescribing antihistamines. The cough had become an office joke – i.e. was driving everyone mad. During the August bank holiday weekend in 2001, I had gone for a walk with the family and coughed so much they became concerned and insisted I should go back to my GP. I saw a locum who made no attempt to examine me and said, in an utterly bored voice: "It's your allergy again, I'll give you a double prescription". I tried to explain how bad things had become by telling her that, when I tried to do the vacuuming, I would struggle to breathe, had to sit down and coughed until I retched. Her reply? "Get someone else to do it"!

I was utterly infuriated by this and decided there was no point in trying to get medical help. But as the year wore on, the cough became even worse and I became short of breath on any exertion. I was having terrible coughing fits and my breathing was dreadful. In November, I went to a restaurant with a 'smoking area' with a friend and, although we had sat as far away from the smokers as possible, when we left I had such a bad attack he described me as being like "a goldfish out of water". He was adamant I should go to see a doctor. Later that week, I went to see my GP who said he wasn't happy with my breathing – well, neither was I!

I was told to go for a chest x-ray. Having been seen, I was sitting in the waiting area with several other people when the radiographer rushed out in a panic, calling my name. I went to speak to him and he told me, in public in the middle of the corridor, that the x-ray looked really bad. Of course, I was terribly scared and asked what he thought it was. The reply was: "Best case scenario, a terrible chest infection."

He told me the hospital would call the GP and said to go home. I was in such a state that I went instead to see my stepmother who lives near the hospital. I had only just got there when I got a call on my mobile from the senior GP at my surgery to go to see her at once. That didn't do anything to calm me down, nor did being put in a side room rather than the main waiting room.

The GP told me that I had pneumonia. She gave me some antibiotics, told me to take a week off and said I should see a specialist. They had scared me so much that I paid to see one privately. The GP must have given me antibiotics as a sort of safety measure, since when I saw the specialist and asked whether I had pneumonia, his reply was evasive. He said that any inflammation of the lungs could be classified as pneumonia. That is true, but the intention of the question (although I would not have been able to find the words at the time) was to find out if the pneumococcal bacteria, which cause pneumococcal pneumonia, had been found. As no pathological laboratory studies had been done, the specialist was unable to give me a straight answer. I noticed he had written 'sarcoidosis?' on his notepad. I hadn't heard of sarcoidosis and had to look it up when I got home.

At the time of that first meeting I was feeling very ill but I recovered in time to return to work at the beginning of 2002. I saw the specialist again and he delivered his diagnosis with the words: “You have something boring and benign...”. Yet again, I was incensed, both with him for being so cavalier and with the radiographer and GP for giving me such a fright. Also, with hindsight, it is evident that he was not exactly truthful in telling me it is benign (although it is in most cases).

The specialist wanted to do a biopsy and as I didn't want it done under local anaesthetic I had a general anaesthetic as a day patient. Afterwards, the registrar came to see me and started with: “I have good news and bad news”. He told me they had managed to get a tissue sample but had torn my lung doing so, and I had to stay in overnight. I saw that specialist once more. When I complained about the surgical mishap, he said airily: “Oh, it could just as well have happened under a local”. Not the point...

I then tried to live with pulmonary sarcoidosis as I really didn't want to take Prednisolone as suggested by that specialist. But eventually, as I was struggling to walk across a room, my GP, a very nice man, said sadly that I really had to. I was on Prednisolone for several months in 2002 but the GP advised me to reduce the dosage 1mg at a time and see how I got on at the lower dose; and I was able gradually to wean myself off.

About this time I came across the Marshall Protocol. My GP agreed to prescribe the tetracycline minocycline, which is the first of the five antibiotics used in the Marshall Protocol, but not the statins which are part of the protocol; in fact he was violently opposed to my taking them. I was wary of ordering some for myself as I have inherited a family tendency to low rather than high blood pressure, and statins are normally prescribed for lowering blood pressure. So I tried taking the minocycline by itself (I can't now recall the starting dosage). The first time I used minocycline, the reaction seemed to be beneficial, but when I tried a second time, I got a nasty Herx. reaction where my throat swelled and I found it hard to swallow (and possibly breathe, but that might have been panic). The Marshall Protocol acolytes tried to get me to continue but I didn't, so maybe I should count as one of those who dropped out in Phase 1, except that I was not really following the protocol, since it also involves cutting out vitamin D and taking statins at about three to four times the normal dose for lowering blood pressure. I was also reluctant to follow the protocol because I had found out that high doses of statins could cause serious side effects such as muscle distension which can lead, in some cases, to extreme pain. The really serious side effect of statins, especially in large doses, is that they can enlarge the heart and trigger heart attacks – the heart is, after all, just a large, valved muscle. Some statins have been withdrawn due to their negative effect on the heart.

Unfortunately, my sarcoidosis hasn't remitted or burnt itself out and the consultant I see now thinks I have the chronic and progressive form. He is likely to be right, as the self-curing form tends to burn itself out in a couple of years. In 2004, my condition deteriorated and he insisted I restart Prednisolone, saying that if I did not: “The outcome was uncertain”.

I had a bad year in 2005 and ended up in A&E due to a sarcoidosis flare up, which involved both coughing up blood and having breathing difficulties. Previously, I had been feeling dreadful and had got to the stage where I had no energy or appetite and was spending most of the day lying on the sofa, in traditional invalid fashion. I recovered after

a massive dose of antibiotics and steroids and a few days in hospital, and my health had picked up by the end of the year. The medical team gave me both antibiotics — even through the blood tests showed no evidence of a pneumococcal infection (the cause of pneumococcal pneumonia) or any other bacterial infection — and steroids to calm the inflammation if the problem was sarcoidosis, the cause of which is of course unknown. I was reasonably well throughout 2006 and have been OK so far this year (fingers crossed), but my breathing is still somewhat below normal. Exactly how far below normal is hard to measure because if I take much exercise I start coughing which upsets the measurement.

Currently, I am taking 3mg to 4mg of Pred. per day, but over the next month will try to get down to 3mg (I also use steroid inhalers twice a day). I will be cautious about going lower than that, as it was at the 3mg level that I got into trouble on a previous occasion. But my general health is good, so I am preparing to have yet another go at getting off Prednisolone. I know steroids are psychologically as well as physically addictive and I do feel very nervous about trying to come off, but as I am breathing so well I'm really hopeful that this time I will manage it. My feelings about steroids are basically negative and, while I accept that I would probably not be as well as I am without them, they have done me some harm, especially my skin which has become extremely thin and dry. And I have put on three kilos since last year when my health and appetite started to improve – although at my age I might have gained the weight anyway!

I attend a clinic and have lung function tests every four months, although I have recently discussed dropping this to once or twice a year, and have a chest x-ray at least once a year. They also do a yearly bone scan. This is because steroids interfere with bone formation and increase the risk of osteoporosis, something which affects all of us as we get older, and post menopausal women in particular. Although the hospital has had a bad press for some things, I have no complaints about my treatment.

I have so far managed to avoid any major weight gain, mainly because I felt sick and had little appetite for four years – a typically sarcoid symptom. My appetite has improved over the last few months and I am trying very hard to separate 'steroid hunger' from a real need to eat. I am lucky that I have so few symptoms, although when I read your Winter/Spring newsletter I recognised the back ache/tiredness link. My solution had been to go back to doing some yoga, but my coughing did not make me popular in yoga classes, so I have dropped them! I would like to try CMO, but understand I will need to get off the Prednisolone first, and as mentioned that may be a problem.

I only found out about SILA recently while Googling for up-to-date information on sarc. The search found a study on [clinicaltrials.gov](http://www.clinicaltrials.gov) entitled 'Atorvastatin to treat pulmonary sarcoidosis'. This appears to be a current and genuine US government-sponsored trial, as can be seen at <http://www.clinicaltrials.gov/ct/show/NCT00279708>. However my registrar mentioned that Atorvastatin is being withdrawn, at least for some purposes or in some countries, because it has been found to cause heart attacks.

Andrew's Story Part 2

Abstract. To start an article in a newsletter with an Abstract is unusual, but I surmise that only the seriously interested will want to absorb the amount of detail provided in the main text. This Abstract, the Notes on page 13, and the four charts which follow them, cover the gist of what is important to convey the results of my CMO experiment so far.

Dramatic improvements to my chronic sarcoidosis started almost immediately after I first used CMO cream, but lung function dropped off rapidly when, after a couple of weeks, I stopped using the cream. Then, as soon as I started using capsules, all lost ground was recovered in a couple of days. That all happened at the start of the CMO experiment, 61 weeks ago. With the use of very little more CMO, progress has continued in a somewhat erratic fashion. There have been a few vicissitudes along the way, such as a couple of cough/colds (upper respiratory tract infections), which make progress harder to assess; but rapid recovery from them confirmed that sarcoidosis was in abeyance. At the time of this 61 week report, an overall 90% improvement in my sarcoidosis can be assessed. Perhaps the most informative way to describe the measured increase in lung function is to say that, at the start it was 40% of what is probably 'normal', and by the end of the 61st week it was 80% of 'normal'. The *relative* change, 40% to 80%, can be taken as accurate, although the *absolute* change depends on an assumption about what is normal.

It should be stated that since my sarcoidosis was not proved by biopsy, the most cautious approach would be to call my illness an undiagnosed disease that exhibited several of the most common symptoms of chronic sarcoidosis, namely erratic and *extreme* fatigue, persistent dry cough, dry eyes, and lung function deteriorating to a point at which it was hard to walk and talk at the same time, even on level ground. The last would have made use of a cortico-steroid inevitable, were I not to have discovered the *temporary palliative* effects of the anti-inflammatory Voltarol Emugel P (diclofenac), which allowed a postponement, and then discovered what appears, at this stage, to be the *curative* effects of cetylmyristoleate (the active constituent of CMO).

Recapitulation

Much has happened since I reported in the Winter/Spring SILA newsletter, at the end of December 2006, on an experiment to use CMO to treat my chronic sarcoidosis. At the start of the experiment, in March 2006, the sarcoidosis, (a) badly affected my lung function, (b) produced a most unpleasant effect that is fairly accurately described by my nickname for it TEES (Totally Erratic Exhaustion Syndrome), (c) caused a persistent dry cough, and (e) dry eyes during the night. In December 2006, at the time of my last report, I was only nine months into the CMO experiment, but was able to report an encouraging sign, namely that I had come through a typical cough/cold infection which had occurred during November 2006, and had made a quick recovery thereafter, which, as I will explain later, was something not at all to be expected based on my previous experience during the time that my sarcoidosis was rampant.

A month after recovery from that penultimate upper respiratory tract virus infection, on 27 December 2006, my lung function on level ground was 9.5 steps per respiration cycle.

That was a 90% improvement on the 5 steps at which I started my CMO experiment in March 2006. Also on 27 December, my lung function on a 1 in 20 slope was 7.6 steps per respiration cycle. That was 120% improvement on the 3.4 steps that I could manage at the start. Putting those together produces an average of improvement of 110%. There are further details regarding the steps per respiration cycle test at endnote 1.

Presenting the data in that way overcomes the problem of determining what ‘normal’ would be for me in the absence of sarcoidosis, but another informative presentation can be made using an estimate of ‘normality’. Based on measurements with three friends, I have settled on 11.7 steps per respiration cycle as being ‘normal’ on level ground, and 10.5 steps on a one in twenty slope — that is ‘normal’ for fit men of my 69 years of age. I should mention that the slope measurements are somewhat crude, being done over 200 metres — instead of 1000 metres. The shorter distance introduces an element of judgement as to whether the speed of walking could be maintained over a longer distance.

Using those standards of ‘normality’, on average, my lung function at the start, in March 2006, was 40% of normal (38% arithmetically but too much accuracy is unrealistic).

Employing the same benchmarks of ‘normality’, my lung function by 27 December 2006 (the end of Chart 2b and about nine months later) had improved to 80% of ‘normal’. At the end of the latest chart, on 16 May 2007, my lung function was still rating 80% of normal (100% improvement), but after some vicissitudes that I will now relate.

The latest chart, 27 Dec 2006 to 16 May 2007

During the first two and a half months of 2007, without any further CMO treatment, my lung function dropped back, as can be seen in Figure 2c (page 17). By 16th March 2007, it was 6.6 steps per respiration cycle, which represents a mere 30% improvement on the starting 5 steps per respiration cycle. My plan had long been to restart using CMO on Saturday 17th March 2007. This date was chosen on the principle that a pre-planned day would remove one possible distortion, namely a subconscious choice of a ‘right’ moment to resume the use of CMO. The day was chosen as being the Saturday occurring one year after I first used CMO. Before the day arrived, it was evident that I was beginning to wheeze slightly. I *surmised* that the sarcoidosis was returning, and that I might be applying the CMO just in time, *although I had an entirely open mind as to whether it was going to work once again*. It seemed too good to be true that it would repeat what it had done so dramatically at the start (see the first three weeks of Fig. 2a). As I remarked to my friends, if it does, then that would remove all possible doubt about the causal relationship between my sarcoidosis and the CMO treatment.

Thus it was a relief when 17th March arrived, and I could start applying Myristin Topical Cream (CMO) to my chest, three times a day. Incidentally one reason for changing from using the CMO cream from CMO Distribution Centers of America to that sold by EHP Products Inc. was that the former had suddenly vanished from cyberspace. That is a good illustration that the sale of CMO is often something of a scam (the range of prices is astonishing). Fortunately I have come to the conclusion that EHP Products Inc., available on the internet at www.cetylmyristoleate.com not only sell the best CMO, but also the best value products both as cream and softgel capsules.

But after the 17th March 2007 had passed, it soon became apparent that the wheeze was turning into a cough/cold that was typical of the virus infection that was going around locally. It would clearly be impossible to determine the effect of the CMO cream while I

was suffering from a virus infection, so after three days I stopped applying the CMO cream. It is necessary to mention this three day use of CMO in view of the remarkable improvement that occurred at the end of the virus infection, but it is, to say the least, uncertain whether the later improvement was due to this short period of applying CMO; nevertheless it is a fact which needs recording as a possibly relevant factor. What is almost certain is that the later rapid improvement, after the infection cleared, would not have occurred without the history of previous CMO treatment, as I will now explain.

During the time that sarcoidosis was virulent, that is for the couple of years before I started on CMO, there was nothing that I feared more than getting a cough, since on occasions the urge to cough became so overwhelming, and impossible to curb, that I could easily get to the state of being unable to pause long enough to draw in breath, which anyhow was sometimes very hard to do because of an apparent blockage in the throat. It may be helpful to dwell on the details of how my cough developed during the later stages of my sarcoidosis, although I suspect it takes different forms in different people. My GP once asked me, during a routine check up, whether I coughed *all the time*. I said no, the trouble only occurred once I had caught a cough, but then not only was the cough exceedingly alarming during the infection, but for many months it continued to be a problem to a lesser extent,. However, no sooner had I told him that, than I found that a cough started for no apparent reason and it continued for at least a year, *in fact it continued entirely unabated until I started on CMO*. So I reckoned that perhaps the doctor had known more than me about the usual course of sarcoidosis. Others can compare notes.

As mentioned, the second and most recent cough/cold infection started in March 2007. It was somewhat different from all previous upper respiratory tract infections that I have ever experienced. It seemed not so much an irresistible urge to cough, but a desire to cough in order to clear the mucus in the lungs which was forming in prodigious quantities. As far as I have been able to ascertain, other people *with no sarcoidosis complications*, suffering this particular viral infection, were experiencing rather the same slightly unusual cough symptoms. The cold symptoms were typical of a cold and don't require further comment. I did sometimes feel unusually tired during this period, but not in that particular way typical of my sarcoidosis. I have to say it did occur to me that perhaps the coughing up of so much mucus would prove to be leading to a clearing out of the lungs, and thus there would be an improvement once the infection was over. Nevertheless the degree of improvement certainly surprised me.

Figure 2c tells most of the story. The infection lasted approximately from 8th March to 20th April 2007. It was only during the last few days that the cough improved rapidly. Doing a 'proper' lung function test, by walking 1000 metres along the tow path by the river, had been impossible for most of this time, because any such attempt would be continuously interrupted by coughing, but on the basis of much shorter tests, e.g. walking in the car park, I could tell that my lung function was down to 5 steps again, or perhaps even less. On 20th April, as soon as the cough had gone away sufficiently to do a proper test and the weather was suitable, I found that, in confirmation of what I had rather been suspecting for the last few days, my lung function had jumped up. In fact, at the first test it was 9.5 steps per respiration cycle, a 90% improvement on the starting point of the CMO experiment.

Figure 2c shows that since 20th April, lung function has followed a somewhat erratic path, but that on the final check for this chart, 16th May 2007, it measured 9.9 steps per

respiration cycle, which is about a 100% improvement on the 5 that I could manage when the CMO experiment started. Up a one in twenty slope, the recorded 7.5 steps per respiration cycle represent a 120% improvement over the starting 3.4 steps per respiration cycle. Lung function is the easiest thing to measure, but I have never rated it as the worst aspect of my sarcoidosis, although in my case it is the only life-threatening aspect. Just because something is hard to measure does not mean that it is not dire! Thus we need to consider the other aspects of the disease as they fit into this experiment into the possible curing of sarcoidosis with CMO.

The broader aspects of the experiment

When I started the CMO experiment, partly in order to make sure it was a *prospective* experiment rather than a *retrospective* survey of what medical conditions happened to have improved, I set out various ailments that it seemed possible the CMO might affect. In the 4-week report that I made (both it and the 2-week report are available on the net²) I tried to estimate the proportions of the total medical problem that each ailment comprised in terms of damaging an acceptable quality of life. This is what I said (in square brackets are the current improvements, but related to *sarcoidosis* symptoms only,):

- 1) **10%** Stomach ache (troublesome only at nights).
- 2) **20%** Sarcoidosis and breathing. [**65%, see explanation below**]
- 3) **10%** Sarcoidosis and coughing. [**100%**]
- 4) **45%** Sarcoidosis and Totally Erratic Exhaustion Syndrome (TEES). [**100%**]
- 5) **10%** BPH (Benign Prostatic Hypertrophy, causing frequent nocturnal urination).
- 6) **3%** Sarcoidosis and dry eyes. [**50%**]
- 7) **2%** Right shoulder (discomfort in same on certain movements).

The sarcoidosis symptoms represent 78% of the total. Taking this assessment of importance into account, the overall improvement in the sarcoidosis symptoms can be assessed as $(20 \times 0.65) + 10 + 45 + (3 \times 0.50) = 69.5$, for an overall improvement in sarcoidosis symptoms of $69.5 / 78 = 89\%$, say 90%.

It is unfortunate that a further measure of lung function has to be introduced, but it has to be in order to integrate the “improvement” in lung function with the other aspects. Obviously the improvement in TEES and a persistent cough is only 100% if they go away completely, or in other words I return to being completely normal. The problem with lung function is that, like height, there is no such thing as normal. Thus it is a problem to make an estimation of “improvement” in the same sense as the other aspects of the illness. The best that can be done is to use the parameters of ‘normal’ already discussed, namely 11.7, on level ground, and 10.5 steps per respiration cycle up a one in twenty slope.

The 80% of normal previously referred to is a useful gauge, but is not actually a measure of improvement. What is needed is the degree to which the 9.9 steps closes the gap to a supposed “normal, from the starting value of 5. With a supposed ‘normal’ of 11.7, the improvement works out at $(9.9 - 5) / (11.7 - 5) = 73\%$. On the upslope, the calculation is $(7.5 - 3.4) / (10.5 - 3.4) = 58\%$. I suspect that the latter reading was unrealistically low, but anyhow the average is 65%. *For present purposes of integration*, therefore lung function has to be estimated as having improved by 65% towards a supposed ‘normal.’

Turning to the rest of the items, I quickly came to appreciate that the CMO was having no effect on item 1, the stomach ache. As a matter of interest, I seem to have substantially ameliorated that by frequently ingesting small amounts of biolive yogurt, but that experiment is ongoing. Item 5, BPH, has also improved which may or may not have a connection with CMO, but anyhow the problem is too erratic to make a useful test, and of course it is not associated with sarcoidosis. Items 3 and 4, coughing and TEES, which are both sarcoidosis problems, are completely solved, at least at present.

Item 7 is of course not associated with sarcoidosis, but is about 80% improved, and some weeks ago appeared 100% improved. As it had been a problem for some years, I do have confidence in ascribing the improvement to CMO. This is not surprising. Most testing of CMO relates to arthritis-type problems, not sarcoidosis. One friend of mine has had almost unbelievably good results from treating his widespread arthritis first with Myristin Topical Cream and then by taking one of their CMO softgel capsules per day (now no longer necessary). Another reason for my confidence that CMO did the trick is that while I was applying the CMO (I have not done so for many months now) I found that the discomfort kept moving away from wherever I was applying the CMO. I had, so to speak, to chase it around from the base of the neck to various places on the shoulder and upper arm. Item 6, the dry eye problem, requires a rather lengthy description, which is worthwhile as the experience raises issues that are generally relevant to CMO, and indeed relevant to another treatment for sarcoidosis, the Marshall Protocol.

The dry eye problem

The dry eye problem first arose nearly thirty years ago, the time at which I have come to think my sarcoidosis started. When I went to see an ophthalmologist, he told me that this was a not unknown accompaniment of a flu-type virus infection, which is what I seemed to have experienced, albeit in a somewhat mild form. He said that there was only a palliative treatment, namely to use eye drops. After a couple of years the problem went away, but only to return in exacerbated form during recent years; I was having to apply about 10 eye drops during the course of the night.

After eight weeks had elapsed from the start of the CMO treatment, I was estimating a 60% improvement in the dry eye problem. Then the assessment was confused by a somewhat alarming reaction which was probably an example of something called the Jarisch-Herxheimer reaction, or Herx reaction for short. Such reactions are a major feature of the Marshall Protocol (a method of treating sarcoidosis with tetracyclines that is currently being tested), and incidentally they occur in one in seven Lyme disease patients. A Herx reaction might be defined as the appearance of undesirable symptoms which are temporary side-effects of the curative process. Incidentally there appears to be a wish to change the name to immunopathological response, or IPR, but then doctors like to have lots of different names for something they don't understand! I will stick to calling them Herx reactions or Herxes.

Herxes, as reported by people following the Marshall Protocol, often seem to be very dramatic, sometimes almost unbearably so. Herx reactions have also been reported by those using CMO to treat arthritis, but nothing particularly dramatic: that is to say some patients report that their problem gets worse for a while under CMO treatment, rather than better. Of course rheumatoid arthritis is so variable that any inter-relation is somewhat indeterminate. If, for example, the CMO is in fact having no effect, the rheumatoid arthritis may still be getting worse of its own accord. The general advice nevertheless is to

stop using CMO for a while, and to take the Herx as a good sign that the CMO is having some effect, then resume treatment after the bad period is at an end. It should be stressed that this Herx reaction is far from universal. Most people, probably about a third of *rheumatoid arthritis* patients, have no reaction to CMO, while another third fairly quickly show a marked improvement, in both cases mainly without any Herx reactions.

But back to my own herx, if that is what it was. The effect on my left eye, which has always been the worst one for the dry eye problem, was dramatic. The eyelids of both eyes became itchy and slightly pink on the rims, and then, for almost a couple of weeks, it looked as though I had been boxed in the left eye, but with the surrounding area becoming pink rather than black and blue. The eye itself was not at all involved. I had been applying CMO cream to within an inch or so of the eye, and when the Herx occurred there was much discussion as to whether this was a Herx reaction or an allergic reaction. I was open minded, and only felt fairly sure it was a Herx six weeks later, when the eyes looked entirely normal again, and there had been a very significant improvement in the dry eye problem, so much so that I was soon able to rate it as a 90% improvement. A further welcome improvement was noted. A problem of variable lack of clarity in the left eye, accompanied by slight signs of viscous fluid at the lower eye lid, disappeared. Moreover it seemed to be a fairly useful confirmation of a Herx reaction that when I was able to apply the CMO as before, that is on the face, it produced no adverse reaction.

So it can be said that the outcome of the first 'Herx' was favourable, and the final results, when looked at in retrospect, confirmed that the reaction was a Herx. Unfortunately nothing about the human body is simple! The improvement gradually wore off, with eye improvement dropping to only about 70%,. Still very worthwhile, but in an attempt to get further improvement I used rather more CMO cream on the face than I had ever used before (though no nearer than a couple of cms from the eye). Then on 15 February 2007, almost the day after I had applied rather more CMO cream than usual, a second Herx type reaction started. It was slightly different from the first in that considerable eye weeping occurred at times. The boxed-in-the-eye appearance of the left eye was somewhat less marked than on the previous Herx. That appearance was almost gone after a couple of weeks. The disappointing aspect was that I was unable to see any definite improvement in the dryness after the incident was over. In fact I have recently been rating the dry eyes improvement at 50%.

So does this second experience prove that it was not a Herx? The situation at present is clouded, with the following variables which might be relevant:

- a) The type of CMO cream, Myristin Topical Cream, was different from the one used before and has many ingredients. This reaction may have been an allergic one even if that was not the case previously.
- b) Applying CMO extracted from Myristin capsules directly to my face still does not cause any reaction.
- c) While the dry eye problem has not fully resolved, the viscous fluid showing in the left eye has not returned.

Improvement assessments related to eye dryness are made on the basis of the number of Hypromellose eye drops that are needed compared to before I started on CMO. Dry eyes were never more than a small part of the problem, but the detailed account above is warranted because of the general association of Herx reactions with some sarcoidosis treatments, and because anything closely associated with the eyes is bound to be a cause

for concern, and prospective patients need to be informed. When mine first occurred I went to a pharmacist, but she, like most doctors in fact, had never heard of CMO.

So that is the record of my sarcoidosis experiment to date, but perhaps I should remind readers that my sarcoidosis was not proved by biopsy, so it might be cautious to call my illness an undiagnosed disease that exhibited several of the most common symptoms of chronic sarcoidosis: (a) erratic and extreme fatigue, (b) persistent dry cough, (d) dry eyes, and (e) lung function deteriorating to a point at which it was hard to walk and talk at the same time, even on level ground, making the use of a cortico-steroid inevitable were I not to have discovered first Voltarol Emulgel P (diclofenac), which had a temporary palliative effect but one which gradually diminished, and then, more importantly, cetylmristoleate (the active constituent of CMO). It is important to note, too, that the qualification of "chronic" in my case is beyond reasonable doubt. I *suspect* that the sarcoidosis started about thirty years ago, but I am *fairly certain* that it was active 24 years ago, since the main reason that I retired early from flying, in 1983 at the age of 46, was because of the erratic feeling of being unwell, which grew to much greater prominence in the later years of rampant sarcoidosis, but has now disappeared.

Conclusion

Reviewing the list of medical problems listed earlier, it is apparent that the ones associated with sarcoidosis represented 78% of the total medical problem as assessed from a subjective viewpoint, which is the only type of assessment that can be made for such a variety of symptoms. On the other hand, actual improvement can be assessed with some objectivity. On one basis, lung function can be assessed as having improved 110%, or alternatively it could be said the lung function at the start was 40% of what is probably 'normal', but by the end of the 61st week it was 80% of 'normal'.

But lung function was not the most trying aspect of the sarcoidosis symptoms, and putting all aspects together (which requires an assessment of lung function as having moved 65% towards 'normality') an overall improvement in sarcoidosis symptoms, weighed for their subjective importance, results in an estimate of a 90% improvement. Constituting this 90% improvement was 100% improvement in TEES and the persistent cough, and 50% improvement in the dry eye problem.

So in my case, CMO has been an outstanding success *as judged at this time*, 61 weeks after the experiment started. But there were quite a lot of ups and downs in lung function that do not have confirmed explanations, so what happens next remains to be seen. I have learnt not to expect definite conclusions in a hurry! I now need further candidates whose symptoms are closely similar to mine, and who can learn the technique of counting their steps per respiration cycle, since taken alone my success can only be taken as an indication that further experimentation is needed. I have already located two candidates who started to experiment around the end of my 61st week. Both are making progress. Perhaps I should add that the need for further experimentation has been evident for a long time because of the anecdotal evidence of CMO being effective in treating sarcoidosis. Anyhow I will be pleased to discuss symptoms with potential candidates either by phone, (01491) 574850, or by email AndrewRBFerguson@hotmail.com, and I don't mind being rung any time up to 1015 pm. It is important that the candidates definitely have chronic pulmonary sarcoidosis and preferably most of my symptoms, and should not have started on cortico-steroids.

Endnotes

1. The great virtue of a steps per respiration cycle test is that it tests both the vital capacity of the lungs (something which is usually tested clinically by spirometry) and the oxygen exchange capability, which is at least as important, but is often not tested clinically. The immediate objection that arises in the minds of most people is that one could 'cheat', for instance by taking shorter steps. This is not so. In fact if one cannot manage to take say 10 normal length strides during a respiration cycle, the only way to continue to do so is to shorten one's step. But the shorter step inevitably slows the speed of walking. And if the time over the 1000 metres is also taken, the assessment of steps per respiration can combine the actual number of steps, say 9 or 10, with the time, so as to produce the value in steps per respiration cycle to the nearest decimal point. The validity of this time-step relation is checked by walking both at 9 and at 10 steps on the same day, and finding that after correction for the time difference, the number of steps works out the same, say 9.6 steps per respiration cycle.
2. The folder on the web that contains my Voltarol and CMO records is at the following address:
www.members.aol.com/CMandCM

Introductory notes to Figures 1, 2a, 2b, and 2c.

Figure 1. The major part of the curve shows a period of 41 weeks before starting using CMO, during which an anti-inflammatory, Voltarol Emugel P (diclofenac) was used. The Voltarol experiment started about five months before this chart. As is evident from the three curves shown, the substance gradually became less effective. Neither did it have any effect on the TEES or the persistent dry cough. The last 11 weeks shown cover the start of treatment with CMO, but the same is better displayed at a larger scale in Figure 2a.

Figure 2a. The most notable features are (a) the rapid improvement, over two weeks, when treatment with CMO cream started, (b) the rapid drop over the next week with no treatment and (c) the even more rapid recovery within two days under treatment with CMO capsules. Continued use of CMO on various parts of the body was not ideal as far as the experiment was concerned, but it was necessary to keep my hip in sufficiently good order to carry out the steps per respiration cycle test. The CMO seemed to help there, and since I was using it anyhow, I thought I might also test it on the shoulder. Another notable effect is that later improvements, i.e. after the early weeks, seemed to take time.

Figure 2b. The most puzzling feature of this 20 week period is that the treatment with CMO cream, starting on 20 October 2006, resulted only in a *drop* from 8.9 steps per respiration cycle to 7.9. Whether there would have been an improvement shortly thereafter if a cough/cold were not to have intervened remains uncertain. It could be the case, as there is no evident reason why lung function improved at the end of the period, finishing at 9.8 steps per respiration cycle. What can be said with some certainty is that the recovery from the cough/cold outcome was quite different from what it would have been at an earlier period, when the sarcoidosis was unchecked.

Figure 2c. The slow decline over the first 12 weeks could be explained in terms of there being no CMO treatment. Unfortunately the definitive confirmation of this was thwarted by the fact that after I had applied Myristin Topical Cream (CMO) for three days it was so evident that another cough/cold virus was going to interrupt the experiment that treatment was discontinued. Whether these three days of treatment had a delayed good effect must be speculative, but once again it can be said that what is certain is that the rapid recovery after the cough/cold would be highly unlikely when sarcoidosis was still unchecked.

Figure 1. 41 weeks using Voltarol gel, then 11 weeks using CMO.

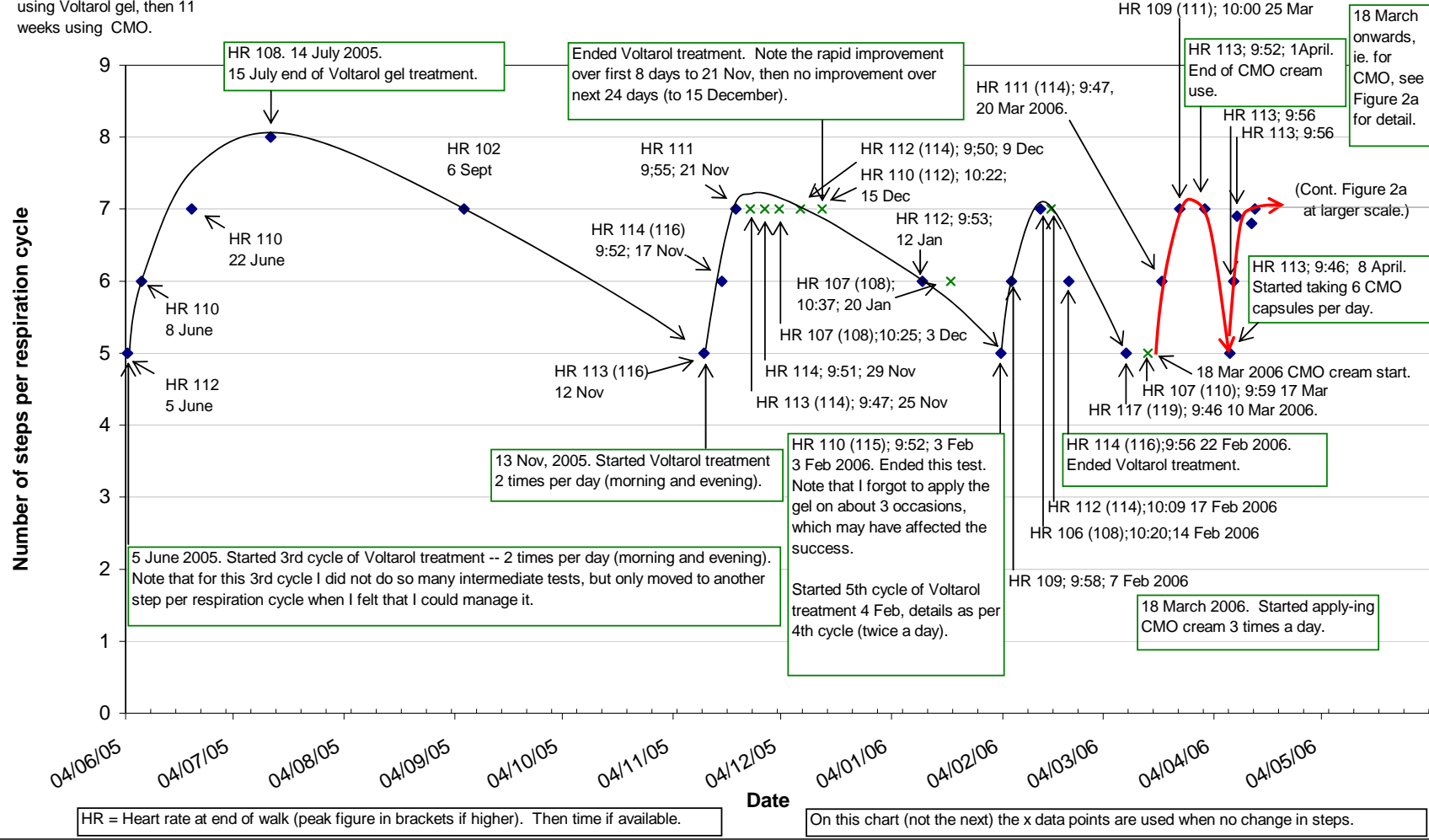


Figure 2a. CMO
weeks 1 to 20

Andrew Ferguson's CMO experiment for relieving sarcoidosis

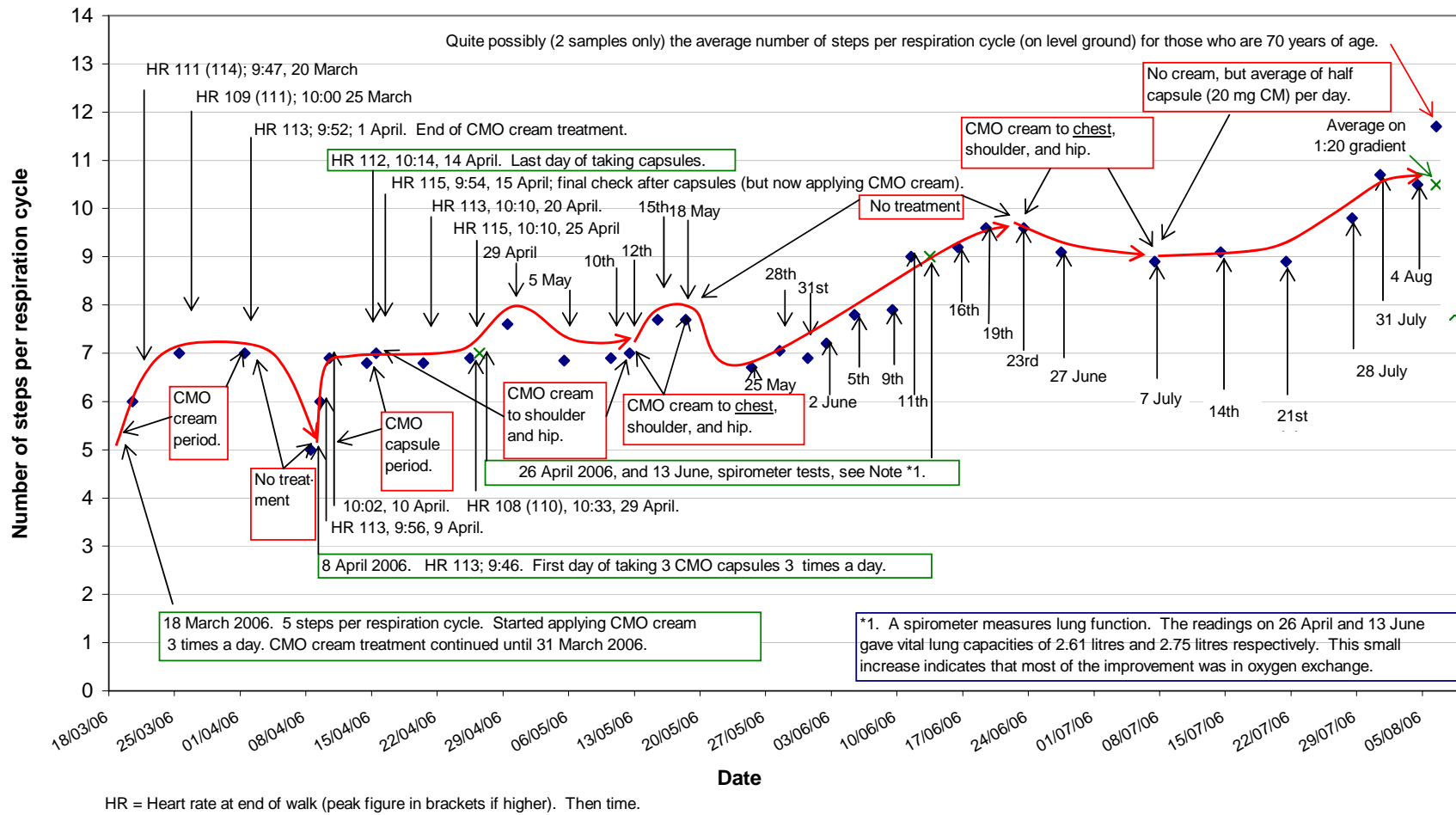


Figure 2b About the 21st to 40th week incl.

Andrew Ferguson's CMO experiment for relieving sarcoidosis

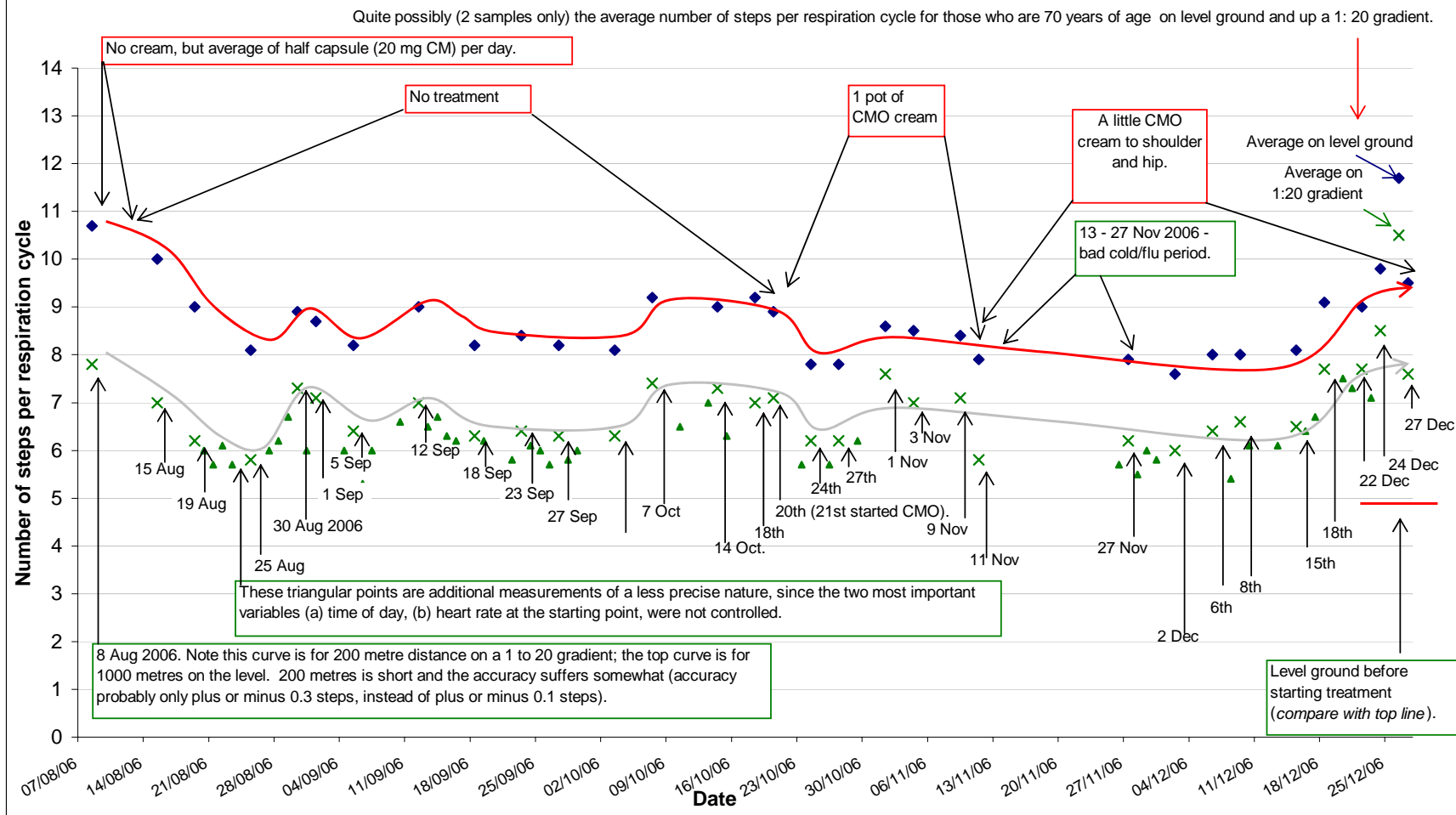
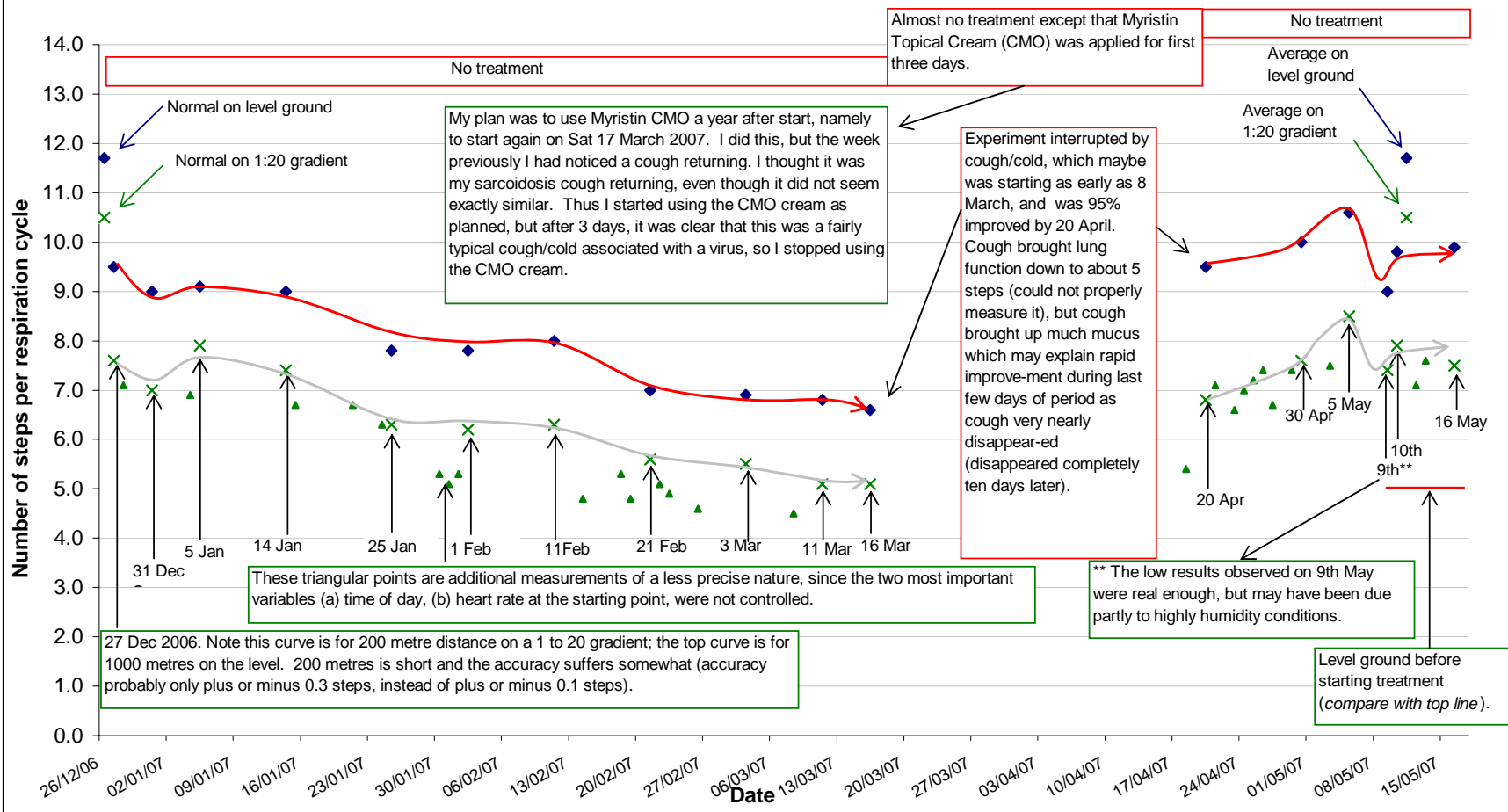


Figure 2c. About the 41st to the 61st week inclusive.

Andrew Ferguson's CMO experiment for relieving sarcoidosis

Quite possibly (2 samples only) the average number of steps per respiration cycle for those who are 70 years of age on level ground and up a 1:20 gradient.



WHY THE MEDICS ARE MISTAKEN ABOUT SMALL SCALE TRIALS

by Andrew R.B. Ferguson

Abstract Doctors and many concerned with medical matters tend to think that useful things can be learnt only from trials with a large number of patients, which are placebo controlled and double-blinded. It is shown here that that is only a generalization, and that in particular cases, chronic sarcoidosis being the chosen example, useful confidence can be placed in results from a sample of three patients, and it is possible to calculate the reduction in the range of uncertainty if the sample is increased to say twelve.

Suppose that you suffer from chronic sarcoidosis, and having read anecdotal reports about a food supplement that is said to be very effective at curing sarcoidosis, you try it. Suppose further that you respond to it rapidly, and within a year you can again breathe freely and it has largely cured your medical problems. Finally suppose that you happen to know two other people with similar sarcoidosis symptoms. You persuade both of them to try the substance. Let us suppose that one has similar success to you, but the other experiences no improvement. That is two successes out of three. I feel sure that you, the reader, especially if you suffer from chronic sarcoidosis, would feel that to be significant. However were you to ask most doctors, or anyone connected with the pharmaceutical industry, or even anyone that is somewhat versed in medical lore — all of whom we will group together under the term “medics” — they would respond, almost to a man, that such a result was not significant, because there were too few people in the trial, and anyhow trials need to be placebo controlled and double-blinded, the latter meaning that until after the results have been ‘scientifically’ assessed, neither the patient nor the doctor knows whether the mooted curative substance has been used, or rather the harmless placebo. Who would be right? Would your gut feeling about the matter be right, or would the doubting medics be right? The simple answer is that *you* would be right. But before proceeding to see why, let us investigate why the medics think as they do.

Suppose that it is desired to find out if the new drug Floop is better than the old drug Bloop, at preventing heart attacks. Not everyone suffers from heart attacks, so the number in the trial has to be sufficient that a difference will show up if Floop is better, so in this case the medics would be right in thinking that the number of people required is large.

Why is a placebo required? Surely, you may think, experience would have already taught the medics how effective a placebo is when used to treat any medical problem about which as much is known as is known about heart disease. The trouble is that when the last medical test of this kind was done, people may have been doing different things that are relevant to keeping a healthy heart, e.g. eating less or more fat. Thus a placebo *at the time of the latest trial* is essential, to check the success rate when people just *think* they are being treated. The results may be better this time than last, not simply because of the placebo effect behaving differently, but because fewer people are having heart problems.

The need for the patient not to know whether they are getting the placebo or Floop is obvious — placebos lose some or all of their power if you know you are getting a placebo. Not so obvious is that the doctors who assess the results should not know. But when there is a marginal case involving a very mild possible heart attack, the doctor may be subconsciously influenced in the task of classification if he or she is aware of whether Floop or the placebo has been used.

So we can readily concede that on every point the medics are right about that sort of trial. So what is different in the small scale trial outlined at the start? The first point is that once sarcoidosis becomes chronic, it is so rare for it to cure itself that the remote possibility of that occurring can be ignored. For a rather similar reason it can be said that the chance of placebos working can be ignored. Often chronic sarcoidosis patients have tried several placebos, and sometimes they start with great optimism for good results, yet no cases of placebos being successful in chronic sarcoidosis have been reported. There is confirmation of this because steroid treatment has serious side effects, so doctors would not inflict prednisone on their patients if they thought that a placebo might be worth a try. Thus in the case of chronic sarcoidosis the placebo effect can be ignored.

How about the argument for double-blinding? Since placebos don't work in the case of chronic sarcoidosis, the patient does not need to be blinded, and because the "cure" we are talking about is not just a marginal improvement but a virtual cure, which would not have happened without intervention of some kind, the doctor is not even needed to assess whether there has been a cure. The patient will readily do that free of charge!

It becomes apparent that the medics fall into the logical error of assuming that since placebo, double-blinded trials are essential in most cases, this must be true of all cases.

We are left with this question: does a sample of only three allow some sort of generalization? And how large a sample is needed to be statistically significant? Once again that depends on the case. Anyone experiencing the situation outlined at the start will have a feeling in their bones (unless they are a dyed-in-the-wool medic) that this result must be important. But how important is it in statistical terms, and how much more certain could one be about the statistical chances if there were more people involved in the trial?

The first thing to be taken into account, in this special case of chronic sarcoidosis that we are considering, is that the food supplement used does not have deleterious consequences, so we are not looking for a particularly high success rate. Since the only treatment that the doctors can offer is the almost inevitably harmful palliative of cortico-steroids, then if the food supplement has only a one in ten chance of being successful, then — especially since it is not expensive — almost everyone would agree that it is worth trying before starting on prednisone. So do the results give us assurance that there is a one in ten chance of success?

When the experimenter found that the anecdotal stories were confirmed by effecting a cure in his own case, there are essentially two possible reactions. One would be to consider that this was a miracle, and as such it would happen to no one else, the other would be to try to ascertain whether there were others who responded similarly. Let us assume that the second option is adopted. On deciding, as just discussed, that a one in ten success rate would be acceptable, the aim should be to study at least ten cases. However, let us consider the probability situation if successes show up early on during this process.

The generally accepted threshold of "significance" is the point at which what has occurred cannot be explained in terms of a one in twenty chance occurrence, that is to say there needs to be a 5% possibility, or less, that the result is a matter of chance. With that in mind, let us then move on to consider how the statistics look if we have two successes out of three subjects tested.

We must remember that the question we are asking is does this substance have a one in ten chance of curing chronic sarcoidosis. If it does, then by chance we could expect two cures in three in only about 1 in 40 trials (see postscript for this and later probability calculations). Thus two successes in three is a "significant" result in terms of the objectives we have in mind. Indeed we could refine our hypothesis that the substance was

worth trying if it cured one in ten cases, to one in eight cases. The reason for the reduction is that if it is assumed to cure one in eight cases the observed result should only occur by chance 1 time in 24 trials, so at a one in eight chance of cure, the results still pass the test of “significance.”

So far so good, but as noted the experiment should have set out to test at least ten subjects. It will be convenient for the maths to consider that we decide to test twelve, and to assume further that there are four cures out of the twelve subjects. Once again I have no doubt that sarcoidosis sufferers would say, Wow!, to that result, but what would that result say statistically? Let us again assume that we would be satisfied were the substance to have a one in ten chance of being successful. On that assumption, the chance of getting four successes out of twelve is about 1 in 50. So once again the test has easily jumped over the hurdle of significance in the terms we have set. Indeed the case is remarkably similar to that with only three subjects, for if we refine our hypothesis and now assume that the substance cures one in eight people, the chance of the four successes is again 1 in 24.

Although the four successes in this larger trial has the same chance of occurring as the two in the smaller trial, the gut feeling is bound to be that the larger trial has made the case more securely. Once again a gut feeling is a good guidance. But can we put numbers on that justifiable sense of increased security?

We can best see the answer by considering the fact that the results which have occurred could have been achieved by a *really* amazing piece of luck. So let us ask, taking first the case of three subjects, could the two successes have happened if the cure rate of the substance was only one in twenty, rather than one in eight? It could, but the chances against that are 140 to 1.

Now let us apply the same reasoning to the case of twelve subjects. In this case the four successes could possibly have occurred if the cure rate of the substance was only one in twenty, but this time the chances against are about 490 to 1. So it is clear that increasing the number in the trial has narrowed the range of likely answers. In this second trial of twelve subjects, if we assume that the cure rate is *one in fourteen*, the odds against getting four successes are 140 to 1, exactly the same in the first case as occurred when assuming a cure rate of *one in twenty*.

To sum up in colloquial terms, after the three person test, you pretty well know that there is at least a one in eight chance that the substance will cure you, and you are pretty *dead* certain that there is at least a *one in twenty* chance it will. After the twelve person test, you again pretty well know that there is a one in eight chance that the substance will cure you, but this time you are pretty *dead* certain that there is a *one in fourteen* chance of it doing so. I think most chronic sarcoidosis patients would be prepared to put a tenner on at such odds, when a “cure” was the reward for the lucky ones; especially since once the disease becomes chronic it is fatal in about one in five cases, despite medical palliatives.

An actual trial of cutaneous sarcoidosis revisited.

We can now look afresh at a nonrandomized, open study performed in patients with cutaneous sarcoidosis, by Bachelez et al. reported in 2001, see Appendix A. It involved only twelve patients. All the medics that I have discussed it with have pointed out that it was not placebo controlled, and that anyhow twelve is too small a sample to be statistically significant, so let us look at that again, following our previous type of analysis.

I have to say that I am less knowledgeable about cutaneous sarcoidosis than chronic pulmonary (of the lungs) sarcoidosis, and I have only been able to read the Abstract. There was no mention therein that care was taken to ensure that these were all patients who had

no chance of a spontaneous cure. One could reasonably assume that this possibility would have been mentioned if that were the case, but nevertheless let us build in a margin and assume that one of the patients underwent a spontaneous cure.

The Abstract tells us that, “A clinical response was observed in 10 patients, consisting of complete responses in 8 patients and partial responses in 2 patients.” We don’t know how partial were the partial responses, so let us just accept the 8 *complete* responses as worthy of analysis, and subtract one, on the basis of one spontaneous cure occurring. Now let us hypothesize that the cure rate is one in three; what are the chances of nevertheless getting the 7 complete responses by chance? The odds are 1 in 21, thus we can accept that this experiment shows that, with some certainty, minocycline and doxycycline treatment, when continued over a year as per the experiment, has at least a one in three chance of being successful. If we want to see the degree of reliability of that result, we can consider the hypothesis of a 1 in 4 cure rate. In that case, getting 7 complete responses would be a chance of 1 in 90. Thus we can be just about dead certain that the cure rate is better than 25%, i.e. better than one in four.

Do doctors appreciate the importance of this small scale trial? It would seem not, because *chronic* pulmonary sarcoidosis currently has no *curative* treatment, and doubtless sarcoidosis specialists meet at least a dozen chronic pulmonary sarcoidosis patients in the course of a year (lung involvement occurs in 90% of sarcoidosis cases), and surely they would have carried out similar trials with a dozen of their patients were they not put off by their mistaken belief that secure knowledge is gathered only by immensely expensive, large-scale, placebo controlled, double-blinded trials. Yet no such small scale trials appear to have been made, at least to the knowledge of the sarcoidosis community.

So what is the message that this gives to the medics? Perhaps doctors would like to be able to assert that they had never fallen into logical error, but discovering their mistake does have a silver lining. It means that doctors can think for themselves, observe their patients, and learn from what they see, instead of just regurgitating what they have read in the BMJ, JAMA, and pharmaceutical journals. It certainly has a silver lining for sarcoidosis patients, insofar as we could wait an awful long time before pharmaceutical companies decide it is worth spending half a million pounds to carry out one of their placebo controlled double-blinded trials on a disease which has such a low incidence and prevalence as sarcoidosis.

Postscript

But are all those statistical chances correct? They are based on using a mathematical formula which a friend was kind enough to locate in his mathematical books and expound to me. However, before I learnt about it, I wrote a simple Basic ‘dice throwing’ program, which allowed the computer to get the results by experiment. It was a slow process, but ‘throwing the dice’ 6000 times — or 120,000 for very long odds — served to prove to me that the formula does produce the correct results.

Should you want to develop the ideas in this article yourself, here is the formula explained in terms of throwing dice. You don’t have to think too hard to adapt it to testing patients (or if you’d like save yourself the cogitation, just ask me to send you the very small Excel file, which due to its origins I call *Diecast*).

The probability of throwing 5 sixes, *in any order*, out of 12 throws is
 $12! / [(12-5)! \times 5!] \times (1/6)^5 \times (5/6)^7 = 792 \times 0.0001286 \times 0.27908 = \underline{0.028}$

5! = 5 factorial = 5 x 4 x 3 x 2 x 1, which is written as FACT(5) in Excel code.

N.B. the 5 in (5/6) refers to the value (6-1) not the 5 sixes that are thrown.

Appendix A (The Abstract relating to a cutaneous sarcoidosis trial)

Authors. Bachelez, Herve MD, PhD, Senet, Patricia MD; Cadranel, Jacques MD, Kaoukhov, Alexandre MD; Dubertret, Louis MD.

Title. The use of Tetracyclines for the Treatment of Sarcoidosis

Source. Archives of Dermatology. 137(1): 69-73 January 2001

Background: To evaluate the safety and efficacy of minocycline in the treatment of sarcoidosis, a nonrandomized, open study performed in patients with cutaneous sarcoidosis.

Observations: 12 patients with cutaneous sarcoidosis were treated with minocycline, 200 mg/d, for a median duration of 12 months. Three patients had extracutaneous lesions at the time of the study. The median follow-up was 26 months. A clinical response was observed in 10 patients, consisting of complete responses in 8 patients and partial responses in 2 patients. A progression of skin lesions was observed in 1 patient, and lesions remained stable in another patient. Adverse effects were minimal, except in 1 patient, who developed hypersensitivity syndrome. A slight hyperpigmentation occurred in 2 patients at the site of previous lesions, which completely disappeared after minocycline use was discontinued. A relapse of skin symptoms occurred after minocycline withdrawal in 3 patients, who further received doxycycline, 200 mg/d, allowing a complete remission of lesions.

Conclusions: These results support that minocycline and doxycycline may be beneficial for the treatment of cutaneous sarcoidosis.

* * * * *

In the above, reference was made to the dire effects of cortico-steroids. The trouble is that prednisone, or prednisolone, have to be used in fairly high doses. Dr Gary Epler (see www.epler.com/wsarc.html) says, "Current treatment protocols indicate the use of 30 to 40 mg of prednisone daily for 8 to 12 weeks, with gradual decreasing of the dose to 10 to 20 mg every other day over a period of 6 to 12 months." Certainly 30 to 40 mg daily is a high dose according to the following, which was passed to me by someone with a medical background who suffers from polymyalgia rheumatica. Polymyalgia rheumatica is not classified as an auto immune disease in the medical books, but the closely associated condition of temporal arteritis is. Moreover we are concerned here with the side effects of cortico-steroids, not their beneficial effects. I suggest only that what the writer says provides some general background on prednisolone, which was the cortico-steroid being taken, and by implication prednisone, as I have never heard it suggested that there is much of a difference.

Steroid problems are highly dose related. The problems are real with doses above about 30 mg daily. Below 5 mg/day, I think one can say there is very little problem, although naturally one wants to get off all drugs if possible. After three years of taking 5-15 mg daily (for polymyalgia rheumatica), I got down to 1 mg daily and finally stopped altogether (tapering off over a period of about 2 months). I then gradually started to feel an old man, with no energy. I was advised to go back onto 1 mg/day and immediately felt many years younger and regained my lost energy. I am now down to 0.75 mg. I think that my adrenals gave up steroid production since I was taking steroids from the chemists so why should they bother! I am trying to persuade them to get back into production.

The SILA website, www.sila.org.uk, expands with a Social Network

SILA's website is proud to announce that it has a new feature – a social network! Now for those of you unfamiliar with this, a social network is a place where people can have discussions, meet each other, swap notes and generally interact online. The majority of the discussions are public which means everyone benefits, and if someone has some knowledge they can jump in. The hope is that by interacting in this way, by spreading knowledge in this way, we can help many people. The website offers you the opportunity to help people by you sharing your knowledge of sarcoidosis and your experiences with it. Right now we have discussions on:

- Methotrexate
- Getting the Disability Living Allowance
- Identifying other organ involvement
- Cetyl Myristoleate (CMO)
- Steroid Treatment

We're working hard to publicize the site. We're doing that because the more people who contribute the better the site is, and the more people who read it the more we help. To get the word out we have a link from the [sil.org.uk](http://www.sila.org.uk) website. We're paying for Google adwords (that means whenever you Google Sarcoidosis our advert comes up on the left. The adwords are a donation from me), and we're using word of mouth. The site is (touch wood) so far very successful with a good group of members and growing all the time. We get roughly 5 new members a day with members coming from all around the world. At the time of writing we've had over four thousand page views. That means we've had over four thousand pages read. **We'd love it if you would join too!**

How to join the site

Go to [sil.org.uk](http://www.sila.org.uk) and click the discussions link.

To see the site you're going to need to create a username and password. Click "Join Sarcoidosis Now" and follow the instructions.

To post just click the "Start discussion" or "reply" depending on if you want a new topic or to respond to someone else.

Lastly, if you have any issues please just email me: henry@shelford.net

I look forward to seeing you there !

Henry Shelford.

Henry Shelford is the SILA webmaster, and has his own interesting Life Story available on the SILA website. It was solely his idea to start up this social network. It certainly pleases me, as it may help me get more suitable candidates for my cetyl myristoleate (CMO) trials. There is one snag. I am not on broadband, and only make occasional forays to the library to use the internet. Thus I appeal to those of you who are internet savvy, and can spend time monitoring the Social Network to keep an eye out for suitable candidates.

Suitable candidates form a small subgroup of sarcoidosis sufferers. The main reason is that about 80% of people recover from sarcoidosis spontaneously, so although anyone may try cetyl myristoleate, and might receive benefit, even a total cure would not prove anything. To prove anything the sarcoidosis needs to be chronic (at least 5 years).

Also, if the results are to influence the medical profession at all, the primary problem needs to be deterioration of lung function. The reason is that sceptics will take little notice of patients merely feeling better, or having overcome their cough. It is necessary to demonstrate that some measurable quantity has changed in a way that could not be expected without treatment. For that, lung function is ideal. Thus my appeal is simple. Keep an eye out for suitable candidates, and tell them about my trial. Andrew Ferguson.

For all **Correspondence** which includes **leaflet requests**, please enclose a large self-addressed envelope together with four separate “large letter” first-class stamps to:

The Secretary, SILA
c/o The Chest Clinic Office
Kings College Hospital
Denmark Hill
London SE5 9RS



Support Meetings are held at King's College Hospital on the first Thursday of each month with the exception of August when there is no meeting. Enquire at the KCH Help Desk for the location of the meeting. Meetings are held between 7 pm and 9 pm. A map with details of how to reach KCH is on SILA's web-site.

SILA West Midlands Branch This branch is run by SILA member Mrs. Carol Bashford, 38 Yew Croft Avenue, Harborne, Birmingham B17 9TR. Contact Carol: 0121 427 5462 or email carol_bashford@hotmail.com for information on future support meetings, help and information.

The Irish Sarcoidosis Support Group ISARC is at www.isarc.ie email info@sarc.ie Mary Walters is Chair of ISARC, telephone number 1903 87216. The Marian Geraghty Sarcoidosis Trust Fund 10k Run/Walk is an annual event.

Information received: Travel Insurance For People Living With Pre-Existing Medical Conditions

Freedom Insurance Services Ltd.,
Richmond House
16-20 Regent Street,
Cambridge CB2 1DB

Telephone 0870 774 3760 Monday to Friday 8.30 am - 5.30pm Saturday 9.00 am - 12 noon. FAX 01233 720 277

www.freedominsure.co.uk

Electronic version of the SILA newsletter: The newsletter is prepared in Word. If anyone would like to receive an electronic copy, please contact the assistant editor directly, AndrewRBFerguson@hotmail.com. If you are sufficiently happy with the electronic form, then you may decide to forsake the hardcopy altogether, but it will not be withdrawn without your say so.

Our web address. A SILA member drew our attention to the fact that the old SILA web address, www.sarcoidosis.org.uk – which is no longer owned by SILA – is now pushing a variety of products which have nothing to do with SILA. In fact the address is offered for sale, but we don't want to buy it back. Instead we have tried to contact all websites linking to our old address. Please let us know if you find any we have missed. SILA's sole web site is now www.sila.org.uk, and is paid for and managed for SILA by Henry Shelford.

Annual Subscription to SILA is still £12 per annum. SILA welcomes comments and contributions to the SILA newsletter; also fundraising ideas or initiatives.

Our website can be found at www.sila.org.uk; the email address is heather@sil.org.uk

