Price £ 3.75 (Free to members)

Pathways

The newsletter of Leger ME/CFS Supporting Myalgic Encephalopathy or Encephalomyelitis (ME), Chronic Fatigue Syndrome (CFS), Post Viral Fatigue Syndrome (PVFS), Fibromyalgia Syndrome (FMS), Patients & Carers.

Welcome to Pathways 40.

In the last few years it has become increasingly obvious to me that there is a major issue that is coming into focus and that is ME/CFS & Cardiovascular (CV Heart disease) issues. We don't seem to hear much about ME/CFS & heart disease, yet there must be a significant threat to wellbeing. After all the heart is basically a resilient bag of muscle which is capable of fatiguing just like any other muscle. We don't hear about death related to ME/CFS and heart disease or do we? I'm not so sure.

In my local doctors' surgery, I was reading a magazine which promoted Coenzyme Q10 as a food supplement. The feature provided an interesting graph showing that after the age of 20 the amount of Coenzyme Q10 within the heart drops dramatically. So I checked on a manufacturer's website. From that I picked up on a research paper about ME/CFS, statins & cholesterol. Statins are a group of drugs given to protect people from heart attacks and strokes due to arthrosclerosis. All the non-ME/CFS medical evidence suggests that they are very beneficial for almost all, apart from a very small number of people with serious side effects. If you speak to any NHS doctor or consultant they are an obligatory prescription for anyone with even the slightest history of CV problems. Yet we have a research paper implying simply that taking a statin with ME/CFS could reduce your life expectancy by 25 years. Then the answer comes—if you have ME/CFS and take a statin for whatever reason you should also be taking a Q10 supplement. So is this true? Is it a nasty scary sales gimmick to sell Q10 supplements?

The major issues I come across on the Leger helpline are related to DWP and Benefit matters. Next are the issues with statins. So from first-hand experience I can say that statins are a significant problem with about 30% of the Leger ME members, but also there are some who can take them without apparent side effects. I think here we have to acknowledge that there are different subtypes of ME/CFS, and it may only be that only a certain subtype is affected. But just how can you quantify this? Without proper research you can't. So for this edition we decided to focus on ME/CFS, Statins and Q10. First we list a *You Write* which members have sent in about Statins. We then move to the artherosclerosis problem, cholesterol, blood tests, and risks. We then print a research extract, followed by Dr. Myhill's views and we quote a warning from the Lancet. We follow with a stocktake of the ME/CFS-related issues. We then list the British Dietetic Association Factsheet on a dietary cholesterol-reduction strategy. Finally we list the Pharma Nord information sheet on the topic.

Points to remember are:

- If you have a major health problem where there is a cholesterol-related issue, keep taking your medicines as prescribed, but by all means ask your doctor searching questions.
- If you are without symptoms and/or just have high cholesterol, just remember that you need to get the level down to reduce your risks. There are at least half a dozen alternative medicines to statins, some of which are very ME/CFS friendly.
- We understand a leading heart hospital in the North East prescribes Q10 along with statins.
- You can get a private blood test for Q10 levels at a reasonable cost.

This *Pathways* is not all health issues of course. We include a selection of the usual features in the following pages, so please read on.

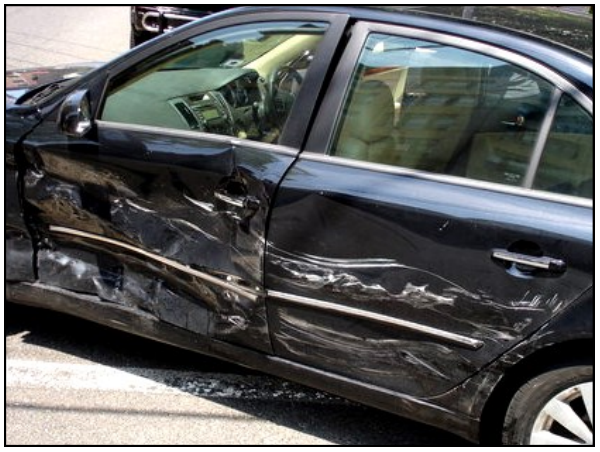
Don't listen to the doubters!

(Or don't let someone prepared to hit and run, get away with it.)

It happened to me in March. There I was 'blissfully sitting in the dental chair', (well, it felt like that in retrospect), while my brand new *3 week* old car parked outside was being beaten up by someone unknown. As I walked out and turned the corner I saw that the door mirror was now residing *under* my car. Another glance across the road told my head that things were worse than this - a lot worse. The whole of the driver's side from front to back was scratched, down to the metal in parts. To say I was hopping mad is an understatement. I was fuming and the language wasn't pretty, so I was glad all possible witnesses had melted away! AH, but someone hadn't just vanished, some kind person had left a note under the wiper on which was written a car registration number and the words "driven off".

I got the motor home and then rang my insurance company who took details and took me through the normal form filling, asked for drawings etc., and after commenting that as the person had not signed the note they had left for me, they would be no good as a witness, they rang off saying they would be in touch.

It was later that day that I had the thought "*could the incident have been caught on CCTV cameras located in premises nearby"?* I had got the make and colour of the vehicle involved from the registration number details so, if it had been caught on camera could we get the lowlife who likes to hit and run?



Friends and relatives all shook their heads saying the insurance company won't be bothered to chase this up, not worth it to them etc. etc., no point in bothering, wasting your time they won't want to know *BUT,* I was still hopping mad. Not mad about the damage, cars get repaired. Not mad that it had happened for the second time in four years (yes, it had happened to another car in the past) just plain fuming that these people think they can get away with it and with the knowledge that if the 'so and so' will do it to me then they'll happily do it to others!

SO, the next thing was a trip back to the scene and YES, sure enough there were CCTV cameras on buildings, so the insurance company were quickly informed. They'd look into it they said; they'd be in touch.

Friends and relatives all shook their heads again, saying no, no, no, the insurance company won't be bothered to chase this up etc., etc. You, dear reader, already know the rest and they were saying it loud and clear!

I waited, and waited with no intention of giving up, and then one day my insurance company 'phoned; they had found camera evidence, located the vehicle AND they had been on to the insurance company of the someone who had hit and run and hoped to get away with it, to invite them to pay for the repairs to my car. That insurance company was not keen to comply and it took time, patience, and sometimes a bit of pushing, but by the end of June the outcome was that my no- claims was re-instated and the excess that had had to be paid to get my car repaired was fully re-imbursed.

My slate was clean again and you'll be glad to know that thanks to the excellent garage repairers, my new car really does look as good as new again. Best of all though *the someone who was prepared to hit and run didn't get away with it this time, so* as I said, when you know you are in the right:

Don't listen to the doubters!

New regulations about driving while taking illegal drugs and certain medicines.

From www.gov.uk

A new offence of driving with certain specified controlled drugs in excess of specified levels is expected to come into force on the 2nd March 2015. The legislation also provides for a statutory 'medical defence' for this new offence, for patients taking their medicines in accordance with instructions. There is already an offence of driving whilst impaired through drugs (whether due to non-medical use of drugs or due to legitimate use of medicines) in section 4 of the Road Traffic Act 1988. This offence will remain in force alongside the new drug driving offence. This offence is not new and it has not changed. The new offence refers to driving, attempting to drive or being in charge of a vehicle with a specified controlled drug in the body, in excess of a specified limit.

The intention is to develop drug screening devices using saliva, a bit like the alcohol breath testing device, and, if positive, the person will be required to provide a blood sample to enable prosecution if above the limit - in a similar way to the current alcohol test. *Bona fide* patients taking prescribed medicines on medical advice, with a higher than specified blood level, will be able to raise a statutory defence.

There will be two groups:
Group 1 will include commonly abused drugs, and there will be a zero-tolerance approach. They include: *Cannabis (THC), cocaine (and a cocaine metabolite, BZE), MDMA (Ecstasy), Lysergic Acid Diethylamide (LSD), *Ketamine, *Heroin/diamorphine metabolite (6-MAM) and methylamphetamine
Group 2 includes medicines likely to be abused and the limits have been set higher. For these a medical defence could be raised: They include benzodiazepines i.e. lorazepam, oxazepam, *diazepam, lorazepam and temazepam and well as methadone, *morphine and amphetamine.

** In our experience these medicines have been prescribed for some people with FMS/ME/CFS. So it is important you are aware of your medicines.*

The statutory "medical defence" can be raised by patients taking medicines in accordance with instructions, from either of these two groups. A patient who was investigated for drug driving would generally be entitled to raise the statutory "medical defence" if:
a) The drug was lawfully prescribed, supplied, or purchased over-the-counter, for medical or dental purposes, and:
b) The drug was taken in accordance with advice given by the person who prescribed or supplied the drug, and in accordance with any accompanying written instructions (so far as the latter are consistent with any advice of the prescriber).

The following factors should be considered when advising a patient as to whether their driving is likely to be impaired:
It is a driver's responsibility to decide whether they consider their driving is, or they believe might be, impaired on any given occasion. As part of their normal professional practice, it is the responsibility of prescribers and suppliers of medicines to give suitable clinical advice (including advice on serious and common side-effects) to patients regarding the likely risks of their medicines. This might include, for example, for some drugs the advice that the drug may cause sleepiness and so might impair driving. Based on existing best practice, current advice given to patients about issues related to 'medicines and driving' typically covers the following points, as relevant to each case:

- Not to drive if any symptoms or signs develop suggesting that their driving may be impaired, such as experiencing sleepiness, poor coordination, impaired or slowed thinking, dizziness, or visual problems.
- Not to drive at certain times when the risk may be temporarily increased, for example, when first starting, or when first increasing or reducing the dose of, a medicine that may potentially impair their driving
- To take particular care in circumstances that may increase the risk of their driving being impaired whilst taking their medicine, and to avoid driving if this occurs.

Such situations could include:

- If another prescribed medicine is added that could also impair their driving alongside the already potentially impairing medicines
- If they take an over-the-counter medicine that could also potentially impair their driving if taken alongside the prescribed medicine;
- If other medicines are being obtained from other prescribers that may have an impact on their driving;
- If there is a developing medical condition that could increase the risk of the impairing side-effects from the prescribed medicine (for example, during the development of a serious illness with recent marked loss of weight);
- If the patient takes any new medicines that are known to be able to affect the metabolism of their existing medicine and so might impair their driving; or
- Other relevant situations, such as the effects of age or the re-initiation of a medicine that previously caused a period of sleepiness that impaired driving.
- alcohol taken in combination with other impairing drugs can substantially increase the risk of accidents.

A patient suffering with a condition that is being treated by a medicine that is also one of the specified drugs for the new offence, should normally still be encouraged to keep taking their prescribed medicine for that clinical condition in accordance with the advice of the prescriber or pharmacist. If the patient has been driving in line with such advice, and has no reason to think themselves impaired to drive (for example, not having developed new symptoms such as sleepiness), they can be advised they will be entitled to raise the statutory "medical defence".

Regarding alcohol, there is a body of evidence which shows that all of the drugs listed in the new drug driving offence result in a significantly greater road safety risk when taken in combination with alcohol, even in small amounts.

Public Notice from the Government Website

Drugs and driving: the law

It's illegal to drive if you are unfit to do so because you are on legal or illegal drugs.

If the police stop you and think you're on drugs they can do a 'Field Impairment Assessment'. This is a series of tests, like asking you to walk in a straight line and checking the size of your pupils.

If they think you're unfit to drive because of taking drugs, you'll be arrested and will have a blood test at a police station. If the test shows you've taken drugs you could be charged with a crime.

You don't have to be on illegal drugs to be unfit to drive - many prescription or over-the-counter drugs can also impair your ability to drive. If you're on legal drugs and not sure, talk to your doctor, pharmacist or healthcare professional before driving.

Penalties for drug-driving

If you're convicted of drug-driving you'll get:
A minimum 1 year driving ban
A fine of up to £5,000
A criminal record
Your driving licence will also show you've been convicted for drug driving. This will last for 11 years.
The penalty for causing death by dangerous driving is a prison sentence of up to 14 years.

Other problems you could face

A conviction for drug-driving also means:
Your car insurance costs will increase significantly
If you drive for work, your employer will see your conviction on your licence
You may have trouble travelling to countries like the US

Recipe Corner *by Carolyn*

Herb Salad with Pomegranate and Pistachios

Serves 4

Nutritional information Kcals: 131; Protein: 4g; Carbs: 8g Fat: 9g; Saturates:1g; Fibre:1g; Sugar:8g; Salt: 0.01g

Method

Mix the juice, vinegar and honey with seasoning. Tip rest of the ingredients into a large mixing bowl, drizzle over the dressing and gently toss to serve.

Note: - This salad goes especially well with lamb and potatoes

Ingredients

Juice of 1 orange
3 tbsp red wine vinegar
1 tbsp clear honey
small bunch dill, very roughly chopped
small bunch mint, picked and torn
bunch spring onions, finely sliced
100g bag mixed salad leaves
120g tub pomegranate seeds (or seeds from 1 pomegranate)
100g bag pistachios, roughly chopped

Slow-cooked lamb with onions and thyme

Serves 4

Cooking time

Ready in 3.5 hours

Five ingredients, one pot, no effort. This meltingly tender lamb dish is satisfyingly rich, and virtually cooks itself.

Nutritional information

Kcals 731; protein 63g; carbs 21g; fat 39g; saturates19g; Fibre 4g; sugar 0g; salt: 0.87g.

Added Information: Freezable

Method

Firstly, prepare the lamb. Heat oven to 160C/ fan140C/gas 3. Wipe the meat all over and season well. Heat 3 tbsp of olive oil in a large heavy flameproof casserole; add the meat and fry all over on a fairly high heat for about 8 mins, turning until it is evenly well browned. Remove to a plate.

Thinly slice the onions. Add to the pan and fry for about 10 minutes, until softened and tinged with brown. Add a few of the thyme sprigs and cook for a further minute or so. Season with salt and pepper.

Sit the lamb on top of the onions, and then add the wine. Cover tightly. Bake for 3 hours. You can make to this stage up to 2 days in advance, and then reheat for 45 minutes.

To finish off, strip the leaves from 2 thyme sprigs and chop them with the parsley. Scatter over before serving.

Ingredients

half a leg of lamb (about 1¼kg/2lb 12oz)
1kg onions (about 4 large ones)
handful of thyme sprigs
300ml red wine

Out and About: The Lynton & Lynmouth Cliff Railway.

Driving west along the A39 in the north part of Exmoor, Devon, is quite a calming yet exhilarating experience. To your left is the wilderness of Exmoor and to the right is the sea. At this point the road is about 900 feet above sea level, and Exmoor drops to meet the sea with steep cliffs. Quite unexpectedly you come across a notice "Counisbury Hill. Gradient, 1 in 4". A little further on is a small discreet signpost warning of a 25% hill and a notice to keep in low gear. Suddenly you find you are on an unfenced road, with a sheer drop to your left of 900 feet to the sea, and on your right the ground starts to rise. As you descend the hill there are exit points for vehicles in case of break failure. At the bottom of the hill, you arrive at the village of Lynmouth. There is a small chocolate box seafront with a small beach and harbour where the River Lyn meets the sea. Crossing over the bridge and driving along the Esplanade, you eventually come to a car park which is about two cars wide. Just at the side of the Exmoor National Park Information Office is a Café, and then a green gate. That is the Lynton & Lynmouth Cliff Railways Station. Looking at car parking, further down the Esplanade is a blind-end car park. There is no free disabled car parking; it is all Pay and Display. After such an adventurous decent from Exmoor, we decided to have a cup of tea and snack. There is a café where you can enjoy a cup of tea on the roof and watch the operation of the railway next to the Exmoor Park Information Office.

The railway is funicular railway, a special type of railway that travels up and down steep slopes, with the carriages being pulled by a strong metal rope. Half way up there are two opposing curves in the track so that the carriages can pass each other. When each carriage is 'docked' with full water tanks of 300 litres at both stations the cars are in balance (weighing the same) and are ready for boarding. As passengers board, the variations are accommodated for by the brakes which clamp the cars to the rails. Each car's brakes can hold the weight of both cars fully laden. In addition to this the lower car has a water-operated locking device which clamps the car to the bottom station.

When loaded, the drivers use pre-arranged bell signals, unlock the safety locking device - then both cars' brakes are released. The lower driver then discharges water (if required) to make the top car heavier. Sometimes this is achieved with the weight of passengers alone and no water is used. The top car then rolls down the rails - at the same time pulling the lower car up. Each car has two sets of brakes which are water-operated. The 'governor', which in turn, is driven by the main wheels, operates one set. These brakes have 'shoes' which press down on the top surface of the rail and actually lift the car off the rail by 2mm, thereby relying on the weight of the car to give maximum friction between the rail and the brake-shoes. The other set of brakes work in reverse to a conventional brake system, such as that found in a motor car. In a car, the driver presses the pedal to apply the brakes. However, on the railway, the brakes are permanently on - operated by a large water accumulator via the drivers hand wheel. This means when the cars are unattended, the brakes clamp it to the rails making it impossible to move under any circumstances. These brakes are a calliper type which grips each side of the crown of the rail.

The railway needs absolutely no power to operate, as the weight of the water is its motive power. The Lift works on a total loss system; water that is released at the bottom is not reused. When the Lynmouth and Lynton Lift Company was formed through an Act of Parliament in 1888 the Act gave them perpetual right to extract up to 60,000 gallons of water a day from the river on the moors, quite a foresight all those years ago. The water is not damaged or polluted in any way, just used as ballast and dropped on the beach at Lynmouth about 100 metres away from the river from which it was taken. The carriages themselves are green. They do not create any emissions; their carbon footprint today has not significantly changed since the lift opened and it is probably one of the most environmentally friendly tourist attractions in the country and has been for well over a hundred and twenty years.

Disabled Access

The railway says that disabled access is available, but due to the nature of the railway and its Victorian carriages it is advisable to telephone prior to your visit for further information as most wheelchairs will not pass through the carriages unfolded and there are three steps through each. They can offer a return journey on the open platform for any wheelchairs that will not pass through the carriages.

To board the railway from the lower station you have to first buy a ticket and wait in a queue. The area is quite small, and on a slope, so someone with a wheelchair may have difficulties. The adult return fare is £3.50. We boarded the carriage - some people choose to travel on the balcony outside. The driver signalled with bells to the driver at the top, turned his brass wheel to let out the water and we started to rise. There is no engine, only silence and a slight rumble. At times the driver applied to brakes to control the rate of climb. As we rose, there was a unique panoramic view of Lynmouth and the surrounding land. Unfortunately on the day we travelled there was a slight haze which became a fog the closer we got to the top station. The photographs we took did not do any justice to the view so I have included one from the railway's website.

Once we got to the top the driver put on the brakes and started to fill up the water tank for the next descent. There is a café with outdoor tables next to the station. There is also a road which takes you into the village of Lynton, on top of the cliff. However for us, with ME, the walk would have been too long. The café & view were well worth the ride.

The way down was very similar and uneventful apart from an air ambulance flying around very close to us. At the bottom we got off—and walked to the car having enjoyed our little adventure and looking forward to the ride home—so we thought. When we got to the bottom of Counisbury Hill our way was blocked. A fire engine was reversing up the 1 in 4 hill. We were told there had been an accident and the A39 to Minehead would be closed for the rest of the afternoon. So we backed into a pub car park to plan what to do next. So up Lynmouth Hill we went on the B3234, which followed the Lyn Valley. The car engine was revving and the best we could do was 12 mph. The road was narrow and winding. Once we got to the top of the hill we stopped and checked the car. Everything was OK. We set the satnav, and found our way home via the B3358 which was a road very similar to those in the rolling landscape around Doncaster. When I later checked on a map of Lynmouth Hill, some of the gradients in that road are 1 in 3. That is about the limit that a family car could take and explains why the best we could do was 12 MPH. It also goes to illustrate the purpose of the Cliff railways to link Lynton & Lynmouth and the top & bottom the cliff. Verdict: Well worth a visit.

You Write on Statins

Nel writes: Further to your last meeting, I used to take statins but I no longer do so. My muscle pains increased and my memory got worse while taking them. I still have high cholesterol levels but just accept that's better than the negative side effects. I don't take anything else, but do eat a very good diet e.g. oats, soya, fruit and veg etc. etc.

If your doctor prescribed a statin there is usually a good reason for it. Unfortunately this is something I hear frequently from members. I don't think you have any option but to get your cholesterol down. I think you should consider the other medicines available. We've expanded this in the later pages of this issue of Pathways.

Mick writes: Further to your last meeting, I have a diagnosis of CFS. I have high blood pressure and angina. I have had two heart attacks and stents. I started taking simvastatin 7-8 years ago. At first I stuck rigorously to a prescribed diet, but my cholesterol increased. The problem I have is hereditary. I am still taking simvastatin and my cholesterol is under control. The simvastatin does not cause me any side effects other members have described.

Given you medical history, you should carry on as you are. It just goes to show that some ME/CFS people can tolerate statins.

Gwengie writes: Several years I was put on simvastatin for a hereditary high-cholesterol problem. The problems I had with simvastin were horrendous. I had a word with my doctor and he put me on bezafibrate and ezetimibe which figures crossed, just keeps my cholesterol under control. I heavily take supplements along Dr Myhill's lines and I think that the Q10 that I take helps keep the side-effects of my cholesterol medicines at bay.

Keep up the good work. There is a feature from Pharma I.INord which will be of interest to you later in this Pathways edition.

Carl Writes: I've just turned 70 and I've just had an over-70s health MOT with my doctors. I had a thyroid operation a few years ago. Apart from the ME/CFS and arthritis everything is OK except my cholesterol figure. I have attached my test results. My LDL cholesterol is 4.1 (should be below 3) and my total cholesterol is 7.5 (should be below 5). My TSH is 1.0 (Normal range 0.3 to 5). I have been told that even if I stuck to a strict diet, it would not be enough, so my doctor has prescribed atorvastatin, but I have checked on the internet with the manufacturer's website and with various internet web sites. I have enough pain and fatigue without the additional problems caused by side effects of atorvastatin.

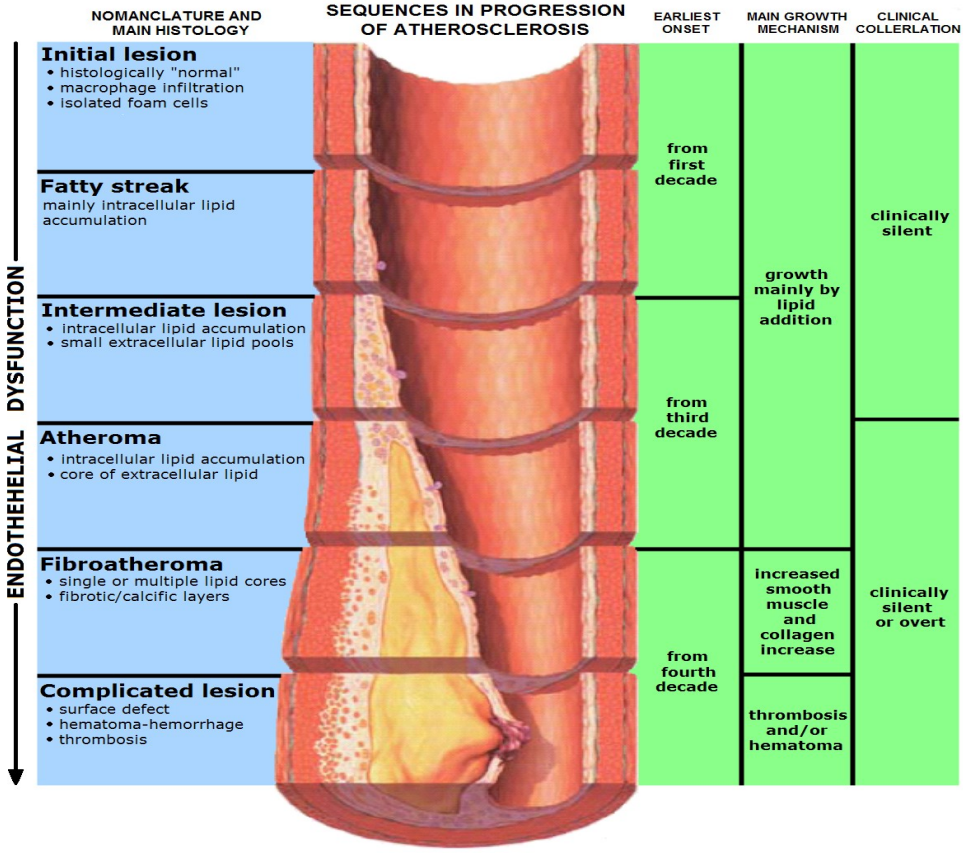
I would say that with those figures, the LDL cholesterol is level is a strong threat to your future health, and I think you need to do something to get the LDL down. I think your doctor has taken your ME/CFS into account by prescribing atorvastatin, because it has a cleaner profile of side effects than simvastatin. I am however concerned that he has not checked your T3 and T4 levels. The normal NHS thing is just to test TSH, but according to Dr Myhill "the TSH test alone tells you very little about the thyroid status. It is essential to see the actual levels of thyroid hormone in blood." (See page 225 of her book.) Also the BNF states that: "Patients with hypothyroidism should be adequately treated with thyroid replacement medicines before any lipid-regulating treatment, because correcting hypothyroidism may sort the problem. Untreated hypothyroidism increases the risk of adverse effects." Thyroid resistance is an issue in ME/CFS so it is important to know the levels of T3 and T4. It may be possible that your current thyroid medicine needs to be increased or changed. As thyroid levels have a major effect on cholesterol levels it may be possible for that to normalise your figures. However I also know that it is standard in many NHS doctors' practice only to test TSH but like Dr. Myhill I think that is inadequate for ME/CFS patients. I think you should see if the T3 & T4 could be tested. If not you could get it done privately for around £77 independently of your NHS doctor. Contact us for further information. In the meantime, it would be prudent to do what you can by dietary measures. The BDA fact sheet which is reproduced on later pages many be useful. I would certainly consider taking Coenzyme Q10; please see the Pharma Nord factsheet in the following pages.

What are atheroma and cardiovascular diseases?

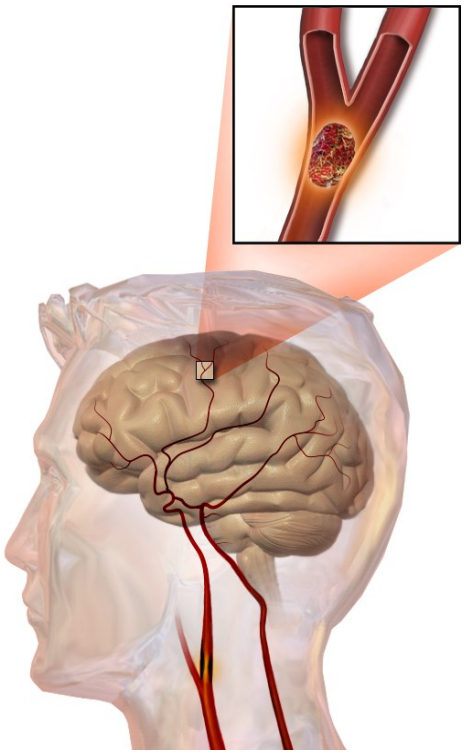
Patches of atheroma are like small fatty lumps that develop within the inside lining of arteries (blood vessels). Atheroma is also known as atherosclerosis and hardening of the arteries. Patches of atheroma are often called plaques of atheroma. Over months or years, patches of atheroma can become larger and thicker. So in time, a patch of atheroma can make an artery narrower. This can reduce the blood flow through the artery. For example, narrowing of the coronary (heart) arteries with atheroma is the cause of angina.

Sometimes, a blood clot (thrombosis) forms over a patch of atheroma and completely blocks the blood flow. Depending on the artery affected, this can cause a heart attack, a stroke, or other serious problems.

Cardiovascular diseases are diseases of the heart (cardiac muscle) or blood vessels (vasculature). However, in practice, when doctors use the term cardiovascular disease they usually mean diseases of the heart or blood vessels that are caused by atheroma.

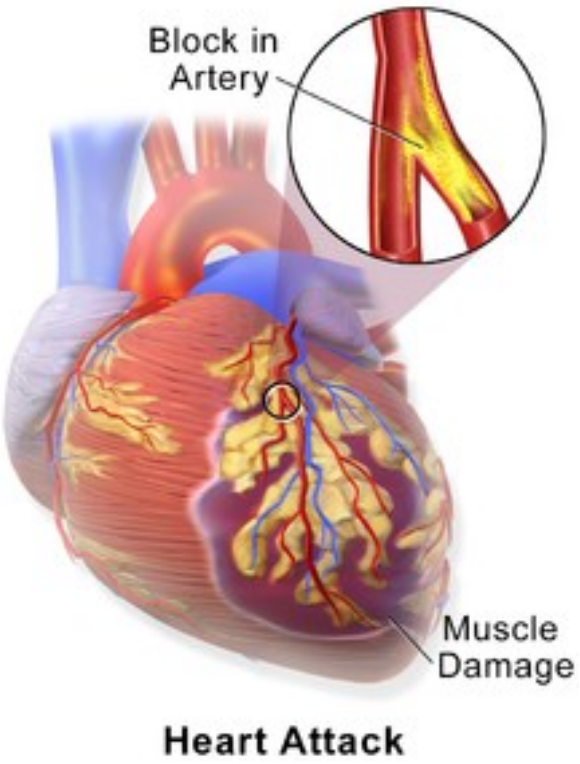


In summary, cardiovascular diseases caused by atheroma include: angina, heart attack, stroke, transient ischaemic attack (TIA) - sometimes called mini-stroke - and peripheral vascular disease. In the UK, cardiovascular diseases are a major cause of poor health and the biggest cause of death.

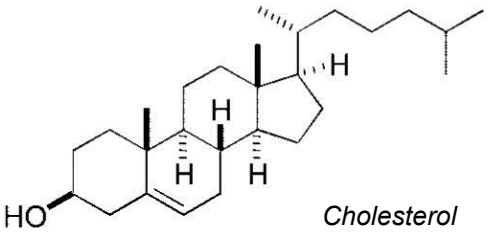


A myocardial infarction (MI) occurs when an atherosclerotic plaque slowly builds up in the inner lining of a coronary artery and then suddenly ruptures, causing catastrophic thrombus formation, totally occluding the artery and preventing blood flow downstream.

Something similar happens with narrowing of the carotid artery or blocking of the artery in the brain. This is one cause of a stroke,



Cholesterol Blood Tests (from Patient UK)



Cholesterol

Cholesterol blood tests are done to help assess your risk of developing heart disease or stroke. If your risk is high then you will usually be advised to take a statin medicine to lower your cholesterol level. Lowering your cholesterol level reduces your risk, even if your cholesterol level is normal. Other factors that can reduce your risk include: not smoking, choosing healthy foods, a low salt intake, regular physical activity, keeping your weight and waist size down and drinking alcohol in moderation. Ensuring your blood pressure level is not raised (or taking medication to lower it if it is high) is also important.

What is cholesterol? Cholesterol is a lipid (fat chemical) that is made in the cells in your body. Many different cells make cholesterol but cells in the liver make about a quarter of the total. Although many foods contain cholesterol, it is poorly absorbed by the gut into the body. Therefore, cholesterol that you eat in food has little effect on your body and blood cholesterol level. A certain amount of cholesterol is present in the bloodstream. You need some cholesterol to keep healthy. Cholesterol is carried in the blood as part of particles called lipoproteins.

There are different types of lipoproteins, but the most relevant to cholesterol are:

- Low-density lipoproteins carrying cholesterol - LDL cholesterol. This is often referred to as bad cholesterol. This is the one mainly involved in forming atheroma. Atheroma is the main underlying cause of various cardiovascular diseases. The majority of cholesterol in the blood is LDL cholesterol, but how much varies from person to person.

- High-density lipoproteins carrying cholesterol - HDL cholesterol. This is often referred to as good cholesterol. This may prevent atheroma forming.

What factors affect the blood level of cholesterol? To an extent your blood cholesterol level can vary depending on your diet. However, different people who eat the same diet can have different blood cholesterol levels. In general, however, if you eat less fatty food in your diet your cholesterol level is likely to go down. In some people a high cholesterol level is due to another condition. For example, an underactive thyroid gland, obesity, drinking a lot of alcohol and some rare kidney and liver disorders can raise the cholesterol level. In some people a very high level of cholesterol runs in the family, due to a genetic problem with the way cholesterol is made by the cells in your body. One example is called familial hypercholesterolaemia.

Risk factors

patient years

Lifestyle risk factors that can be prevented or changed:

- Smoking.
- Lack of physical activity (a sedentary lifestyle)
- Obesity
- Unhealthy diet
- Excess salt.
- Excess alcohol.

Treatable or partly treatable risk factors:

- High blood pressure (hypertension).
- High cholesterol blood level.
- High triglyceride (another type of fat) level.
- Diabetes.
- Kidney diseases that affect kidney function.

Fixed risk factors - ones that you cannot alter:

- A strong family history.
  This means if you have a father or brother who developed heart disease or a stroke before they were 55, or in a mother or sister before they were 65.
- Being male.
- An early menopause in women.
- Age. You are more likely to develop atheroma as you get older.
- Ethnic group.
  For example, people who live in the UK whose family came from India, Pakistan, Bangladesh, or Sri Lanka have an increased risk.

A summary of the British National Formulary (BNF) recommendations.

These are for people with a high risk of developing cardiovascular disease (primary prevention) and to prevent recurrence of events in those with established cardiovascular disease (secondary prevention).. Those with high risk include those who already have atherosclerotic disease, those with diabetes over 40 years, and those with familial hypercholesterolaemia. The risk also increases with age. Those over 75 years are at particularly high risk, particularly with smokers or people with high blood pressure. Preventative measures are required for those with a 10-year risk of cardiovascular disease of 20% or more. The risk is assessed on the basis of lipid concentration as well taking into account the other risk factors.

Patients with hypothyroidism should be adequately treated with thyroid replacement medicines before any lipid-regulating treatment, because correcting hypothyroidism might resolve the problem. Untreated hypothyroidism increases the risk of adverse effects. *(Thyroid resistance is a known issue in some cases of ME/CFS. Ed)*

Lowering the concentration of low-density lipoprotein (LDL) cholesterol and raising high-density lipoprotein (HDL) cholesterol slows the progression of atherosclerosis and may even induce regression. Lifestyle modifications are beneficial e.g. changes to diet, exercise *(not usually feasible with ME/CFS),* weight management, reducing alcohol consumption, and smoking cessation.

A statin reduces the risk of cardiovascular disease events, irrespective of serum cholesterol concentration, and is the drug of first choice for primary and secondary prevention. Second choices are fibrates and/or a bile acid sequestrant.

A statin is recommended for all patients with families with high cholesterol. The BNF recommends a high-intensity statin. Simvastatin, or atorvastatin should be considered in order to achieve the recommended reduction in LDL-cholesterol, but is associated with an increased risk of muscle toxicity the combination of a statin and ezetimibe can be considered if a statin alone fails to provide adequate control and when a switch to an alternative statin is being considered. Patients, for whom statins and ezetimibe are inappropriate, should be referred to a specialist for the consideration of treatment with a bile acid sequestrant, nicotinic acid, or a fibrate.

Other options

Fibrates used are alternative to statins and carry some risk of muscle side effects
Bile acid sequestrants deplete fat-soluble vitamins; supplements of vitamins A, D, K, and folic acid may be required when treatment is prolonged. They may adversely affect IBS type symptomology experienced by some ME/CFS patients.
Ezetimibe inhibits the intestinal absorption of cholesterol. It is an add in second line medicine, but can increase side effects of statins
Lomitapide is bolt on relatively recent medicine also associated with gastro-intestinal motility disorders,
The following are included in the BNF and people who take these report some benefit with their ME/CFS.
Nicotinic acid group (B3 group) main side effect is flushing, Increases HDL (good) cholesterol.
Omega-3 fatty acids (eg. Omcor, fish oil) reduce triglycerides. They are components of VegEPA/ Maxepa etc. taken for ME/CFS.

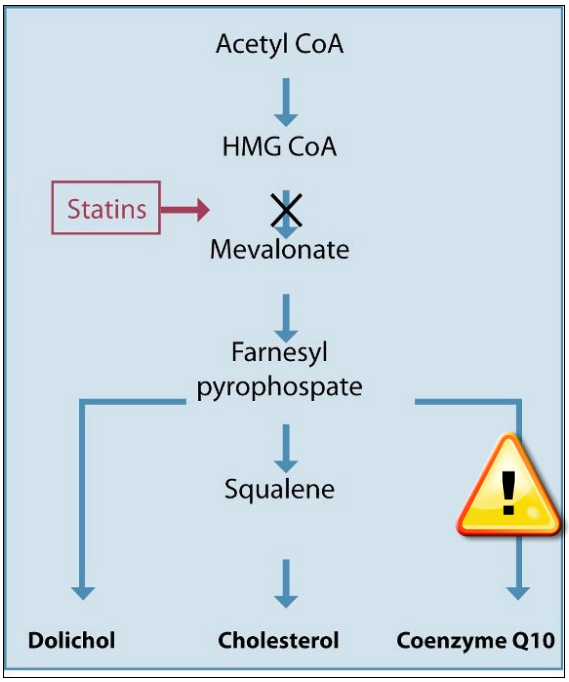
Target Cholesterol blood levels

The following levels are generally regarded as desirable but vary according to the source and country:-

- Total cholesterol (TC) - 5.0 mmol/L or less. However, about 2 in 3 adults in the UK have a TC level of 5.0 mmol/L or above.
- Low-density lipoprotein (LDL) cholesterol after an overnight fast: 3.0 mmol/L or less.
- High-density lipoprotein (HDL) cholesterol: 1.2 mmol/L or more.
- TC/HDL ratio: 4.5 or less. That is, your total cholesterol divided by your HDL cholesterol. This reflects the fact that for any given TC level, the more HDL, the better.

The higher the LDL cholesterol level, the greater the risk to health, however this has to be taken in context.

Issues around ME/CFS patients taking statins.



I was browsing some research papers and came across an interesting abstract:
Reference:
Maes M1, Mihaylova I, Kubera M, Uytterhoeven M, Vrydags N, Bosmans E.Neuro Endocrinol Lett. 2009;30(4):470-6.)

Coenzyme Q10 deficiency in Myalgic Encephalomyelitis/ Chronic Fatigue Syndrome (ME/CFS) is related to fatigue, autonomic and neurocognitive symptoms and is another risk factor explaining the early mortality in ME/CFS due to cardiovascular disorder.

This reviews the role of Coenzyme Q10 (CoQ10), a mitochondrial nutrient which acts as an essential cofactor for the production of ATP in mitochondria and which displays significant antioxidant activities. The authors assessed Plasma CoQ10 in 58 patients with ME/CFS and in 22 normal controls. The relationships between CoQ10 and the severity of ME/CFS as measured by means of the Fibro Fatigue (FF) scale were measured. They found that Plasma CoQ10 was significantly lower in ME/CFS patients than in normal controls. They claim that up to 44.8% of patients with ME/CFS had values beneath the lowest plasma CoQ10 value detected in the normal controls, i.e. 490 micrograms per litre (µg/L). In ME/CFS, there were significant and inverse relationships between CoQ10 and the total score on the FF scale, fatigue and autonomic symptoms. Patients with very low CoQ10 (<390 µg/L) suffered significantly more from concentration and memory disturbances. In their discussion the researchers concluded that the results show that lowered levels of CoQ10 play a role in the pathophysiology of ME/CFS and that symptoms such as fatigue and autonomic and neurocognitive symptoms may be caused by CoQ10 depletion. They further suggest that patients with ME/CFS would benefit from CoQ10 supplementation in order to normalize the low CoQ10 syndrome and the IO&NS disorders. The findings, that lower CoQ10 is an independent predictor of chronic heart failure (CHF) and mortality due to CHF, may explain previous reports that the mean age of ME/CFS patients dying from CHF is 25 years younger than the age of those dying from CHF in the general population. Since statins significantly decrease plasma CoQ10, ME/CFS should be regarded as a relative contraindication for treatment with statins without CoQ10 supplementation.

Advice from the ME Association on Statins

- MEA remind you to report changes in muscle symptoms if you take statins (13th August 2013)
- Statins are drugs that are prescribed to reduce the risk of heart problems by lowering the level of cholesterol in the blood – when this is elevated.
- The most common side-effects of statins include adverse effects on skeletal muscle (myalgia = muscle pain, myositis = inflammation of muscle and occasionally rhabdomyolysis). Where muscle problems occur the drug may have to be discontinued.
- People with pre-existing muscle disorders appear to be at increased risk of developing these side-effects. So these symptoms should always be taken into consideration before a statin is being considered.
- Doctors have now been issued with comprehensive guidance on how to deal with patients who report muscle symptoms when taking statins.
- www.eguidelines.co.uk/eguidelinesmain/guidelines/summaries/cardiovascular/wpg_statin_2013.php#.Ugn9DBb3DJx
- Anyone with ME/CFS who is taking statins and notices new muscle symptoms or an exacerbation of existing muscle symptoms should always consult their doctor – who will arrange for a blood test to measure the level of a muscle enzyme called CK (creatine kinase)
- The MEA receives regular queries on the use of statins and has been sending out this information for some time!

Just a minute, this seems a bit contradictory. Is there not a massive investment within the NHS in advocating statin type drugs to protect people from the catastrophic consequences of heart attacks and strokes due to arthrosclerosis and high cholesterol levels? Yet it seems to imply that people with ME/CFS are dying because the statin-type drugs intended to protect them? I do know from my own experience that many people with ME/CFS who have been given statins have suffered serious side effects, and I have in the past sent around nine yellow adverse reaction cards to the MHRA. I also know a couple of cardiologists that advocate statins for everyone with any hint of a cardiovascular condition. I need to look into this further.

On visiting Dr. Sarah Myhill's website I found the following.

The Problem with Statins

The interesting thing about statins is that they do reduce one's risk of many diseases, but the degree to which they protect one is not commensurate with the degree with which they reduce cholesterol levels. We now suspect the reason why. Statins are vitamin D mimics - they look exactly like vitamin D and have many of vitamin D's beneficial effects. Vitamin D evolved because of sunshine which is markedly pro-inflammatory. By making vitamin D in the skin in response to the sunshine, and vitamin D is very anti-inflammatory, this allowed people to tolerate the pro-inflammatory effects of sunshine. This anti-inflammatory effect of vitamin D spreads through the whole body. Many degenerative diseases of ageing are associated with inflammation and vitamin D protects against this. Therefore it is highly protective against arterial disease, heart disease, neoplastic disease, autoimmunity (including multiple sclerosis and type I diabetes), neurodegenerative conditions, osteoporosis, allergies and so on, indeed any condition associated with inflammation.

The main problem with statins is that they inhibit two important enzyme systems. The first of these is Coenzyme Q 10 - this is the most important antioxidant inside mitochondria and the main acceptor and donor of electrons. This means that statins will slow down mitochondria and the ageing process may be accelerated. There is now good evidence to show that poor mitochondrial function is a central part of Chronic Fatigue Syndrome and this explains why statins almost invariably make patients with chronic fatigue worse. Statins also inhibit formation of selenium-based proteins such as glutathione peroxidase. This is one of the most important antioxidants in the blood and is essential to maintain cholesterol in its desirable unoxidised state.

It is a combination of the above two factors which explains the devastating effect statins have on some people's muscle metabolism. People get obvious muscle soreness, stiffness, weakness and fatigability. Heart muscle is little different from normal muscle so it is no wonder that the heart is also affected and theoretically this could result in heart failure.
Because statins interfere with antioxidant defences and energy supply they may be contributing to the raised levels epidemic of Alzheimer's symptomology we are now seeing. E.g. Brain fog - poor memory, difficulty thinking clearly etc.

There is a feature from the Lancet (The Lancet, Vol 361, Issue 9363, Pages 1134 - 1135, 29/3/03) Entitled Statins and Coenzyme Q10 by Dr's. CJ Ellis and ,R Scott

Their comment is that interest in the ability of statins to block the biosynthesis of Coenzyme Q10 could unintentionally result in a decrease in the use of these medicines by patients who are at risk of major cardiovascular events. They note an increase in the number of patients who decline statin medication, after being influenced by statements and claims regarding Coenzyme Q10. They say an alarmist letter is readily available on the internet. The dialogue then continues that:

"*Clinicians are already very aware of the problem of under treatment of high-risk patients who would benefit from a statin drug. We should not compound this difficulty by allowing our limited knowledge of Coenzyme Q10 to interfere with the prescription of proven, safe medicines for our patients."*
(Presumably this is outside the ME/CFS Context. Ed)

Statins: Balancing benefits and risks with reference to ME/CFS.

Having ME/CFS does not exempt anyone from cardiovascular risks or other disease. Here is what I see locally, taking a conventional medicine viewpoint of the risk levels can be summarised as follows:

CV incident Risk Level	Typical History	Examples	Usual advised cardiovascular protection
Very High risk	Patients with history of cardiovascular obstruction due to arthrosclerosis.	History heart attack or stroke	Mandatory with multiple medicines
High	Patients with a condition known to carry a high risk of a CV incident.	History of heart arrhythmias & angina	Essential with multiple medicines.
Moderate	Patient suffering from a chronic condition known to make people prone to CV incidents	Diabetes, various inflammatory diseases like some forms of arthritis, some neoplastic disease, poor dental hygiene	High level mitigation insurance, mandatory with medicines and diet
Moderate to low	Patients without symptoms showing a high blood level of lipids	Patient with a family history of high cholesterol or with a high cholesterol count from blood tests	Mitigation insurance desirable with medicines. Diet may help and lifestyle changes may help.
Low	Patients without symptoms suffering from a chronic condition limiting physical activity or with an otherwise poor lifestyle	ME/CFS/FMS patients and those suffering from other neurological conditions. Activity-limiting conditions	Diet alone may help and other lifestyle changes may help. Over the counter statins are available.
Very low	Patients without symptoms leading a healthy lifestyle.	An active healthy fit person	No intervention needed

According to the MHRA website (May 2014) the statins currently available in the UK are simvastatin, atorvastatin, pravastatin, fluvastatin, and rosuvastatin.

The ME/CFS related issues that concern us are:

- Generally UK conventional medicine does not acknowledge ME/CFS specifically
- The British National Formulary does not acknowledge ME/CFS patients in its entirety as a specific group or even mention ME/CFS.
- The current NICE guidelines on ME/CFS (as opposed to the Canadian guidelines) do not adequately reflect the cardiovascular issue around ME/CFS.
- The Kerr sub typing which means there are different types of ME/CFS, is not acknowledged.
- Many patients with ME/CFS have risk factors which increase their CV risk e.g. limited ability to exercise, poor diet, sedentary lifestyles & disturbed sleep.
- When statins are prescribed in local hospitals or GP surgeries CoQ10 levels are not checked. Our local hospital Doncaster Royal Infirmary appears not to have a facility to supply or test for CoQ10.
- Issues of thyroid resistance are not checked for, which could have a big influence on cholesterol levels. Only TSH is checked. Many people with ME/CFS have T4 to T3 conversion problems.

Putting statins, cholesterol and patients with ME/CFS in context

Muscle-related side effects of statins. According to the MHRA website, all effective medicines can cause side-effects in some patients and a small proportion of patients taking statins will inevitably experience side-effects. Although they may be distressing to the individual concerned and limit that individual's willingness or ability to tolerate statin use, statin-related side-effects are generally mild and not medically serious. Muscle-related problems are the most frequently reported side-effect of statins. The following statin side-effect incidences have been estimated based on randomised trial data, cohort studies, published case reports and spontaneous reports:-

Mild muscle pain: 190 cases per 100,000 patient years
Myopathy: 5 cases per 100,000 patient years
Rhabdomyolysis: 1.6 cases per 100,000 patient years

I decided to ask the Leger ME members, so I sent out an email on the group network asking:

I*'m doing a feature for Pathways about member's experiences with statin-type medicines. Common ones locally are simvastatin or atorvastatin. I'm interested in your experiences either positive or negative.*

Do they work for you? Of the 11 replies received, 6 were currently being prescribed a statin. We can assume that they are being prescribed and monitored by the GP. It would be reasonable to assume that they are working satisfactorily.
Do or did you get any side effects? Three reported issues with fatigue and/or muscle pain. There was one case with hallmarks of rhabdomyolysis. One person with a moderate risk asked their GP to prescribe a statin and was refused on the grounds of the risk of increasing fatigue and muscle pain.
Are you still taking a statin now? Five were taking simvastatin and two atorvastatin which included one taking both of these medicines.
Have you tried any alternatives to statins? Two reported taking Bezafibrate (a non-statin alternative). One of these was after apparent rhabdomyolysis.
Have you taken Co-enzyme Q10 ubiquinone, ubidecarenone) and did it help your ME/CFS? Three reported taking CoQ10 helps and one said it didn't make a difference.

Interestingly no one responded in the high risk groups or the low risk groups,

So could we assume that about half of people with ME/CFS could tolerate a statin, and around one third will experience adverse side effects, and stop taking them and that about half of these will take alternative?

If your doctors want you to take a statin then take it seriously. You have to get your cholesterol down. It would seem logical that the following steps are taken prior to prescribing:

1) A thyroid function test is done measuring TSH, T4 & T3 to ensure that this is not a problem. Commonly only TSH is measured. All three are available at NHS facilities locally.
2) Blood levels creatine kinase levels are checked. This is normally raised in some patients with ME/CFS, and is also raised if rhabdomyolysis is an issue.
3) Blood levels of CoQ10 are checked if they have not previously been checked. This test is not available from the NHS locally. You own doctor could arrange for this privately though Biolab the cost being around £50. Alternatively you could contact Dr Myhill, but would incur a bill for just under £200
The tests are repeated along with the usual cholesterol tests at frequent intervals.

Whatever you are prescribed, please remember that it will be lifelong — so it is important to you have the confidence to carry on with ongoing treatment. I have included a hand-out from the BDA, and an information sheet from Pharma Nord, which may be useful should you wish to supplement .

BDA The Association of UK Dietitians

Food Fact Sheet

Stanols and Sterols

Plant stanols and sterols, also called phytostanols and phytosterols, occur naturally in small amounts in a range of plant foods such as fruits, vegetables, nuts, seeds, legumes, cereals and vegetable oils (e.g. soyabean and corn oil).

On average, most people's diets provide a small amount of plant stanols (20-50mg) and sterols (150-400mg) per day. Vegetarian diets can contain 50% higher amounts of sterols compared to non-vegetarian diets.

What do Plant Stanols and Sterols do?

Plant stanols and sterols have a similar structure to cholesterol and, because of this, help to reduce the absorption of cholesterol in the gut. This helps to lower total cholesterol and LDL cholesterol (the bad type of cholesterol) levels in the blood. Both plant stanols and sterols have similar effects on cholesterol. High blood cholesterol levels increase the risk for coronary heart disease.

How much is needed to help lower blood cholesterol levels?

Plant stanols and sterols are not an essential part of managing high cholesterol levels. Research has shown that 2-3 grams is needed each day to help lower cholesterol in people with raised cholesterol levels. The majority of studies show that eating more than 3 grams per day of plant stanols/sterols will not lower cholesterol levels further. However, recent studies have shown greater cholesterol lowering effects with up to 9 grams per day of plant stanols without any harmful side effects. Further research is needed to confirm these findings.

Why are they added to foods?

Our diets do not provide enough plant stanols and sterols to help lower cholesterol. A range of food products have plant stanols and sterols added to them. These products can help us to meet the suggested effective dose to help lower cholesterol. These include milk, yogurts and yogurt drinks. Examples of brands of foods that contain these plant stanols and sterols include Benecol and Flora pro.activ as well as supermarket own brands. These products tend to be more expensive compared to ordinary spreads, milk and yogurts. If you are trying to reduce your weight, choosing the lower fat versions of these products will provide a lower amount of calories.

Products containing added plant stanols and sterols need to be eaten every day in the recommended amounts to have a beneficial effect on lowering cholesterol. The beneficial effect is lost when you stop using these products.

> "Following a healthy diet low in saturated fats in combination with eating the recommended amounts of plant stanols and sterols each day can lower LDL cholesterol by 15%."

www.bda.uk.com/foodfacts

Is it safe to use them with cholesterol-lowering drugs?

Studies have shown that products containing plant stanols and sterols are safe. They should not replace cholesterol-lowering medications prescribed by your doctor but can be used in addition to these medications. This may have a greater overall effect on lowering cholesterol levels. If you do not have raised cholesterol levels, there is no benefit from including these foods in the diet. Products containing plant stanols and sterols are not recommended for children under the age of 5 years, breastfeeding or pregnant women.

> "Products containing plant stanols and sterols are not recommended for children under the age of 5 years, breastfeeding or pregnant women"

Examples of how the suggested amount of daily plant stanols and sterols can be achieved:

Product	Package Size
Asda cholesterol lowering yogurt drink	100g
Benecol dairy free yogurt drink	65.5g
Benecol yogurt drinks	67.5g (original flavour is 70g)
Flora pro.activ mini yogurt drinks	100g
Tesco healthy living cholesterol-reducing yogurt drink	100g

EITHER

one of the drinks to the left per day

OR

2-3 servings of the products below per day

Product Type	Product	Serving amount providing 1 portion of stanols/sterols
Spreads 	Benecol spreads (light, olive and buttery tastes)	12g (>2 level teaspoons)
	Flora pro.activ speads (light, olive and buttery tastes)	10g (2 level teaspoons)
Milk 	Flora pro.activ skimmed milk	250ml (1 medium glass)
Yogurts	Benecol low fat bio yogurts	125g (1 pot)
Cream cheese style spread 	Benecol light cream cheese style spread	20g (4 level teaspoons)

Summary

High blood cholesterol levels increase he risk for coronary heart disease. Although plant stanols and sterols are not an essential part of managing high cholesterol levels, research has shown that following a healthy diet low in saturated fats in combination with eating the recommended amounts of plant stanols and sterols each day can lower LDL cholesterol by 15%.

Our diets do not provide enough to help lower cholesterol but there are a range of food products that have plant stanols and sterols added to them which can help us meet the suggested effective dose to help lower cholesterol. However, they should not replace cholesterol-lowering medications prescribed by your doctor but can be used in addition.

Co-enzyme Q10 *from the Pharma Nord Website (with consent).*

- Coenzyme Q10 helps to maintain a healthy cardiovascular system, and benefits patients with cardiovascular disease, with benefits for hypertension, hyperlipidaemia, coronary artery disease and heart failure, and treatment with lipid-lowering statin drugs
- Coenzyme Q10 inhibits the development of periodontal disease.
- Coenzyme Q10 benefits patients with Parkinson's disease.

What is coenzyme Q10?

Coenzyme Q10 (also known as ubiqinone) is a vitamin-like substance which plays a vital role in the body's energy supply mechanism, acting in conjunction with enzymes (hence the name Coenzyme Q10) to convert sugars and fat into energy. Coenzyme Q10 is also important as an antioxidant within the body. Coenzyme quinones occur in several chemical forms, with Coenzyme Q10 being the only form found in human tissues. The human body is able to synthesize (in the liver) a limited amount of Coenzyme Q10, with the remainder being obtained from the diet. Rich sources of Coenzyme Q10 include fish (mackerel, salmon, sardines) and nuts. Under normal circumstances, the body is able to maintain adequate levels of Coenzyme Q10. However, Coenzyme Q10 levels decrease with age, and are depleted by intense physical exercise or illness.

What does coenzyme Q10 do?

Coenzyme Q10 is an essential cofactor of enzymes involved in the energy production process. Coenzyme Q10 is stored in mitochondria, structures found within cells responsible for the generation of energy (in the form of a molecule called ATP). Tissues with a high energy requirement (heart, liver, skeletal muscles) contain higher numbers of mitochondria within their cells. Coenzyme Q10 is also important within the body as a major fat-soluble antioxidant, protecting cell membranes from the damaging effects of free radicals which have been implicated in the pathogenesis of many diseases, in a complementary manner to vitamin E. Clinical studies have shown coenzyme Q10 to have the following benefits:

•Coenzyme Q10 helps to maintain a healthy cardiovascular system, and benefits patients with cardiovascular disease, with benefits for hypertension, hyperlipidaemia, coronary artery disease and heart failure, and treatment with lipid lowering statin drugs
•Coenzyme Q10 promotes normal immune function (cells involved in immune response have a high energy dependence)).
•Coenzyme Q10 inhibits the development of periodontal disease.
•Coenzyme Q10 benefits patients with Parkinson's disease.

What evidence is there for the efficacy of Coenzyme Q10?

(i)Cardiovascular disease: Coenzyme Q10 protects against atherosclerosis by inhibiting the oxidation of LDL cholesterol, inhibits inappropriate clotting of blood, lowers blood pressure, and benefits heart function in patients with myocardial infarction and cardiomyopathy (Alleva et al, 1995; Langsjoen et al, 1994; Mortensen, 1993; Singh, 1998). A meta-analysis by Soja & Mortensen (1997) and a review by Mortensen (2003) have demonstrated the benefits of Coenzyme Q10 supplementation for congestive heart failure. The potential benefits of Coenzyme Q10 supplementation in paediatric cardiomyopathy have been reviewed by Bhagavan & Chopra (2005). The use of Coenzyme Q10 in the treatment of hypertension has been reviewed (Rosenfeldt et al, 2003; Wilburn et al, 2004).

(ii) Statin therapy: Statins are drugs that reduce circulatory cholesterol levels, and are used primarily to protect at-risk patients from adverse cardiovascular events. Statins are potent inhibitors of 3-hydroxy-3-methylglutaryl coenzyme A (HMGCoA) reductase, the rate-limiting enzyme in cholesterol biosynthesis. Whilst the safety record of these drugs is generally considered to be

acceptable (particularly in limited time-frame usage), adverse effects do occur in a significant number of patients; these include skeletal muscle pain and weakness (occasionally resulting in potentially life-threatening rhabdomyolysis and renal failure), gastrointestinal disturbance, liver dysfunction, initiation or acceleration of cataracts, cognitive dysfunction and increased risk of polyneuropathy.

The inhibitory effect of statins on cholesterol biosynthesis is not selective, resulting in the inhibition of several nonsterol isoprenoid end products, including Coenzyme Q10. The statin-induced reduction in coenzyme Q10 levels has been well documented in both animal model and clinical studies (reviewed by Langsjoen & Langsjoen, 2003). Many of the adverse effects resulting from statin use can be rationalized in terms of concomitant Coenzyme Q10 depletion. Rundek et al (2004) have reported even brief exposure to atorvastatin reduces Coenzyme Q10 levels, with adverse effects on heart function (Silver et al, 2004). It is possible that the true therapeutic potential of statin drugs is being partially negated by reduced Coenzyme Q10 levels, and that co-administration of both substances would lead to an even greater reduction in cardiovascular morbidity and mortality. Oral supplementation of Coenzyme Q10 would be necessary, as the latter is not available from the diet in sufficient amounts (100-200mg/day) to compensate for the depletion in levels induced by statins. Whilst Coenzyme Q10-depletion may be tolerated in younger patients, particularly in the short term, with the trend to use statins in higher doses or in longer term treatment regimes, individuals are increasingly at risk from the effects of statin induced Coenzyme Q10 depletion, particularly the elderly and those with chronic cardiovascular disease. (Mabuchi H, Nohara A et al, 2007)

iii) Periodontal disease: Coenzyme Q10 plays a vital role in providing energy to all of the cells in the human body, as well as providing the energy requirements of certain types of bacteria.

The mouth contains both beneficial and potentially harmful bacteria. The beneficial bacteria typically rely on Coenzyme Q10 for their energy provision, while the harmful bacteria typically rely on vitamin K for their energy supply. Supplementation with Coenzyme Q10 can alter the balance of oral bacteria in favour of beneficial organisms, while providing a less favourable environment for the bacteria responsible for gum disease.

Supplementation is particularly effective in maintaining healthy Coenzyme Q10 levels in the tooth pockets of the gums, where bacteria responsible for plaque formation and gum disease may congregate.

Thus Nakamura et al (1974) reported evidence for a deficiency of Coenzyme Q10 in patients with periodontal disease. Similarly Hansen et al (1976) found significant gingival and leukocytic deficiencies (typically 20-60%) of Coenzyme Q10 in patients with periodontal disease. In randomized placebo controlled clinical trials, Wilkinson et al (1976) and Hanioka et al (1994) reported systemic or topical application of Coenzyme Q10 reported significant improvement in clinical status (e.g. pocket depth, gingival crevicular fluid flow, plaque scores) in patients with periodontal disease.

(iv) Parkinson's disease: Clinical trials have demonstrated supplementation with Coenzyme Q10 slows progression and benefits symptoms of patients with Parkinson's disease (Shults et al, 2002; Muller et al, 2003)

Are there adverse effects from taking Coenzyme Q10?

Coenzyme Q10 is generally well tolerated, with no serious adverse effects reported in long term use. Very rarely, individuals may experience gastrointestinal disturbance. There are no known toxic effects, and Coenzyme Q10 cannot be overdosed. Coenzyme Q10 is not recommended for pregnant or lactating women, in whom the effects of Coenzyme Q10 have not been extensively studied. No significant interference of Coenzyme Q10 with other drugs has been reported.

How much Coenzyme Q10 should you take?

The generally recommended intake for Coenzyme Q10 is 30 to 100mg/day, taken in split doses (e.g. morning and evening) with meals, to enhance absorption. Coenzyme Q10 is routinely prescribed for the treatment of heart disease in many countries, including Italy, Scandinavia, Japan and Canada.

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The Memorial Day for Dr Gordon R B Skinner

By Elizabeth McDonagh

On 3rd May 2014, I joined a large number of Dr Skinner's family, friends, former patients and supporters who gathered at the Novotel in Birmingham for a day in celebration of Dr Skinner's life and work. We were welcomed by Dr Skinner's friend and colleague, Dr Afshan Ahmad and by Dr Skinner's sons and daughter. The morning's events comprised a number of short speeches by his friends, family and colleagues, about Gordon Skinner's life:

The early years
The path towards academic excellence
Life in obstetrics and gynaecology
Life at the top of the vaccine research field
Man of many talents and
Family man at heart.

After lunch, Dr Barry Durrant Peatfield and Sheila Turner of Thyroid Patient Advocacy UK spoke about Dr Skinner's thyroid work. Dr Skinner successfully treated over 5,000 patients for undiagnosed hypothyroidism. Many of his patients had been diagnosed with ME. Dr Skinner worked mainly in a 'tertiary care' setting, helping patients who had previously received unsuccessful and inappropriate care from their GPs and endocrine specialists. At much personal cost, he tried to influence the medical authorities to improve diagnosis and treatment for all thyroid patients. We were shown a video of the speech given by Dr Skinner, just a month before he died, to the 2013 Conference of Thyroid Patient Advocacy UK.

Before working with thyroid patients, Dr Skinner had an outstanding career in virology, developing a number of vaccines including one for genital herpes - 'The Skinner Vaccine'. Fiona Skinner told us about the work of The Vaccine Research Trust, the Charity founded by her father. The family is determined to carry on the work of the Trust in tribute to Gordon and as his legacy.
http://www.vaccineresearchtrust.com/

Dr Afshan Ahmad has since written on behalf of Dr Skinner's family, to thank all those who attended the Memorial Day and who generously gave donations to The Vaccine Research Trust.
Elizabeth apologises to readers for an error in her Obituary for Dr Skinner in the last issue of Pathways. Dr Skinner died on 26th November 2013, not 26th December as stated.

The Medical Record Summary Sheet.

If you have a DWP form to fill out, please ask your doctor to print you out a copy of your medical record summary sheet. This document contains much of the information needed in the early pages of DWP forms and other forms and simplifies the procedure. This is something most GP Practices can print from their records. This is at most two or three sheets of paper. This contains the following information:*

- Your name and address
- Your current ongoing condition(s)
- The results of any tests
- Any current ongoing treatment
- Repeat prescriptions.

Although many surgeries provide this information free, your doctors may charge for this information. When they do it is usually around £10-£15.

*** Please note: this is NOT the same as your complete medical history which some surgeries provide by mistake at great cost.**

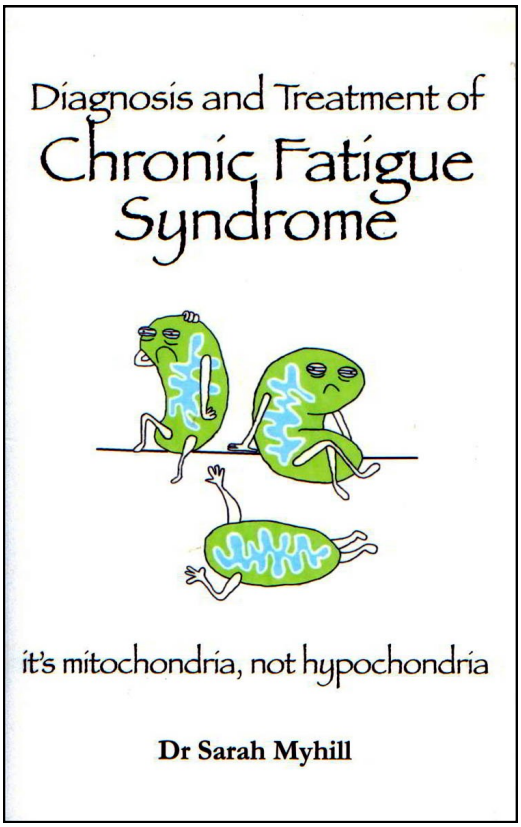
Book Review: Diagnosis & Treatment of Chronic Fatigue syndrome

By Dr. Sarah Myhill IBSN 987 78161 034 3

Until a few years ago, Sarah held a clinic in Mansfield which many Leger ME members attended. After a horse-riding accident Sarah decided to concentrate he practice near her home in Knighton, South Wales. Some Leger ME members still consider the stress of the journey to South Wales well worthwhile

We have 'mitochondria' in each and every cell in our bodies. They are the powerhouses of our cells - 'the engine of the car', as Dr Myhill says - essential for the production and management of energy at cell level. If they go slow, we go slow.

Dr Sarah Myhill, together with Dr John McLaren-Howard of Acumen Laboratory and Or Norman Booth of Mansfield College Oxford, has spent many years studying the relationship between mitochondrial malfunction and one of the commonest problems seen by GPs in the UK ¬fatigue. Thanks to their research findings we can now measure mitochondrial activity and relate it directly to clinical symptoms - they found patients with the worst fatigue had the worst mitochondrial function and that objective measurement of function correlated with subjective measurement of symptoms as both improved with treatment.



With mitochondrial function pin-pointed as the central problem in Chronic Fatigue Syndrome, Dr Myhill explains what this implies for diagnosis and treatment. For sufferers who wish to help themselves, and for health practitioners seeking an evidence-based approach, she sets out the package of lifestyle changes and supplements that she has developed over many years in collaboration with thousands of patients.

Regular Pathways readers will be aware that we often carry features by Dr Myhill. Although Pathways has carried much of the content of this book in short features, Sarah goes on to describe her ideas in greater depth and much more detail. There are contributions from many other supporters who have supported Sarah during recent years in various ways.

For the typical Leger ME member this book provides an insight into Dr Myhill's background and her strategies for treating ME/CFS/FMS. She is the lead in the mitochondria model of chronic fatigue, and many of her private practising colleagues follow and use her ideas. To date, I have not seen any alternative mitochondria model. I have, however, seen other references to mitochondrial abnormalities in other health conditions, but as with ME/CFS in the NHS general practice these are not taken into account. To make her knowledge, her unique position and experiences with ME/CFS available, Sarah offers an outreach service for those who cannot attend her clinic personally. Further to this she offers a range of food supplements and medicines to her patients. She also offers various tests some of which are not available on the NHS. This book details all these services as well as providing much information to the background of these tests.

For anyone who is a Health Professional, there is a limited listing reference and resources. Much of the information is from Sarah's own experience and practice.

Verdict: Well worth reading *****

We have purchased a copy for the Leger ME Library. It will be available via Susan, our Librarian .

A significant achievement

The first 1 million of funding awarded by ME Research UK represents 35 specific biomedical projects, the results of which have now been published as 58 research papers in peer-reviewed scientific journals. In fact, we have been able to fund more specific research projects on ME/CFS than any other single organisation in the world outside the American continent and this is thanks to generous donations from patients, their families and friends, and other ME organisations (such as the Irish ME Trust and the John Richardson Research Foundation). Our research has taken place at institutions in the UK and overseas and has involved many of the systems of the body.

To date, the most important findings have centred around the autonomic nervous system, which controls some core body functions such as heart rate, digestion and breathing; the immune system, which protects us from infection; the circulatory system, particularly the heart and blood vessels which supply oxygen to tissues; and the musculoskeletal system, which is a source of pain and fatigue for many people with ME/CFS.

As the body works as a single functioning unit, however, the research findings on one system of the body can also apply to another (immune cells are carried in the blood circulation, for instance). This is why different aspects of a particular study are sometimes mentioned under different headings in this booklet. It is worth remembering that without our involvement, impetus or funding (alone or with partners) most of the studies described in this booklet would never have taken place.

For instance, Professor Julia Newton's research on autonomic dysfunction at the University of Newcastle would not have begun and flourished into the much larger programme we see today; and the Vascular and Inflammatory Diseases Research Group at the University of Dundee would not have uncovered the range of abnormalities in blood and blood vessels. Similarly, Prof. Jo Nijs' programme in Belgium that is focused on exercise, immunology and its consequences, as well as single investigations such as the exploration of retrovirus in Swedish patients or the experience of pain in Scottish patients would not have been instigated or completed. Of course, we've done much more than simply fund research projects; after all, our mission is to energise research. We act as an information resource for researchers, healthcare professionals and serious journalists.

We also try to raise awareness of the plight of people with ME/CFS, bringing the illness to the attention of a wide range of audiences, including media and government, though public talks, conferences, articles and social media. However, we never lose sight of the fact that research is our core function, and, with your help, building on the projects we have funded to date, we intend to continue commissioning and funding biomedical research projects across the world.

North of Doncaster *Personal comment by Trevor Wainwright*

The Town of Terre Haute pronounced 'Tair Hote' is a small town in Indiana just over the border from Illinois situated on the Wabash River It was a place I have had an interest in for a while and this year I decided to pay it a visit, taking time out between poetry events on my annual Texas poetry tour. Unlike other towns, it wasn't mentioned in my Rough Guide to America but I went anyway.

Driving up there from Texas one thing I noticed was the lack of iron bars on the vending machines in the rest areas, also the appearance of coffee machines the cost of which got cheaper the further we went, to as low as 50 cents. The town lies to the left of Interstate 70, although coming off at its junction with West National Avenue the old Interstate 40 will take you to the centre of the town, the road which initially opened the West for settlement,

To the right of I-70 lie motels, eating places, shopping malls and petrol stations. Again a change–in Texas and other states it's prepay, here it was fill-first-pay-later. Coming off to the left is the main street running from East to West mainly food houses from different countries. Prior to going along the main street which is the old Highway 41 (a major North - South route and designated part of the Federal Highway system in 1926) if you take a left turn it brings you to a riverside walk known as Fairbanks Park, a pleasant walk where an information board tells of the town's origins.

Its name means "High Land". Occupied by Indians, French and later Americans who realised its strategic value, it was ceded to the US by the Indians in 1809, the town being platted (mapped to scale) in 1816. Driving further takes you into the residential area of the town, away from the commercial areas, where its traditional wooden houses of varying designs gives it a tranquil air. Children play happily outside these houses.

The town began to develop prior to 1860 largely thanks to transportation –the Wabash River, the building of the National Road (now U.S. 40) and the Wabash and Erie Canal linked it to the world and broadened the city's range of influence. The economy was based on iron and steel mills, hominy plants and, late in the 19th century, distilleries, breweries and bottle makers. Coal mines and coal-operating companies developed to support the railroads, yet agriculture remained predominant, largely due to the role of corn in making alcoholic beverages and food items.

With steady growth and development in the later part of the 19th Century, the vibrant neighbourhoods of the city benefited from improved fire protection, the founding of two hospitals, a dozen churches and a number of outlets for amusement. Terre Haute's position as an educational hub was fostered as several institutions of higher education were established. The city developed a reputation for its arts and entertainment offerings. Grand opera houses were built that hosted hundreds of operas and theatrical performances. It became a stop on the popular vaudeville circuit.

In other developments over the years, railroad overpasses eased traffic congestion, law enforcement strengthened, and several national and state awards for volunteerism and citizen participation boosted local pride. None more so that the Native Indian museum and nearby nature reserve on the Eastern outskirts of the town, where solitude and history combine to make an interesting time away from the noise of the town. New areas are also being planned and existing ones extended.

The home of Indiana State University was built on the site of the birthplace of Eugene V Debs, one of its famous sons. Born into a wealthy family, he dropped out of public school at 14 and took on various jobs before joining the Union movement, becoming a leader and fighting for better conditions for the working man. As he told an audience in Detroit in 1906, he was not wholly comfortable with his standing as a leader.

"I am not a Labour Leader; I do not want you to follow me or anyone else. If you are looking for a Moses to lead you out of this capitalist wilderness, you will stay right where you are. I would not lead you into the Promised Land if I could, because if I led you in, someone else would lead you out. You must use your heads as well as your hands, and get yourself out of your present condition". He married Kate Metzel on June 9, 1885. Their home still stands in Terre Haute, preserved within the campus of Indiana State University.

Terre Haute is a town that honours its fallen sons by dedicating streets such as Memorial Way in their name. Outside one of the town's most prominent buildings, The Vigo Valley Courthouse, there are war memorials dedicated to the fallen of various wars and prisoners of war missing in action, from the civil war to the present day. This shows the town is determined that its sons that never returned shall not be forgotten. Perhaps though, the most moving monument is a poem called "The Wall" by Dr C David Hay, a retired dentist and international poet who on seeing the Vietnam Veterans Wall in Washington DC was inspired to write one of his most famous poems. The poem was engraved on a metal plaque and added to the Vietnam Veterans Memorial on the Vigo County Courthouse lawn in 2012. It was another of Terre Haute's famous sons that brought me there–strange though, not many Americans know it or even him. His name is Max Ehrmann, one of the 20th century's greatest philosophers and poets, also a friend of Eugene Debs and author of the timeless classic 'Desiderata' which many agree is a guide to living outside the confines of religion and other ordered lifestyles.

In the next issue I shall be writing about him and his inspiration to me.