Price £ 5.00 (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 No 74.

Winter 2022 Edition.

Featuring more about cholesterol problems and ME/CFS

You write in: *A selection from the pathways postbag*

Carolyn writes: Here is a top tip for keeping bags of salad leaves fresh through Christmas. I sometimes used buy fresh leaf salad from the Supermarket. However once the bag is opened, I used to find that leaves became unfit to eat within a day. The other day I came across a simple tip that keeps the leaves fresh for longer. It works like this:-

The moment you're home or, if you shop online, the delivery man has been pop open that bag of salad leaves. Take a piece of paper towel from the roll and slide it carefully in and down the side of the bag. The paper towel will absorb moisture that would otherwise lead your leaves to rot. This will help the salad leaves to stay fresh for days longer, giving you more time to use up the bag. Be sure to change the paper towel each day for best results

Some people find it helps to close the bag with a peg but I keep my bag of salad leaves in the salad drawer at the bottom of the fridge and find it best just to leave the bag open. I find that this method adds a good four days of freshness to lettuce leaves. Ideal for over Christmas when the shops are closed.

Gwengi Writes: I would like to share my experience about PIP interview over the phone.

Oh my, it wasn't easy or pleasant. I had a previous phone call to check I would be available on the day and time (can't remember if it was one or two days before). The session lasted over two hours... I requested a break after an hour as I was getting tired which was offered and granted.

She kept asking me the same question in different ways and would fire one question in particular about the last time I saw the doctor which fortunately for me, I had a face to face with six days before. I had blood tests 4 days before the interview and of course future appointments at the hospital was important.
Something I would recommend to anyone else in the future that no one should admit they have a pet. I was GRILLED over having a cat.

If you have a car they want to know where you go and how often. There were a lot of questions on food shopping and your finances... even if you don't do internet banking it's better to admit to it. She asked about cleaning the house, food preparation including tools that help and bathing particularly about cleaning my the feet and personal hygiene and how often you change your clothes. obviously how far you can walk (with aids) and pain threshold.

It is best to have in front of you a prescription list because I was questioned strictly on that and some errors were made by her (maybe deliberately) which I was able to correct and some drugs she didn't even mention and I told her what she had left out. Thank goodness I have the forethought and I had the prescription information in front of me

I was so exhausted after I couldn't stay awake and had to sleep. It took me three days to get over the ordeal. She told me it would be 8 weeks before I would hear but since then I have received an email from PIP that it would be 6 weeks. I hope you find this helpful and can pass on to others.

This is a typical experience of our members when a PIP claim is disputed. Another oddity was the telephone call from Glasgow. We think that this is a hangover from Covid 19 lockdowns when face to face interviews were stopped. Quite often we received reports of telephone calls from allow over the country. Before a PIP claim is sent in you have to understand that you may have an ordeal like the above if it is disputed. Contact our office for further guidance if you are filling out any DWP form.

The outdoor Christmas Tree in Sprotborough

Welfare Rights Matters

With thanks to Steve Donnison from Work and Benefits

Autumn Budget Details

The good news is that there will be no means-testing of PIP and DLA, in spite of fears that this might be introduced. Benefits will go up by 10.1% next April, in line with the September 2022 Consumer Price Index. Given the hints that benefits might only rise by 5% in line with wages, this is also very welcome news. Whilst the DWP has yet to publish the benefits rates for the coming year, we do know that both income-related and contribution-based ESA are included in the 10.1% increase, as are all the main benefits covered by this site, such as PIP, DLA, and UC. The bad news is that inflation is already running at 11.1% and uprating does not apply until next April, so this is already a real terms cut.

Worse still, disabled claimants spend a much larger part of their income on food and energy, which are particular drivers of inflation at present. The real rate of inflation for many claimants has been calculated as between 14% and 20%, so a 10.1 increase actually represents a very serious cut.
In addition, Local Housing Allowance rates will not be increased, meaning rent rises could be extremely hard to manage. There will, however, be more cost of living payments next year. But for claimants on contribution based benefits there is scant comfort here.

The government says that in 2023-24:

- Households on means-tested benefits will get an additional £900
- Pensioner households will receive an additional £300
- Individuals on disability benefits will receive an additional £150

These additional sums are clearly good news, for at least some disabled claimants, but they will not be enough to make-up for the increased costs most claimants will be facing in the coming year.

ESA To UC Forced Migration Postponed

The government is pushing back the forced migration of claimants from income-related ESA to UC to 2028. The transfer was due to be completed in 2024, with an impossible target of 2.5 million claimants being moved in that year. The fact that it is being delayed is more good news.

The downside is that many thousand more claimants will now be subject to natural migration because of a change of circumstances, meaning that they will lose out on the transitional protection that managed migration claimants receive. Claimants receiving child tax credit are not included in this postponement.

Beware Cruelty of UC System, Warning

The Disability News Service (DNS) has published a collection of stories highlighting the cruelty inflicted on disabled claimants by Universal Credit (UC). One article covers the death of a disabled woman left traumatised by the daily demands of the UC system. The DWP had been told of her mental distress, suicidal thoughts and fear of the department and the universal credit system, but this did not prevent them hounding her.

Rebecca (not her real name) took her own life just four days after being told she would need to attend a face-to-face meeting with a work coach. Another article covers the struggles of disabled claimant Philip Manion who saw his income from UC cut from £1,260 to £500 a month because of a mistake by the DWP. When he tried to attend a meeting about the issue, he was removed from the job centre by a gang of seven security guards because he was unable to log into his online journal from his mobile phone. He was then recorded as having failed to attend the meeting and his UC was completely stopped. Former nurse Shirley Rudolph spent 10 years caring for her husband and had been placed in the limited capability for work category due to generalised anxiety disorder.

In July Rudolphs husband died and she told her work coach that she would be unable to attend a scheduled meeting because she was arranging her husband's funeral. The work coach expressed no sympathy whatsoever, delayed the appointment for just a week and sent Rudolph a job application to complete. As a result, Rudolph ended her universal credit claim and is surviving on her NHS pension. In a further article entitled Universal credit: €˜Chaos, fear and preventable deaths DNS interviewed disabled activists who talked about how the cruel system hounds disabled claimants into complying with strict rules. One activist explained:

The journal seems to work one way only, with the claimant's communications simply ignored. So, I am frequently left in state of high anxiety even though I am in the limited capability for work-related activity group due to mental distress.

So, while it is good news that the forced migration of ESA claimants to UC has now been postponed, the system is still urgently in need of reform - for the sake of all those claimants who are already subject to its cruelties.

Second Cost of Living Payment, Have You Had Yours Yet?

The comments from people who have not received their £150 disability cost of living payment have slowed to a trickle, so we are now asking if you have received your second cost of living payment. The target for payments was between 8 and 23 November, except for tax credit claimants who should be paid between 23 and 30 November. The second cost of living payment is the £324 sum that follows on from the £326 paid in July 2022. Generally, administration of this set of payments seems to have gone more smoothly than the disability payments, but we still like to track whether any readers are missing out.

Automatic 12 Month Extensions of PIP Not Happening Yet

In a sign of continuing chaos, the DWP has gone back on its undertaking to automatically extend PIP awards by 12 months when a review is due, the welfare rights workers site Rights net has reported. It now seems that the scheme will not be up and running until early to mid-December. No reason for the delay has been given, though the DWP did say that anyone needing a statement before this can contact the helpline 0800 1214 433 and we will issue a certification of entitlement for proof of their PIP award. Otherwise, claimants do not need to contact us unless their circumstances change. There are very few deadlines that the DWP cannot manage to miss or schemes they cannot mess up. However. they continue to do so with impunity, whilst harshly punishing claimants for the smallest mistake.

New Uprated Disability Benefits Rates from April 2023.

ATTENDANCE ALLOWANCE	**Higher rate** Increased by £9.35 to **£101.75** **Lower rate** Increased by £6.25 **£68.10**
DISABILITY LIVING ALLOWANCE	**Care Component** **Highest** Increased by £9.35 to **£101.75** **Middle** Increased by £6.25 to **£68.10** **Lowest** Increased by £2.45 to **£26.90** **Mobility Component** **Higher** Increased by £6.50 to **£71.00** **Lower** Increased by £2.45 to **£26.90**
PERSONAL INDEPENDENCE PAYMENT	**Daily Living Component** **Enhanced** Increased by £9.35 to **£101.75** **Standard** Increased by £6.25 to **£68.10** **Mobility Component** **Enhanced** Increased by £6.50 to **£71.00** **Standard** Increased by £2.45 to **£26.90**

Craft Corner at Christmas

Although we have not featured the craft group work for a while, over the year they have been busy making things for Christmas. Here is a selection of the work. If anyone is interested in purchasing an item please contact the member concerned via the Leger ME Facebook page or contact the Office.

Kerry. D

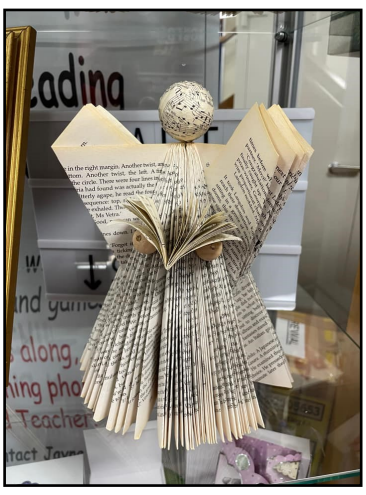
Here is sample of Kerry's Christmas ornaments.

Anne. F.

Here are samples of Annes Needlecraft.

Claire. P.

Here are examples from Claire in recycling books.

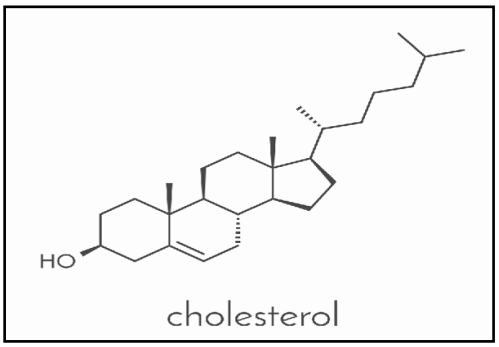


Cholesterol - The Whys and Wherefores. A nutritional Approach

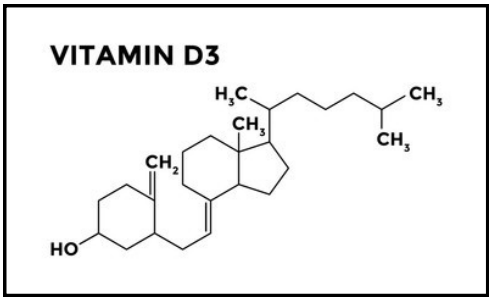
With thanks to Dr. Sarah Myhill

There is a general belief that a high fat diet results in high cholesterol, which results in arterial and heart disease. The evidence to support this theory is poor - the two largest culprits in arterial disease are refined carbohydrates (including sugars and fruit sugars) and high blood pressure from adrenalin - the stress hormone. The commonest cause of stress is rapidly falling levels of blood sugar, but any stress - lack of sleep, financial, emotional stress will also result in adrenalin.

What is cholesterol?



Both cholesterol and Vitamin D3 have similar structural formulas.. See text



Cholesterol is an essential molecule without which we would all be dead. It is an integral part of all cell membranes on which all metabolic activity takes place. Cell membranes are made up of one third polyunsaturated fat, one third saturated fat and one third cholesterol, of which 80% comes from the liver, 20% from diet. It is the raw material from which many essential molecules are made including hormones, in particular the adrenal and sex hormones, serotonin receptors (which help protect us against depression), vitamin D (through the action of sunshine on cholesterol in the skin - vitamin D is highly protective against heart disease and cancer), bile salts (essential for digesting foods) and so on. The highest concentrations of cholesterol are found in breast milk, where it is essential for infant nourishment and brain development.

Cholesterol comes into play in healing and repair of blood vessels. Blood vessels have a delicate lining and are at the mercy of turbulent blood flow. Turbulence is created where blood vessels divide and blood pressure is high. In this event the lining becomes damaged and has to be repaired. The first sign of repair is a fatty streak, then plaque formation and plaque stabilisation.

Cholesterol is a fat and can only be carried in the bloodstream wrapped up in a protein carrier. The problem is that at the site of healing there is potential for damage by oxygen (because the process of repair is carried out by the immune system, which produces free radicals when it is busy).

If cholesterol becomes oxidised then it is damaging to blood vessels. LDL cholesterol is the oxidised version - often referred to as the "bad" cholesterol. HDL cholesterol is not oxidised (the technical term is "reduced"), hence often called the "good" cholesterol. A happy ratio of "good" to "bad" cholesterol is achieved by antioxidants, which keep cholesterol in this "reduced" state. These anti-oxidants are: (PON 1 - interestingly this also protects against organophosphate poisoning!), peroxidase (needs glutathione and selenium), and superoxide dismutase.

What measure should you look at?

So, do not be satisfied with just being told that your total cholesterol is whatever. There are two much more important results to know. The first is the ratio between so-called good cholesterol, HDL, and so-called bad cholesterol, LDL. Good HDL means good levels of PON 1. The second important factor is your level of frontline antioxidants - if these are good, then cholesterol is protected from oxidation. The percentage of HDL cholesterol should be at least 20% of total cholesterol. People doing high fat paleo-ketogenic diets would expect to run a percentage above 40%.

This view suggests that a high cholesterol may be a symptom of arterial disease rather than the cause. It is only when LDL becomes oxidised into lipid peroxides as a result of poor antioxidant status that it may be directly damaging to arteries. There is no doubt some people do have genetically high levels of LDL cholesterol (the "bad" one) and do suffer excessively from arterial disease, but this should only occur where there is poor antioxidant status.

This also explains why low fat diets do not reduce arterial disease - indeed they probably increase arterial disease because the right fats in the right proportion are essential for healthy membranes!

We need saturated, polyunsaturated fats, and cholesterol together with omega oils 6 and 3 in the proportion 4 to 1. In other words, a balanced nutritional approach is needed..

Causes of a high total cholesterol with poor ratio.

1) Diet high in sugar and refined carbohydrate.

2) Poor antioxidant status.

3) Vitamin D deficiency. Cholesterol is the raw material which, through the action of sunshine on the skin, is converted to vitamin D. If the body perceives the deficiency in vitamin D3, and this is almost universal in our low sunshine climate, then the liver pushes out more cholesterol so that when sunshine does land on the skin there is plenty of substrate for vitamin D3 to be made. Vitamin D3 deficiency is itself a major risk factor for arterial disease - most of us do not get enough.

4) The wrong sort of exercise, causing Post Exertional Malaise.

5) Borderline hypothyroidism.30 years ago a raised cholesterol was almost routinely treated with thyroid hormones.

6) Vitamin B3. deficiency.B3 is essential for the metabolism of cholesterol and deficiencies are common. The converse is also true - high levels of vitamin B3 bring cholesterol levels down. The only problem is that the form of B3 (Niacin) which does this has a tendency to cause flushing. The body does acclimatise to this and so one needs to start off with small doses, such as 100mgs three times daily, and build up gradually. Vitamin B3 is used by NHS doctors for treatment of high cholesterol usually in combination with other medicines.

7) Iodine deficiency.

8) Low levels of DHEA.

9) High levels of trans fatty acids in the diet e.g., margarines and refined foods.

10) Copper deficiency. There is an inverse relationship between cholesterol levels and copper - so the higher the copper in the blood (so long as it is in the normal range) the lower the cholesterol and vice versa. The best test of copper is to measure superoxide dismutase (SODase), another vital antioxidant, since this is a good functional test of copper (and, incidentally, also of zinc and manganese levels). Copper, Zinc and Manganese are required to synthesise superoxide dismutase.

A high cholesterol with poor ratio of HDL/LDL may be a symptom of arterial damage, that is to say, cholesterol is being mobilised for healing and repair. So anything which damages arteries will cause a secondary rise in LDL cholesterol. Things to consider would be:

- Dairy and carbohydrates- dairy is pro-inflammatory, refined carbohydrates are high glycaemic index. A diet - the practical details help prevents obesity and Diabetes
- Exercise - the right sort of cardio workout to limits without cause a rebound.
- Stress - lack of sleep, high adrenaline lifestyle resulting in high sugar levels.
- Poor Antioxidants status - results in excessive inflammatory reactions.
- Poisoning by heavy metals, pollution- damages artery walls directly. (e.g., smoking).
- Wrong fats in the diet.

Any or all of the above could also result in high blood pressure.

Visit https://www.drmyhill.co.uk for further information.

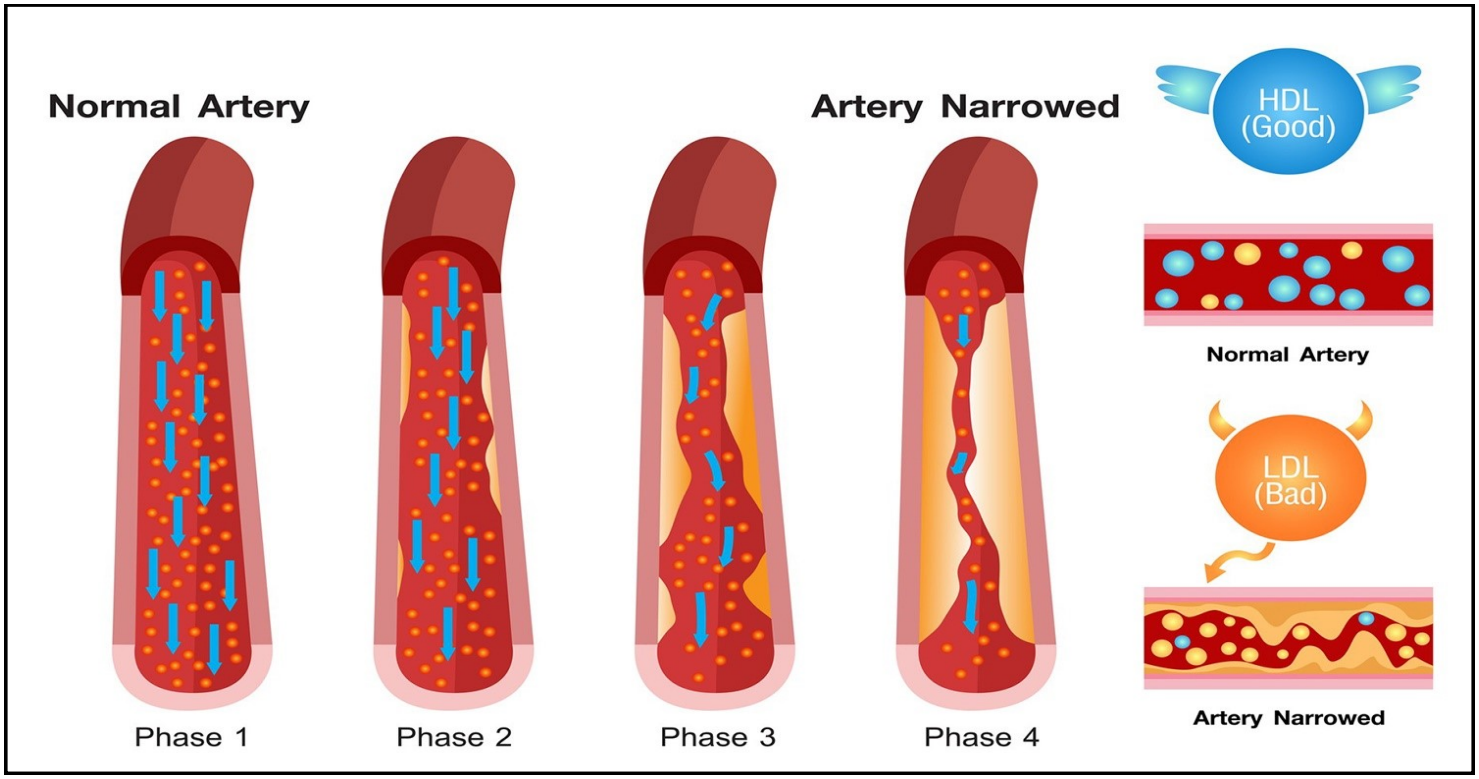
Cholesterol in the Dock or Atherosclerosis.

Above. This is a section of a copper water pipe on the outer wall over many years lime scale has been deposited the point where the amount of water the pipe can carry is very much reduced. Doncaster's water is borehole sourced, and as a consequence is a known source of hard water. This is a problem is known as the 'Cholesterol of Plumbing'.

Atherosclerosis is a potentially serious condition where arteries become clogged with fatty substances called plaques, or atheroma. These plaques cause the arteries to harden and narrow, restricting the blood flow and oxygen supply to vital organs, and increasing the risk of blood clots that could potentially block the flow of blood to the heart or brain.

Atherosclerosis does not tend to have any symptoms at first and many people may be unaware they have it, but it can eventually cause life-threatening problems, such as heart attacks and strokes, if it gets worse. But the condition is largely preventable with a healthy lifestyle, and treatment can help reduce the risk of serious problems happening.

The plaque formation is related to the levels of Low Density Cholesterol (LDL) in the blood - known a bad cholesterol. There is a second type of cholesterol know as High Density Cholesterol which is protective to some extent as so is know as Good cholesterol. The follow graphic explains the process simply.



Angina, often felt at a pain in the chest of is the most well known problems due to atherosclerosis of the coronary arteries. In this case a temporary solution is to use nitro-glycerine (yes, the explosive) in the form of a spray or tablets to make the arteries to dilate or widen as a temporary measure. The heart is the most susceptible organ for effects because for a large part of the pump cycle it squeezes blood out of its muscle tissue during contraction.

However arthrosclerosis can affect many other blood vessels. For example the blood supply to the brain may be cut off or restricted cause a transient ischemic attack or permanent stroke damage. A type of vascular dementia is associated with this mechanism. Plaques can cause damage to the main arteries and veins of the body causing problems like phlebitis. Plague can also cause kidney damage and also damage to the small veins and hepatic portal view which carries digested food from the intestine to the liver. Very often with catastrophic results. So, if possible the plaque formation should be minimised - and the key to this is keep LDL as low as possible.

BDA The Association of UK Dietitians

Food Fact Sheet

Cholesterol

This fact sheet explains what cholesterol is and how eating better can help to lower your cholesterol if it is too high.

What is cholesterol?

Cholesterol is a fatty substance found in your blood. It is produced naturally in the liver. We need some cholesterol to stay healthy. It is used to make certain hormones and vitamin D, as well as bile acids, which help digest and absorb dietary fat. Your blood carries cholesterol around your body on proteins called lipoproteins. There are two main types:

- High density lipoproteins (HDL cholesterol) take cholesterol you don't need back to the liver to be broken down and passed out of the body. It's often known as 'good' cholesterol as it removes cholesterol from the blood.
- Non-high-density lipoproteins (Non-HDL cholesterol) take cholesterol from the liver to the cells around the body. It's often known as 'bad' cholesterol because when there is too much, it can build up in your arteries. This can cause them to become narrowed or blocked and increase your risk of having a heart attack or stroke.

What causes high cholesterol?

Having high cholesterol is mainly caused by:

- eating foods high in saturated fat
- not being active enough
- smoking
- having too much body fat, especially around your middle

It can also run in families. Changing what you eat, being more active, and stopping smoking can help get your cholesterol back to a healthy level.

Lowering your cholesterol with diet

A few small changes to your diet can make a big difference to your cholesterol level.

1. Choose healthier fats

To help lower your cholesterol you don't need to avoid fats altogether. You should cut down on foods high in saturated fat and replace them with food high in unsaturated fat like vegetable oils (olive, rapeseed and sunflower oil), nuts, seeds, avocado and oily fish. *(see Table 1)*

Cholesterol Table 1

Saturated Fat	Unsaturated Fat
Full-fat dairy products	Polyunsaturated fat
Fatty meat and meat products such as pasties, sausages and pies	Oily fish
Biscuits, cakes and pastries	Sunflower, soya, corn or safflower oils and spreads
Butter, cream, ghee and lard	Flax, pumpkin and sesame seeds
Coconut and palm oils	Walnuts
	Monounsaturated fat
	Olive and rapeseed oil
	Avocado
	Nuts such as almonds, cashews, and hazelnuts

2. Look at food labels

Compare labels and choose foods with green or amber labels for 'saturates'. Foods are high (red) in saturated fat if they contain more than 5g of saturates per 100g. Foods containing 1.5g or less per 100g are low (green) in saturated fat.

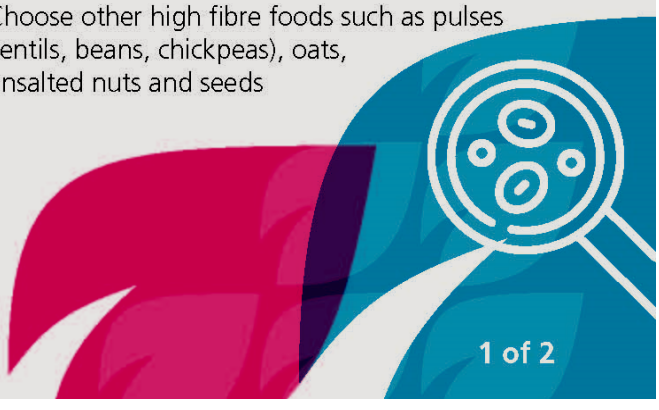
Some healthy foods that are high in fat like oily fish, nuts and oils, may be red for saturated fat. This is okay, as they contain more of the healthy unsaturated fat. Swap saturated fats for unsaturated fats.

Try the smart swaps in Table 2 to help cut back on saturated fat.

3. Eat more high fibre foods

Eating plenty of fibre helps lower your risk of heart disease and some high fibre foods can help lower your cholesterol. To make sure you get enough fibre:

- Aim for five portions of fruit and vegetables a day
- Switch to wholegrain varieties of bread, cereals, pasta and rice
- Choose other high fibre foods such as pulses (lentils, beans, chickpeas), oats, unsalted nuts and seeds



Cholesterol Table 2	
Eat less	Smart swap
Creamy or cheesy sauces	Tomato or vegetable-based sauces
Fatty meat products such as sausages, burgers, pate, salami, meat pies and pasties	Lean cuts of meat and mince Chicken and turkey with the skin removed Fish especially oily fish such as mackerel, sardines, salmon Vegetarian options like lentils, chickpeas, soya
Crisps and chocolate	Fresh or dried fruit or a handful of unsalted nuts and seeds
Full-fat milk, cheese, cream and yoghurt	Lower fat dairy foods such as 1% milk, reduced fat cheddar, low-fat yoghurt
Lard, dripping, ghee, butter and coconut oil	Vegetable oils - such as olive, sunflower, soya or rapeseed oil and their spreads

Further information

Food Fact Sheets on other many other topics including:

- Stanols & Sterols
- Heart Health
- Fat

are available at bda.uk.com/foodfacts

The British Heart Foundation
bhf.org.uk

HEART UK - The Cholesterol Charity
heartuk.org.uk

Change 4 Life
nhs.uk/healthier-families/food-facts/fat

Cholesterol Food Fact Sheet

Top Tips

- Cholesterol is a fatty substance found in your blood.
- There are two main types: HDL- cholesterol or 'good' cholesterol and non- HDL cholesterol or 'bad' cholesterol.
- Too much non-HDL cholesterol can cause your arteries to become blocked. This increases your risk of having a heart attack or stroke.
- A healthy balanced diet, being physically active, stopping smoking and keeping a healthy weight and shape can all help to lower your cholesterol.
- Replace foods containing saturated fats with those that contain polyunsaturated and monounsaturated fats.
- You can do this by choosing healthy fats such as olive or rapeseed oil, nuts, seeds, fish and avocado.
- Increase your fibre intake by choosing vegetables, fruits, wholegrains, pulses, nuts and seeds every day.

What about Plant stanols or sterols products?

If you have high cholesterol, using foods with added plant stanols and sterols has been shown to help to lower cholesterol levels. You need to eat 1.5-3g of plant stanols or sterols, in combination with a healthy diet, to see a reduction in cholesterol.

You can get this from fortified foods such as mini drinks, spreads, milk and yoghurts from both branded and supermarket own label products. If you decide to use these products, follow guidelines on the packet to get the right amount. However, they are not a substitute for healthy diet nor a replacement for cholesterol lowering medication. And if you don't have high cholesterol, these products are not recommended.

Dietary cholesterol – don't get confused!

Some foods naturally contain dietary cholesterol, but don't make a big difference to the cholesterol in your blood. These are foods like eggs, some shellfish such as prawns and crab and offal such as liver, liver pate and kidney. They are low in saturated fat and so are fine to eat as part of a healthy diet. Only cut down on these foods if your doctor or a dietitian has advised you to. To lower cholesterol, it's more important to cut down on the amount of saturated fat you eat.

Keeping lipid levels under control, especially LDL by using medicines

While is possible to keep lipids under control by lifestyle and dietary measures, there are times when medicines are needed. Many of the medicines are far better at keeping cholesterol levels down than by lifestyle and dietary strategies. There are several classes of established lipid-lowering, or antihyperlipidemic.

Statins are designed to lower LDL, the 'bad cholesterol' most strongly linked to vascular disease. Notable side-effects include myopathy and rhabdomyolysis. Some evidence suggests that statins should not be used in patients older than 75 who have no history of heart disease or stroke. Statins inhibit the enzyme hydroxymethylglutaryl-CoA reductase (HMGCR) which also deplete the respiratory chain and coenzyme Q10 which is already depleted in some people with ME/CFS. See following page. Examples of statins include atorvastatin, cerivastatin, Fluvastatin, lovastatin, pravastatin, rosuvastatin and simvastatin. All are prescription medicines except low dose simvastatin can be purchased over the chemist counter.

Bempadoic Acid is a new kid on the block. It is a prodrug which works higher up in the same biochemical pathways as statins. It is however absent in adipose (fatty) tissue and muscle cells and reputed to be less myotoxic that statins. Theoretically it has the same limitation for people with ME as statins. At the time of writing, it is ‘under probation surveillance’, and so is a prescription only medicine.

Fibrates are prescribed to reduce hypercholesterolemia in patients intolerant of or unsuitable for statin therapy. Fibrates increase HDL ('good cholesterol') and lower triglyceride levels. The fibrate-induced reduction in insulin resistance is useful when the dyslipidemia is associated with other indicators of the metabolic syndrome (e.g. hypertension and type 2 diabetes mellitus). Fibrate therapy is unsuitable for patients with low HDL levels, and treatment should be withdrawn if HDL-C levels are severely depressed soon after initiation. Fibrates are recommended as first line therapy only in patients with very high triglyceride levels. Fibrates activate peroxisome proliferator-activated receptors (PPARs), especially PPARα. They are metabolised by cytochrome P450 3A4 (CYP3A4). Examples of approved fibrates include bezafibrate, ciprofibrate, clofibrate, fenofibrate and gemfibrozil. All are prescription only medicines.

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Niacin (nicotinic acid derivatives or vitamin B3) is used as a vitamin supplement. It lowers both cholesterol and triglyceride concentrations by inhibiting synthesis; it also increases HDL cholesterol. Used as an adjunct to statin therapy, or alone if statins are not tolerated. Not to be used if the patient has a history of peptic ulcer disease or arterial bleeding. The nicotinic acid derivative akepiro can be used in similar situations. Inositol nicotinate can be used for peripheral vascular disease, but not in patients in the acute phase of a cerebrovascular accident or who have suffered recent myocardial infarction. NICE does not recommend the use of inositol nicotinate to treat intermittent claudication in patients with peripheral arterial disease. It is mainly use along with fibrates for people who are statin intolerant. Niacin is freely available as a food supplement.

Bile acid sequestrants are synthetic polymeric resins which prevent reabsorption of bile constituents from the gut, thereby acting a hypolipidemic agents. Also used for other purposes such as the treatment of chronic diarrhoea due to bile acid malabsorption and for the prevention of pruritus in patients with chronic liver disease. As hypolipidemic agents these are less effective than statins. Not to be used when blood triglyceride levels are elevated. Examples include cholestyramine, colestipol and colesevelam.

Ezetimibe inhibits the intestinal cholesterol uptake protein Niemann-Pick C1-like protein 1, a critical mediator of intestinal cholesterol absorption. Has only a modest effect as a mono-therapy but can be used adjunctively with dietary measures with or without statins to treat primary hypercholesterolemia and homozygous familial hypercholesterolemia. Avoid its use in patients with moderate and severe hepatic impairment. It is a prescription only medicine.

Phytosterols (plant sterols and stanols) are naturally occurring steroid-like compounds similar to cholesterol. Stanols are saturated sterols, having no double bonds in the sterol ring structure. Despite being effective in lowering LDL cholesterol, the benefits of phytosterol-enriched foods and dietary supplements in cardiovascular disease and overall mortality are yet to be proven. The most commonly

occurring phytosterols in the human diet are β-sitosterol, campesterol and stigmasterol; the most common stanols are sitostanol and campestanol. They are present in some food products.

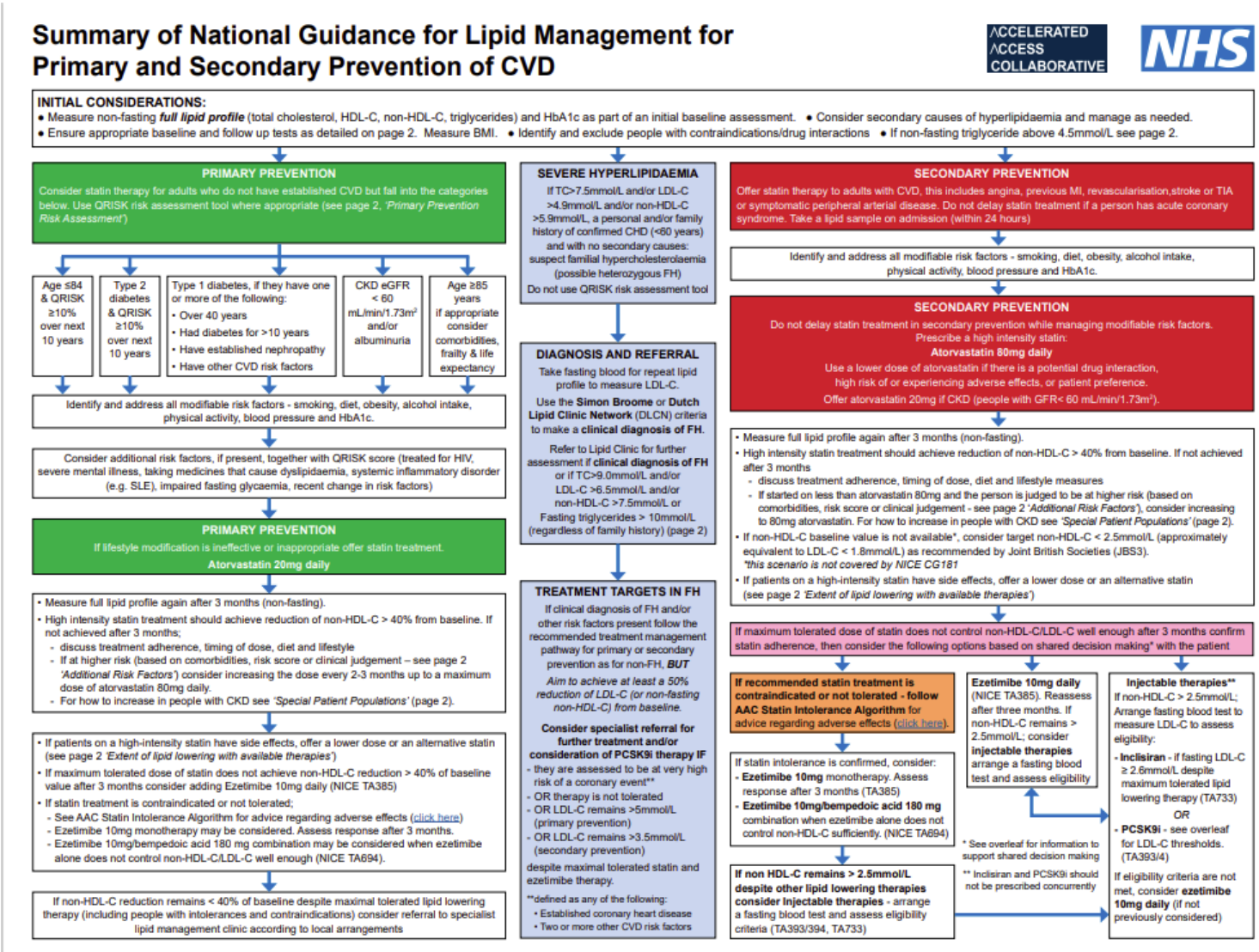
Orlistat inhibits pancreatic lipase, thereby reducing absorption of fats from the diet. Used as an adjunct to a reduced-calorie diet as an anti-obesity therapy, in obese patients suffering additional risk factors such as type 2 diabetes, hypertension, or hypercholesterolemia.

There are three main ways that cholesterol lowering medicines are used within the NHS.

1) **Primary prevention.** This strategy is for people without proven clinical disease, but who may suffer from a cholesterol related problems at a future date due to ongoing health problems. In effect this is a medical insurance policy to try and avoid at problem occurring further in future. The dose of whatever medicine is chosen is normally on lower side.

2) Secondary Prevention. This is a strategy for people with proven atherosclerotic disease like angina. previous heart attack or stoke. The dose of the medicines may be multiple and are given in higher doses. The strategy is to try and prevent the disease from progressing can causing further health problems.

3) Severe Hyperlipidemia. Here the cholesterol levels are very high and very often occur in families. There is often a family history of atherosclerotic disease. So apart from the person concerned, the family need to be checked. Usually, it needs heavy use of cholesterol control medications with unusual combinations.



Can Plaque be reversed ? Mostly no, but recent research has shown that intense treatment with pravastatin and/or rosuvastatin can partially reduce the plaque in some cases. The LDL has to be less than 1.7 mml/l for this to happen. It is quite clear that prevention is better than cure.

Recipe Corner

Make Ahead Mushroom Soufflés

A soufflé to start always gives your dinner party a 'wow' factor and this make-ahead recipe gives you more time to be the perfect host

Preparation

1) Fry the mushrooms in the butter for about 3 mins, then remove from the heat and reserve a good spoonful. Add the flour to the rest, then blend in the milk and return to the heat, stirring all the time to make a thick sauce. Stir in the cheese, season to taste, then leave to cool.
2) Heat oven to 200C/fan 180C/gas 6. Butter 8 x 150ml soufflé dishes and line the bases with baking paper. Stir the egg yolks into the soufflé mixture, then whisk the egg whites until stiff before folding in carefully. Spoon into the soufflé dishes and bake in a roasting tin, half-filled with cold water, for 15 mins until risen and golden. Leave to cool (they will sink, but they are meant to). You can make the soufflés up to this stage up to 2 days ahead. Cover and chill.
3) When ready to serve, turn the soufflés out of their dishes, peel off the lining paper, then put them on a baking sheet lined with small squares of baking paper. Top each soufflé with 1 tsp crème fraiche and a little cheese, then scatter with the reserved mushrooms. Bake at 190C/fan 170C/gas 5 for 10-15 mins until slightly risen and warmed through. Sprinkle with chives and serve.

Ingredients list

140g small button mushroom, sliced
50g butter, plus extra for greasing
25g plain flour
325 ml milk
85g gruyere (hard cheese), finely grated, plus a little extra
3 large eggs, separated
8 tsp crème fraiche
snipped chive, to serve

Nutrition: per serving:- Kcal 179, fat14g, saturates 8g, carbs 4g, sugars 2g, protein 8g, salt 0.43g

Chocolate Tiffin Tart

Ready in 55 minutes ten portions.

Preparation

1) 1Blitz the flour, butter and icing sugar in a food processor until it resembles fine breadcrumbs. Add the egg yolk plus 2 tbsp cold water and pulse until a crumbly dough forms. Turn onto a floured surface and knead until smooth. Shape into a flattened oblong, wrap in cling film and chill for 30 mins.
2) Roll out the pastry on a lightly floured surface and use to line a 34cm x 10cm rectangular loose-bottomed tart tin, leaving any excess pastry raised over the edges. Chill for 30 mins, then trim off the excess pastry and prick the base all over with a fork.
3) Preheat the oven to 200 C/fan 180 C/ gas 6 and put a baking tray inside to heat up. Line the pastry case with baking paper and fill with baking beans. Bake on the heated tray for 20 mins, until the edges are pale golden. Remove the beans and paper, and bake for a further 8-1 0 mins, until the base is crisp and golden. Leave to cool completely.
4) Put the chocolate in a heatproof bowl. Heat the cream in a small pan until almost boiling. Pour it over the chocolate, leave to stand for 1 minute, then stir with a wooden spoon until smooth and glossy.
5) Pour the chocolate mixture into the pastry case, then chill in the fridge for 20-30 mins. Scatter with the shortbread, walnut pieces, cherries and raisins, then sprinkle over the glimmer sugar. Chill for 2-3 hours more, or until set. To serve, remove from the tin and slice.

Ingredients list

200g plain white flour,
plus extra for dusting
100g unsalted butter,
chilled and diced
½ tbsp icing sugar
1 egg yolk
150g 70% cocoa dark chocolate,
finely chopped
300ml double cream
2 all butter shortbread fingers,
roughly chopped
40g walnut pieces
30g glace cherries, quartered
30g seedless raisins Y
½ tsp gold glitter sugar

Nutrition: per serving: Kcal 422, fat 32.2g, saturates 18.38g, sugars 12.6 g, salt 0.06g

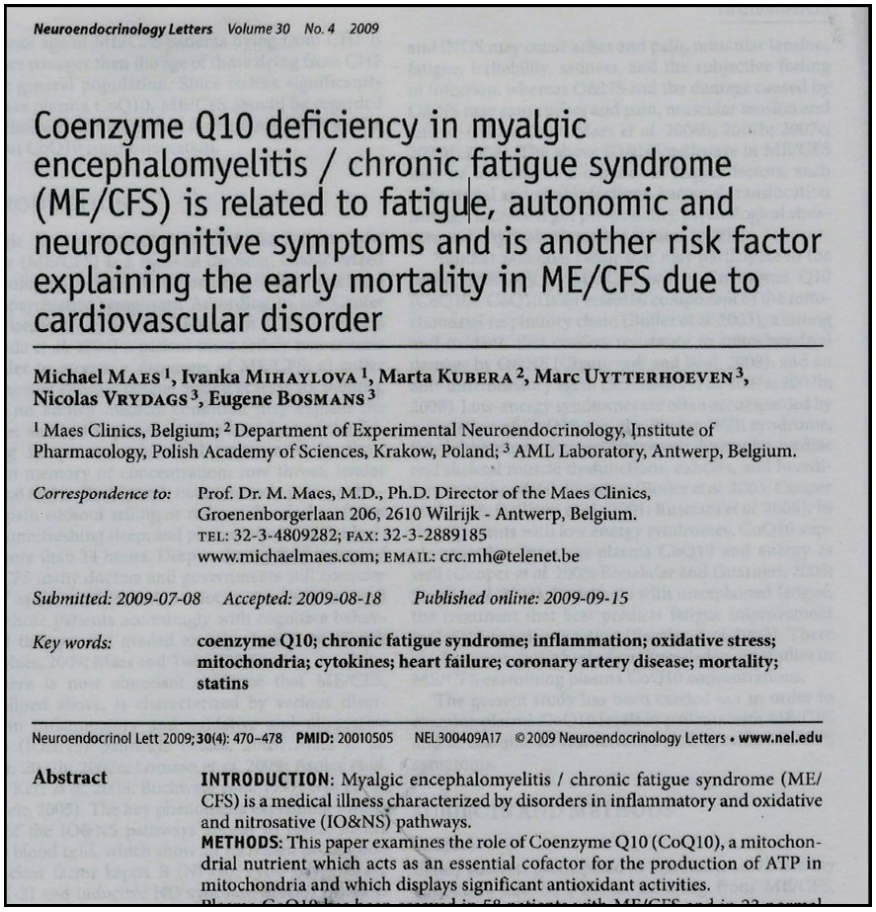
The Problem with Statins and people with ME/CFS

From Dr .Myhill, written on her website.

The interesting thing about statins is that they do reduce ones' 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, cancer, 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. Firstly, Coenzyme Q 10 - this is the most important antioxidant inside mitochondria and the main acceptor and donor of electrons. This means that mitochondria will go slow, 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 essential to maintain cholesterol in its desirable unoxidized state.

It is a combination of the above two factors which explains the devastating effect statins have on some people with muscle metabolism. People get obvious muscle soreness, stiffness, weakness and fatiguability. 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 epidemic of Alzheimer's disease we are now seeing.



Neuroendocrinology Letters Volume 30 No. 4 2009

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

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Submitted: 2009-07-08 Accepted: 2009-08-18 Published online: 2009-09-15

Key words: **coenzyme Q10; chronic fatigue syndrome; inflammation; oxidative stress; mitochondria; cytokines; heart failure; coronary artery disease; mortality; statins**

Neuroendocrinol Lett 2009; **30**(4): 470–478 **PMID:** 20010505 NEL300409A17 © 2009 Neuroendocrinology Letters • **www.nel.edu**

Abstract

INTRODUCTION: Myalgic encephalomyelitis / chronic fatigue syndrome (ME/CFS) is a medical illness characterized by disorders in inflammatory and oxidative and nitrosative (IO&NS) pathways.

METHODS: This paper examines 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.

One of the most worrying aspects about statins and people with ME/CFS was the Maes research paper which is pictures on the right. In pathways 40 and 41 I covered this topic in depth. You can download these edition from our website www.danum.me

My first encounter with the popularity of statins and the medical profession was in 2007 when I organised a professional meeting on the topic. I came back from the session feeling that all the propaganda ‘Statins were for everyone ‘ from the drug company was too good to be true.

Sure enough it was not long before reports of serious side effects were in the medical literature. I did discuss this with a colleague form the British Heart Foundation, and I was told that statins are a bit like Marmite - you either love them or hate them, and the split was 50:50. The manufacturers of one particular statin have funded research to effectively say that statin side effects are ‘all in the mind’.

Haven’t we heard that somewhere before ?

So what is the general consensus about people with ME/CFS and Statins. We asked the members about their experiences. Here is a sample of the responses I received.

Julie: "I was prescribed rosuvastatin because I have high cholesterol. Just one tablet made my fatigue and pains worse, so I just rejected the treatment."

Gwen: **"**I was prescribed a statin for high cholesterol. The side effects and were horrendous. However my doctor changed me over to a Fish oil, Niacinamide and a Fibrate. This cocktail seem to be keeping my cholesterol levels under control without the horrible side effects."

Mick: "I have a heart problems so I needed to take something. I and my doctor did a grand tour of all the statins, and experienced the various the side effects. I eventually settled on Pravastatin which seems to work for me."

Bari: I am diabetic and I have been prescribed Atrovastin. I have not noticed any side effects at all.

The latest thinking is that the Water soluble statins have less side effects than the Lipid soluble. It is the lipid soluble one that produce waste products which get into the muscle and nervous system causing the side effects. However these are most expensive but these are likely to cause the least side effects.

Statins

Water-soluble	Lipid soluble
Rosuvastatin	Atorvastatin
Pravastatin	Simvastatin
	Fluvastatin

Approximate reduction in LDL-C					
Statin dose mg/day	5	10	20	40	80
Fluvastatin			21%	27%	33%
Pravastatin		20%	24%	29%	
Simvastatin		27%	32%	37%	42%
Atorvastatin		37%	43%	49%	55%
Rosuvastatin	38%	43%	48%	53%	
Atorvastatin + Ezetimibe 10mg		52%	54%	57%	61%

Low intensity statins will produce an LDL-C reduction of 20-30%
Medium intensity statins will produce an LDL-C reduction of 31-40%
High intensity statins will produce an LDL-C reduction above 40%
Simvastatin 80mg is not recommended due to risk of muscle toxicity

In the case of primary prevention (cholesterol insurance), if you were offered a statin what should you do ? Firstly. have your Q10 levels checked. This test is available privately. If your Q10 levels are low, avoid a statin, go for another cholesterol reducing strategy. If your Q10 levels are Ok, then try a statin, preferably a water soluble one and see how you go. Just remember that all poeple with ME/CFS are susceptible to medicines side effects, so do not start at the prescribed dose. Try about a quarter of a tablet just to see how things work out and the slowly increase to the prescribed dose. If you cannot tolerate statins go back to your doctor and try something else. If you do tolerate a statin, have your Q10 levels checked from time to time.

In the case of secondary presentation where you have established atherosclerotic disease, the same applies. However statins are the only proven cholesterol medicines that stabilise the plaque or in some cases reduce it. If possible go for a water soluble statin for a possibility of reducing the plaque. In order to reduce that plaque, LDL levels needs to be 1.7 mm/l or less.

Whether or not you take a statin or any other medicines that reduce cholesterol you need to periodically check that the treatment is working. Normally at a doctors surgery the cholesterol parameters are checked by a blood sample being taken and sent off to the lab.

More recently home cholesterol testing devices have become available like the one on the right. These devices use a finger prick sample in a similarly way to how diabetics test their blood sugar. The advantage of this sort of device is that you can check your own cholesterol levels, and try different strategies and see how they work. However these devices need knowledge and skill to use them correctly. Don't forget that some things can briefly raised cholesterol levels to give a false reading. The best example is a covid 19 infection

ME/CFS and Long COVID: Questions and Answers

By Dr. Jennifer Cope, MD, MPH with thanks to Medscape

The growing number of individuals experiencing Long COVID — now estimated to be as high as 1 in 5 adults who have had COVID-19 — has brought greater attention to myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS). The two conditions share many symptoms, but their causes are still unknown. Lucy Hicks of Medscape Medical News spoke with Jennifer Cope, MD, MPH, a medical officer with the Centers for Disease Control and Prevention (CDC) Chronic Viral Diseases Branch, to answer questions on what providers should know about Long COVID and ME/CFS, how to diagnose and treat these conditions, and ongoing areas of research.

Why should healthcare providers care about ME/CFS?
ME/CFS, which stands for myalgic encephalomyelitis/chronic fatigue syndrome, is a debilitating and complex illness that affects a substantial number of people. The estimated prevalence of ME/CFS is between 836,000 and 2.5 million Americans. We do think that because of the COVID-19 pandemic, we could start to see increasing numbers of ME/CFS cases.

Do we know what causes ME/CFS?
The cause — or causes — of ME/CFS are not known, and that is one of the more perplexing and frustrating aspects of this illness. Postinfectious aetiology has long been suspected, but environmental toxins and immune system changes, chronic stress, mitochondrial defects as well genetic links could also be involved.

How is ME/CFS diagnosed?
There is no specific diagnostic test for ME/CFS; we rely on the Institute of Medicine 2015 case definition for ME/CFS. Symptoms include (1) significant limitations in the ability to participate in routine activities associated with profound fatigue lasting at least 6 months and (2) post exertional malaise (PEM), which is worsening of symptoms after physical, mental, or emotional exertion that would not have caused a problem before the illness. PEM is considered the hallmark symptom of ME/CFS. The third symptom is unrefreshing sleep. Additionally, you need at least one of the following: cognitive impairment and/or orthostatic intolerance.

What is Long COVID, and is it similar to ME/CFS?
Long COVID is currently being used as a broad, umbrella-like term: there are different subgroups of Long COVID that fall under that that title. It is not one entity, and that has certainly contributed to some of the complexities around studying it.

At one end of the spectrum, there are conditions and symptoms that are likely due to direct organ damage by the SARS-CoV-2 virus that causes COVID-19, and these include lung fibrosis, myocarditis, or kidney damage. Routine imaging and laboratory tests can identify these types of issues. Then, at the other end of the spectrum, we have syndromes that are similar to ME/CFS that include disabling symptoms like PEM, fatigue, brain fog, unrefreshing sleep, headaches, dysautonomia, mast cell activation, and orthostatic intolerance. These are often in the absence of abnormalities that are detectable through routine laboratory tests or imaging. The clinical approach for both conditions is directed toward supportive care and symptom management.

Are there notable differences between patients with Long COVID vs ME/CFS?
Long COVID does encompass a number of different subtypes, and the organ system damage we see in a subtype of people previously infected with SARS-CoV-2 is not something that is usually seen in ME/CFS. An example is someone who has lung damage from having acute COVID, and they continue to have shortness of breath and reduced pulmonary function. That is different from what we see with ME/CFS. Another difference would be people with COVID who have had disturbances to their sense of smell and taste. That was something novel that we saw with both acute COVID and in some people who are suffering with Long COVID.

What do we know about what may be causing ME/CFS-like symptoms in patients with Long COVID?
This is an area of active investigation, with the National Institute of Health's Researching COVID to

Enhance Recovery (RECOVER) Initiative being one of the largest efforts around this topic in the United States. Current mechanisms that are being postulated and investigated include persistence of SARS-CoV-2 antigens, abnormal inflammatory reactions, reactivation of latent herpes viruses, gut dysbiosis, microvascular dysfunction, and then viral-induced autoantibodies. It is a pretty lengthy list.

When would a clinician diagnose ME/CFS, and when would they diagnose Long COVID?
Starting with ME/CFS, that diagnosis is going to be made when the patient meets those Institute of Medicine case definition criteria. As of October 1, 2022, we now have a new ICD-10 diagnostic code that is specific for myalgic encephalomyelitis and chronic fatigue syndrome: G93.32. This is an advance, because previously, there was no specific code for ME/CFS. Providers had to use less specific codes like "chronic fatigue, unspecified" or "benign encephalomyelitis." We're excited because ICD-10 codes are used in so many ways, including in analyses where we try to establish the prevalence and burden of illness. If someone meets criteria for ME/CFS and it's occurring after having COVID, the clinician would use both the ME/CFS code and use it in conjunction with the post-COVID condition code, which is U09.9.

While ME/CFS has criteria outlined for diagnosis, we're not there yet with Long COVID. This is really a clinical diagnosis, where certain symptoms are occurring after having COVID. One thing to note is that a positive COVID test is not necessarily required. It obviously supports the diagnosis, but a clinician can make a Long COVID diagnosis based on the clinical symptoms that the patient might have had or if they had an exposure to someone with COVID.

What are the current treatments available for ME/CFS and Long COVID?
For both conditions, it's really focused on targeting the most troublesome symptoms. Clinicians can turn to what is already known about what works for some of the symptoms of ME/CFS and apply that to patients with Long COVID who have similar symptoms. The American Academy of Physical Medicine and Rehabilitation has put out consensus statements that are focusing on the common types of symptoms of Long COVID: the fatigue, cognitive impairment, and some of the breathing difficulties. They're drawing from strategies that have worked for ME/CFS and other conditions for those symptoms.

How can growing research in Long COVID contribute to our understanding of ME/CFS?
There are notable similarities among the presentation and the symptoms of both conditions. ME/CFS might have an infectious trigger, and we know that's the case for Long COVID as well. Findings from studies on Long COVID on the causes and therapies that might work can then be potentially investigated and applied to patients living with ME/CFS. If there have been any positives to this pandemic, one is that Long COVID has shone a light on ME/CFS and raised the awareness of the condition.

Are there any promising areas of research in ME/CFS?
Given how little we know about the causes, there are really a lot of avenues worth investigating. Studies on mitochondrial dysfunction, neuroinflammation, glial activation, endothelial dysfunction, and immune dysregulation are all being pursued. Another crucial area to highlight is identifying biomarkers for diagnosis and monitoring the impact of therapy, as well as studies characterizing the mechanism of Post Exertional Malalise (PEM). PEM continues to be a hallmark of ME/CFS, and it's one of the most baffling and disabling symptoms that patients face.

One area that is a bit different, but also really important, is researching models of clinical care that will allow healthcare providers the time and support necessary to provide the comprehensive care that patients with complex chronic illnesses like ME/CFS and Long COVID really need.

Have there been any areas of ME/CFS research that have been blind alleys?
We would not say that there's truly any research that hasn't been useful, because even negative studies contribute to the overall knowledge of the illness. Sometimes, there are problems that occur when there are exciting findings that might be overinterpreted. One example is research that detected the virus XMRV in patients with ME/CFS. This was very exciting, because we thought we had identified one thing that we thought caused ME/CFS. Then, it ended up being determined through comprehensive collaborative study that XRMV was a laboratory contaminant and not the aetiology of ME/CFS. But overall, you know, even these detours have had the advantage of raising awareness about ME/CFS and enlisting more investigators to study the condition.

Leger-ME:

www.danum.me

Mutual Support and Signposting Group for people suffering with Fatigue Syndromes *

Meetings are held,
on the 3rd Thursday
of each month between
1pm and 3:30pm
@ The Linney Centre,
Weston Road (behind the shops),
Balby Doncaster DN4 8NF.

1 to 1's sessions are available by appointment only.
For more information contact: Mike at Leger ME, Doncaster
mike@danum.me.uk 01302 787353 (Please leave a message)

*We have a policy of not accepting unregistered calls,
with all calls being logged.

* Fatigue Syndromes are long-term health conditions and where Fatigue and Pain are the primary symptoms which include ME/CFS, Post Viral Fatigue Syndrome, Fibromyalgia Syndrome and Post/Long Covid Fatigue Syndrome.

Home Finance : Tips For Managing as Energy Bills and Living Costs Rise.

With thanks to ME Essential

With energy costs on the up and up, ideas for how to save money and keep warm through the winter is at the forefront of everyone's mind. Nothing will be able to solve all the issues, but they may offer some help on how to cut energy use and save some money.

Wear More Clothes for Winter

This is so obvious but it's amazing how many people don't think to do it. An extra layer will warm you up in no time and at least put off the need to turn the heating up for a bit longer. Wear thermals and slippers and if your hands get cold, try fingerless gloves as these will keep you warm and also allow you to do things.

Blankets

Keep a cosy blanket nearby and snuggle up in it whenever you feel remotely cooler. Do not allow yourself to become cold before you use it, wrap up as soon as you feel the twinge. With vacuum flasks every time you boil a kettle, pour the left-over hot water into a flask so that you don't need to boil another kettle the next time you want a cuppa.

Cooking

Microwaves, slow cookers, and air fryers are cheaper to use than the oven. If you do use the oven to make a meal, cook in bulk and then use the microwave to reheat it another day. Think carefully about what you are cooking - healthy soups can be made in big batches, as can casseroles. Don't look in the oven baking! Every time you look, the temperature drop 10°C, making your oven use more energy to bring the temperature back up.

Check the larder, fridge and freezer and try to use up as much as you can, making as many different meals as possible. It will cut down your shopping bill on the run-up to Christmas, reduce food waste, and allow you more flexibility for buying festive treat. Make a menu 'plan, write the shopping list and stick to it. You will save a small fortune by not being tempted by 'offers' that are only really offers if you need them. Turn off the oven a few minutes before cooking time runs out. Your food will continue to cook without using the extra electricity.

Laundry

Only wash when there's a full load and turn the temperature down. Laundry will wash just as well at 30 degrees as it does at 40. Choose a shorter cycle and wash on fine days so you can hang it all outside to dry. The tumble drier is the worst of all culprits for increasing the energy bill, so forget you even own one unless there's an emergency.

Lights And Electrical Appliances

If you are not in the room turn the lights off. If you're not using it, turn it off at the mains and pull the plug out - anything kept on standby is costing you money.

Only charge your devices for as long as they take to charge. Rather than leaving them charging all night, put them on to charge when you're awake so you can keep an eye on them. Change your lightbulbs to LED which give the same amount of light but use a fraction of the power in doing so

Furniture

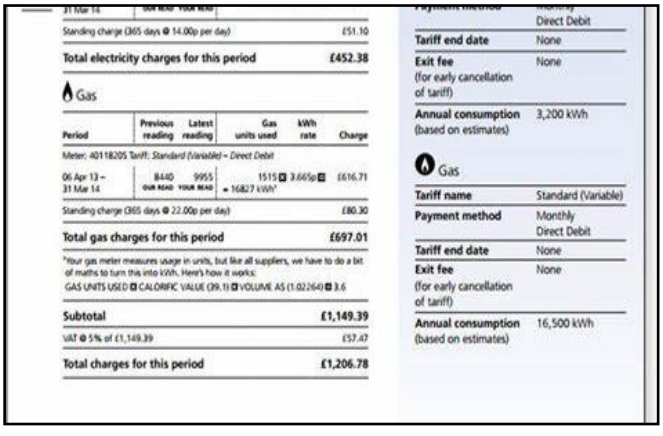
Move the sofa if it is blocking the radiator. Even moving it away by an inch or two will help. If the sofa is a long way from the fire, move it closer (be careful how close you go through). Put draught excluders on all your doors, especially the external ones, to keep the warmth in. Your rooms will be much cosier by adding such a simple item.

Curtains and windows

As soon as the sun goes down it is time to close the curtains and blinds. Windows get cold and curtains keep the warmth in. Use thicker curtains for winter to keep in the heat. If you don no have double glazing, check your doors and windows for drafts and if necessary, install draft excluders. It possible to fit temporary double glazing.

Heating controls and Thermostats

Set your thermostat to 18° for bedrooms and 21on rooms which you regularly use. Every very degree of extra heating or cooling will increase energy usage 3-4 %. Setting your thermostat to a lower temperature than normal will not allow your home to cool naturally. Install a programmable thermostat will automatically adjust the temperature according to your preferences. Don't forget to have your central heating system serviced regularly. Fan hearers should only be used in an emergency. Just remember that electricity is about three times more expensive for space heating than gas.



Standing charge (365 days @ 14.00p per day) £51.10

Total electricity charges for this period **£452.38**

Gas

Period	Previous reading	Latest reading	Gas units used	kWh rate	Charge
Meter: 4011820S Tariff: Standard (Variable) – Direct Debit					
06 Apr 13 – 31 Mar 14	8440 OUR READ	9955 YOUR READ	1515 × 3.665p = 16827 kWh*		£616.71

Standing charge (365 days @ 22.00p per day) £80.30

Total gas charges for this period **£697.01**

*Your gas meter measures usage in units, but like all suppliers, we have to do a bit of maths to turn this into kWh. Here's how it works:
GAS UNITS USED × CALORIFIC VALUE (39.1) × VOLUME AS (1.02264) × 3.6

Subtotal **£1,149.39**

VAT @ 5% of £1,149.39 £57.47

Total charges for this period **£1,206.78**

Direct Debit

Tariff end date	None
Exit fee (for early cancellation of tariff)	None
Annual consumption (based on estimates)	3,200 kWh

Gas

Tariff name	Standard (Variable)
Payment method	Monthly Direct Debit
Tariff end date	None
Exit fee (for early cancellation of tariff)	None
Annual consumption (based on estimates)	16,500 kWh

Finally, Understand Your Energy Bill

Understanding the information on your energy bill can go a long way to helping you get to grips with your energy use at home. Your energy supplier will have guides to what everything on your bill means and it's worth knowing what everything means, so you will be in a better position to control the costs.

Is Vitamin D3 all its supporters claim?

With thanks to Medscape

Hello. This is Dr JoAnn Manson, professor of medicine at Harvard Medical School and Brigham and Women's Hospital. I'd like to talk with you about the recent research (particularly randomized clinical trials) of vitamin D supplementation and the implications for clinical practice.

As a director of the Vitamin D and Omega-3 trial (VITAL), the largest randomized clinical trial in the world, I'm often asked, "How much vitamin D do we need, and should I take a vitamin D supplement?" I want to review the findings from recent randomized clinical trials and the implications for practice.

For a long time, vitamin D has been perceived as a magic bullet, a panacea, and a cure-all for many chronic health conditions such as cancer, cardiovascular disease, diabetes, bone fractures, cognitive decline, and depression. Many of the findings, though, have been from observational studies where a higher blood level of 25-hydroxy vitamin D has been linked to a lower risk for these health conditions.

We know in epidemiology that correlation doesn't prove causation. Other factors could be involved; for example, people who have higher blood levels of vitamin D may have healthier diets, or they may be spending more time outdoors, being physically active and exposed to the sun. Some of these other factors could be lowering their risk.

When the randomized trials began to emerge, in many of these large-scale trials, the findings were generally neutral or null for cardiovascular disease, total cancer, diabetes, cognitive decline, depression, and many other health outcomes, including fracture. So, the question was asked, does this mean that vitamin D is not important to health?

To the contrary, these findings suggest that vitamin D is so essential to health that we need only small to moderate amounts of vitamin D. Vitamin D is very tightly regulated in the body — the metabolism and function of vitamin D. Even small to moderate amounts will meet the requirements for vitamin D and bone health and many other outcomes.

This is what the National Academy of Medicine, US Preventive Services Task Force, and many other professional organizations have advised, that widespread screening for vitamin D deficiency and blanket universal supplementation with vitamin D would not be indicated.

The randomized trials of vitamin D, including the VITAL study, have generally not shown reductions in the major health outcomes. We found two exceptions in VITAL. We saw promising signals, including a 22% reduction in autoimmune conditions (rheumatoid arthritis and psoriasis) and a 17% reduction in advanced (metastatic or fatal) cancers. In meta-analyses of other large-scale randomized trials, the findings were a signal for a reduction in advanced cancers, even with very small doses of vitamin D (400-800 IUs daily). We tested 2000 IUs daily in VITAL.

Overall, it's recommended that small to moderate amounts of vitamin D are adequate, and among the healthy population, most people do not need screening or supplements.

The reduction in autoimmune diseases suggests that vitamin D may play a role in tamping down inflammation. The question has been raised about whether vitamin D is beneficial in reducing the severity of COVID illness, the need for hospitalization, and long COVID. We are looking at this question in a separate trial called VIVID (Vitamin D for COVID Trial) which tests a higher dose (> 3000 IUs daily) of vitamin D. Those results will be available at the end of this year or early next year.

In other randomized trials of COVID and vitamin D, the results have been mixed and inconsistent, with no clear answer. During the COVID pandemic, I have generally advised that it's reasonable to take 1000-2000 IUs of vitamin D daily as a form of insurance. This dose is known to be very safe. Over 5.3 years in the VITAL trial we saw that a dose of 2000 IUs was very safe.

But it's not essential to take a supplement. And overall, aside from some high-risk groups, most people do not need a supplement. The high-risk groups include patients in nursing homes who may have restricted diets and limited time out of doors. For people with malabsorption conditions such as Crohn's disease, celiac disease, post–gastric bypass surgery, and those with osteoporosis who are on medications for osteoporosis, it's still quite reasonable to prescribe calcium and vitamin D.

Recommendations for vitamin D in the generally healthy population really should focus on a healthy diet. The United States has a fortified food supply. Vitamin D is added to many foods, dairy products, and cereals, as well as beverages. Natural sources of vitamin D include fatty fish and wild mushrooms.

We should be looking at food labels (which now include vitamin D content) and try to get adequate vitamin D from our diet, and also do our best to spend time outdoors, being physically active, because it is of great benefit to our health. The general principle is that a dietary supplement will never be a substitute for a healthy diet or healthy lifestyle. And those other behaviours really should be the focus at this time.

Did you know about Christmas ...

The 12 Days of Christmas Involving Mince Pies

A partridge in a pear tree, two turtle doves and all the rest — there is a good chance you'd be able to sing the entire lyrics of *The 12 Days of Christmas* without giving it a moment's thought. However, do you know the links to mince pies during this period of the festive season? Back in the Middle Ages, people across Europe would eat one mince pie every day from Christmas Day up to and including January 6th. Rather than being the same taste though, mince pies back then would be filled with a mixture of meats and fruit and spiced with everything from cinnamon to cloves and nutmeg.

Santa Claus in a Green Suit ?

Head to a Santa's grotto with your family throughout December and we're sure you'll be expecting to see a jolly fellow with a huge white beard wearing a red suit. However, what if we told you that Santa Claus was once known to wear a green suit? What has caused the change in look to come about is likely that we now see Father Christmas and Santa Claus to be the same mystical figure. In the past though, they were two — or possibly even more — very different people.

One of these individuals was St Nicholas, the Bishop of Myra in Turkey during the 3rd Century. St Nicholas was known to travel around in his red robes and give gifts to the poor but was said to be so shy that he gave families money secretly by dropping coins down a chimney of a home — which landed in a stocking in the room below. The legend of St Nicholas came to Britain when the Normans arrived across the nation, where it appeared to quickly absorb into the British legend of Father Christmas.

However, Father Christmas had actually been around for much longer, with various stories and legends talking about pagan winter festivals that included such a figure. In centuries gone by though, Father Christmas appeared to represent the coming of spring and his attire included a long green hooded cloak, as well as a wreath made of holly, mistletoe or ivy. His role was quite similar to the Father Christmas we associate with today mind — to make people happier throughout the winter months.

In 1066 AD: William, The Conqueror was crowned King of England.

We all know that on 14th October 1066, Duke William of Normandy (better known as William the Conqueror) defeated the army of King Harold II of England at the Battle of Hastings, arguably the most famous and era-defining battle in English history. After the battle, William advanced on London picking off any final resistance and completing his conquest of the English mainland. On the 25th of December 1066 in Westminster Abbey, William was crowned King of England becoming the country's first Norman king, ending a period of over 600 years of Anglo-Saxon rule. The Norman era brought about huge cultural, social and political changes that would have a massive impact on English as well as European history.

A Swedish Christmas Eve Tradition

Families in Sweden traditionally celebrate Christmas by watching the well know cartoon charter Donald Duck. The tradition started in 1959 when a Disney film *'From All of us to all of You'* but also known as ' Donald duck and his friends wish you all a Merry Christmas came out. At that time in Sweden, as in the UK there were very few TV channels so it was watched by many Swedes and became a Christmas Tradition.

North of Doncaster – South of Wakefield (2)

Personal comment from Trevor Wainwright.

The lake as viewed from the A61

Following my visit to Newmillerdam's Gnome Roam, it would be late Summer or early Autumn before I got to visit Newmillerdam Lake itself, the purpose being to add more photos to my, My Beloved Yorkshire Album on Facebook to add to the already 1457 there.

The village itself starts at Pledwick Cricket Club pitch, continues past the lake where there is a right turn called Boyne hill, it ends just past The Bay Horse on Stoney Lane, keeping on the A61 it ends at the top of the hill just after Wood Lane on the right .

Lodge at the North entrance

Originally called Thurstonhaigh, this suggests it to be a Norse settlement named in three parts: *Thur* meaning Thor, *ston* meaning stone or rock and *haigh* derived from the old Norse word 'hagi' describing a person who lived near a hedged or fenced enclosure.

The village is currently named from the construction of a grain mill powered by water from the dammed lake, and thus it is called the new mill on the dam. The mill still stands, although it is non -operational and privately owned, but there is no indication of which building is the mill which was originally owned and operated by the Pashley family, who lived in the village until the 1980s. The Pashleys owned many local businesses overthe centuries, which included blacksmiths, coal mines and a furniture making business. These furniture makers were also general carpenters and installed one of the first public toilets in the yard of The Three Houses Public House in 1852. The Pashley family were Methodists and were provided money to build two chapels in the village. The chapels are situated on School Hill and Barnsley Road. The Pashley reference is noted by the dedication stones to William M Pashley. The family also funded and built Newmillerdam School, which is located on School Hill. However, the land owning gentry of the time, the Pilkington family, took most of the credit and introduced the Miss Pilkington Scripture Prize as an annual award in the school.

Apparently the land was initially owned by the Neviles, who in 1529 built Chevet Hall. The east of Newmillerdam demolished as a result of mining subsidence in the 1960s, despite a massive outcry from locals. In 1765, the hall and estate was acquired by the Pilkingtons; in 1820, they built the boathouse on the lake in their private grounds. The Pilkingtons built lodges around their private estate to deter poachers who if caught were severely punished. Locals also were not allowed in the grounds. There were nine lodges in all, now only two survive, one either side of the dam. The grounds were opened to the public two years after Wakefield Council bought the estate in 1954.

One of the nature trails

Leaving the car park to the east walking to the lake past what could have been one of the lodges but is now a, Italian lakeside restaurant, walking along the road which forms the dam it was to what is known as the 'main entrance', just through the gates is the remaining lodge, although small 0,it would make a good information centre were there the resources for it.

(Sweet) Chestnuts

So it was follow the path which is wheelchair friendly all the way round. I decided to take a higher path which offered good views of the lake and was quiet with only the solitary squirrels for company. Coming to where two paths cross I headed back down to the lake, noticing some sweet chestnuts on the floor I picked them up and opened them, not quite ripe but eatable. I had come out near the boathouse which in its heyday the men shot wildfowl on the lake from punts launched from the Boathouse, the ladies joining them for lunch.

Now the only sport is fishing, for which there are platforms built going slightly out into the lake. For a long while it had been unused now, it was good to see it as a thriving cafe. Partway round there is a pontoon bridge for anyone not wanting to go the whole way round but the day I went it was close, so onwards to the end, where there are a variety of paths to take to other parts of the estate, one even going to a viaduct over what was the old Chevet Branch Line which is now a nature reserve trail. I decided to make my way back along the opposite side of the lake, walking along the side of Lawns Dike as it ran parallel to the main lake before joining it.

I came across two more gnomes who do not feature on the roam but by the lake itself. Gnome 'Roger the Bug Watcher' and Gnome 'Muriel (Strange name for a bearded gnome) the Bird Watcher'. Leaving them I glanced in to the woods and saw yet another shelter example of outdoor activities and woodcraft. Then it was to the end of the path but not the end of the day. The run off is under the A61 and it the past used to be part of a weekend ritual for car owner as it ran as a ford over Slack Lane, brush and wellies were the order of the day as cars were driven into the ford and washed. This came to an end with concerns about river pollution. What can be seen on Slack Lane now is a footbridge over the ford and the land developed for housing.

The boathouse ...

From the ford it runs to Kettlethorpe Hall Lake where it becomes Owler Beck and runs to the West of Sandal Castle into the River Calder. Kettlethorpe Hall Lake is reached by an unsigned road heading back into Wakefield just past the Pledwick Well Inn on the left. The road is marked as a cycle path to Pugneys but is in fact the entrance road to Kettlethorpe Hall. Driving down it you come to a car park just before the hall gates, as you go over the bridge to it there are two notices, one explains how it was part of Kettlethorpe Hall Ornamental Gardens before being taken over by Wakefield Council and leased to Wakefield Fishing Club, the other is the partnership between Yorkshire Wildlife Trust and Kettlethorpe High School's involvement project in 2019 to bring it to the beauty spot it is today. A small but easy lake to walk round with an array of waterfowl, there was a small patch of blue bells out when I walked round.

Across from the car park is Kettlethorpe Hall built by the Pilkington Family in 1747. In 1950, Kettlethorpe Hall was purchased by Wakefield Council, and was used as a home in 1983. In 1988, the hall was bought by Yorkshire Preservation Trust, who converted it into two separate houses. Wakefield Council still owned the grounds surrounding the hall. Nowadays, the house is privately owned, but the grounds are a public park, in 1953, Kettlethorpe Hall became a Grade I listed building which no doubt has its own unique history.

...Now a thriving lakeside cafe