Pathways

Price £ 3.75 (Free to members)

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. 55. (Spring 2018)

Springtime in the Yorkshire Sculpture Park by Carolyn

You write in

***Clare Writes**: There is a community screening of UNREST at Selby Town Hall, York Street, Selby on 23/05/18 at 7.30pm. I realise that this may be too far for some of your group to attend. If anyone would like tickets they can email me directly at*

clare-Bromley@sky.com.

My story is also available to read there. A brief over view would be that I am the parent of Isobel aged 17 who was diagnosed with ME 6 years ago. She spends most of her time house bound and at times bed bound. If you need any further info please let me know.
I will put the tickets behind the desk, so they can bed picked up on the night. I have attached a poster for you to share. Tickets are by donation and donations can be made via my just giving page.

www.justgiving.com/clarebromley.

***Bill Writes**: I suffer from arthritis as well as ME. I was talking with my neighbours and has says that I shouldn't be eating tomatoes. Is there any truth in it?*

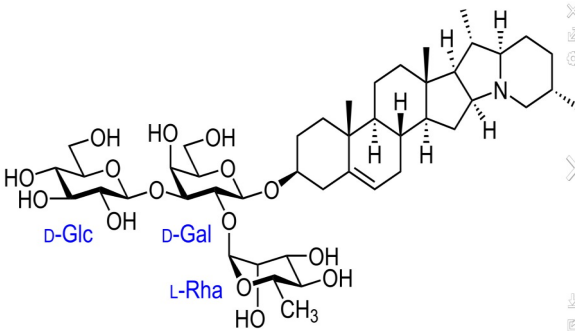
I've heard of this too. Some people may react to solanine from the nightshade family of plants (tomatoes, peppers, potatoes, aubergines and chilli). Although the evidence seems to be anecdotal, many people report improvement of symptoms after following a nightshade-free diet.

Solanine is a toxic glycoside, and though to be produced as a defence mechanism by the plant. Most of it is present just under the skin in potatoes, so peeling would help reduce the levels. Cooking only reduces levels by about half.

Green potatoes usually go black on cooking and so are usually discarded.

The mechanism is Solanum glycoalkaloids can inhibit cholinesterase (see later in pages), disrupt cell membranes, and cause birth defects. One study suggests that the toxic mechanism of solanine is caused by the chemical's interaction with mitochondrial membranes.

Experiments show that solanine exposure opens the potassium (K+) channels of mitochondria, decreasing their membrane potential. This in turn, leads to K+ being transported from the mitochondria into the cytoplasm, and this increased concentration of K+ in the cytoplasm triggers cell damage and apoptosis (cell death). While for a normal health person solanine may be well tolerated, for someone with a chronic condition this may cause further distress.



Mick Writes: *I'm one of those who has Diabetes as well as ME/CFS. I heard on the BBC that there are now five types of diabetes. What can you tell me about it?*

A high proportion of leger ME members are diabetic. So, I think it's worth reviewing. I've had a look at the research, and I've summarised the findings on a table:

Cluster	Current Designation	People affected	Mechanism	Over weight ?	Complications risk increased
1) Severe autoimmune diabetes	Type 1	Relatively young	Islet cells damaged by autoimmune action. No insulin produced	No	
2) Severe insulin-deficient diabetes	Type 2 but looks likes type 1	Relatively young	No immune damage, but islet cells do not produce enough insulin	Not necessarily	Retinopathy. Back of eye damage
3) Severe insulin-resistant diabetes	Type 2	Any age after teens	Metabolic insulin resistance high, but produce insulin in large quantities.	Yes	Kidney disease
4) Mild obesity-related diabetes	Type 2	Middle age	Being overweight, and not producing enough insulin for overweight body	Yes	
5) Mild age-related diabetes	Type 2	Older	Aging of islet cells, not producing enough insulin.	Yes	

All this is as an observational study. It effectively splits type 2 into 4 subtypes.

Those people involved with diabetes are aware of the subtypes – but as regarding treatment and management it does not really change anything. Here are my three golden rules for management.

1) See you GP, practice, or diabetic nurse at least yearly, more often if necessary. Attend the various test clinics as recommended to check for complications and management problems. With Type 2 diabetes, high blood pressure and high cholesterol and commonly found.

2) Monitor your blood sugar level under control by regular testing with a blood glucose meter. Metformin is a good first choice for people with ME/CFS if tolerated, to control blood sugar levels because of favorable side effects.

3) Modify your diet to a heathier food choice, minimising the sugar content. Avoid smoking and chemical fumes as far as practicable.

Carolyn Writes: *I look forward every spring to the daffodils and narcissi flowering. For us with ME/CFS, they brighten up our garden with very little work being needed for an effective display.*

This year I've been disappointed because something has been eating my flowers. See photos. Additionally, some of my daffodil heads have just been snapped off completely. I have a mixture of different types. One clue is that all my 'Tete a tete' daffodils have been untouched. Any ideas of what or who is creating the damage, and what can be done about it?

It could be slugs or bird damage? Is there anyone out there who can offer an explanation or that has had a similar experience? 'Tete a tete' daffodils are a different race and may taste or smell different and that's why they have been preserved. Maybe that is the way forward. For now, I would cut off the damaged flower heads and leave the leaves to make another bulb for next year. Hopeful we may have an answer by then.

Welfare Rights Matter *with Thanks to Benefits and Work*

Tribunals face dumbing down as they go digital: The cost-cutting agenda behind moving tribunal hearings online has been further exposed in new regulations which will to allow ministers and senior judges to cut the number of people sitting on panels, including social security appeal tribunals. The First-tier Tribunal and Upper Tribunal (Composition of Tribunal) (Amendment) Order 2018 allows the Senior President of Tribunals (SPT) to decide on the number of panel members for any type of tribunal. In addition, in the future the SPT must first consult with a government minister before making any decision. The government claim that the aim is to make "the use of tribunal panel members should be more tailored and flexible". The fear is that the aim is simply to cut costs by ensuring that most tribunals have only a judge sitting alone.

At present ESA tribunals have a medical member as well as a judge. PIP and DLA tribunals have a judge, a medical member and a person with specialist knowledge of disability issues. Under the new rules the SPT could decide that all social security hearings would be heard by a judge sitting alone, with a medical member only in special cases. The use of a disability specialist could be dropped altogether. Many Benefits and Work readers will be able to tell tales of medical members, and even disability members, of panels who behaved in a distrustful and even hostile manner, whilst the judge appeared to be fair and sympathetic. But it is equally the case that it is often the medical member who points out flaws in the evidence collected in the face-to-face assessment. And disability members frequently ask questions touching on the daily lives of claimants which help to make clear the real difficulties they face with everyday activities. A judge sitting alone may have no lived experience of disability and lack any knowledge of the medical condition at issue. It is hard to see how there can be any advantage to claimants in reducing the number or range of people sitting on PIP, ESA or DLA appeal panels. But the cost advantages to the government are clear.

Specialist MS nurses condemn benefits assessment process: Specialist MS nurses have spoken out about how damaging the assessment process for PIP and ESA is and how it is affecting their ability to support patients. The situation is something similar with ME/CFS.

- A survey by the MS Society of over 100 MS nurses revealed that:
- 90% of nurses said they provided supporting evidence for benefits applications.
- Of those, 58% said they worked outside of working hours to provide this evidence.
- 75% said providing evidence increased their workload either a moderate amount or a lot.
- 83% of everyone who answered said their patients asked for help with filling in benefits applications.

One nurse explained:
"I see the effects of patients not being able to get benefits. One had to stop his treatment because a cut to his benefits meant he could no longer get to the hospital. So, I feel a lot of pressure to make sure I do as much as I can to help my patients. But on average I'm getting asked to do these five times a week, it's overwhelming."

Another stated:
"We're not given any guidance about what to put in these letters, and it's not a simple process. All my patients going through this find it very stressful and some have told me how they've lost sleep over their applications or had increased anxiety. Both stress and anxiety make MS symptoms like fatigue and pain worse."

ESA Claimants to Get Up To £20,000, But Many Miss Out. A National Audit Office report has accused the DWP of failing to "get a proper grip on the problem" of ESA underpayments for years. It points out that even though some claimants will be repaid up to £20,000 "not everyone will be repaid all the money they have missed out on.". The problem arose from 2011 onwards, when some claimants who transferred from incapacity benefit to contribution-based ESA were not also assessed for income-related ESA, even though the legislation said they should have been. As a result, the DWP estimate that 45,000 claimants are owed around £2,500, another 20,000 are owed £11,500 and a small number

Staying Safe Online.

Following the recent scandal about Facebook and person data being harvested we thought it would be an idea to run this feature. Using the internet has made many of our everyday tasks quicker and easier. We can research financial 'or health issues, keep in touch with family and friends on social media, and use banking and shopping websites. However, it's now more important than ever to avoid becoming an easy target for scams.

Following these simple steps will mean you can enjoy using the internet safely.

Think before you click
Phishing - pronounced 'fishing' - is a term used to describe fraudulent emails, web pages or even phone calls that aim to trick us into providing our personal information or login details. Generally, be suspicious of any email, text or phone call that asks you to provide information (even if it appears to be from someone you know).

- Never click on links or open email attachments unless you know exactly what it is, and you were expecting to receive it.
- If you have doubts, don't reply - instead, make sure the message is genuine by contacting the sender directly, either through the official email address or website, or over the phone.

Social networking - be careful what you share
The more personal information you post online or include in your profile, the easier it is for thieves to steal your identity or commit other crimes like stalking or fraud.

- Don't connect to anyone you don't know personally and make sure only your friends can see your profile. Beware of fake friend requests and posts from individuals or companies inviting you to visit other pages or sites.
- Be wary of social media advertisements and pop-up windows - close pop-ups and don't click on anything that appears unexpectedly.

Protect your computer, mobile phone and tablet
There are lots of steps you can take to protect the equipment you use to connect to the internet.

- Install an antivirus package that suits your needs and keep it up to date - some have extra features like password managers and webcam protection.
- Set up a hard-to-guess password to protect your phone, laptop, tablet, external hard drives and USB sticks when not in use.
- Only install apps from official sources (like Apple App Store or Google Play), delete apps you no longer use, and always use the latest versions of software and apps.

Perfect passwords
Passwords are keys for opening your online your passwords like real keys and use a similar common-sense approach to look after them.

- Don't use the same password for more than one account to make sure that, if someone does find out your password, they can't use it anywhere else.
- Some companies like Apple, Gmail, Facebook and Amazon offer two-step verification. If you choose to enable this in your privacy settings, it will protect your online accounts by adding an extra layer of security when logging in. You'll need two pieces of information to access your account - for example, your password plus a unique code texted to your phone.

The difference between Good and Bad passwords

A Good Password is Random, A Bad Password is Predictable
The more random your password the better. Why? Because if your password is made up of patterns of numbers or keystroke patterns then it will likely be easily cracked by hackers using dictionary-based password cracking tools.

A Good Password is Complex, A Bad Password is Simple
If you only use numbers in your password, then it will likely be cracked in a matter of seconds by a password cracking tool. Creating Alpha-numeric passwords increases the total number of possible combinations which also increases the amount of time and effort needed to crack the password. Adding special characters to the mix also helps.

A Good Password is Long, A Bad Password is Short
The length of a password is one of the biggest factors in how quickly it can be cracked by password cracking tools. The is the longer the password the better. Make your password as long as you possibly can stand. Traditionally, password cracking tools will require much more time and computing power to tackle longer passwords, such as those 15 characters or longer, however, future advancements in processing power may change the current password limit standards.

Secure password choices:
Gu*82me@
Ug(1!n88
Gn32(*2d
()@!#mMw
****#$@2Mp**

Please don't use these as is !
Make up your own.

Password Creation Cheats You Should Avoid:

Reusing Old Passwords
While reusing old passwords seems like a brain saver, it increases the likelihood that your account might be hacked because if someone had one of your old passwords and you've cycled back to using that password then your account may become compromised.

Keyboard Patterns
Using a keyboard pattern may help you bypass your systems password complexity checking, but keyboard patterns are part of every good cracking dictionary file that hackers use to crack passwords. Even a long and complex keyboard pattern is likely already part of the hacking dictionary file and will likely result in your password being cracked in mere seconds.

Password Doubling
Simply typing the same password twice to meet password length requirements doesn't make it a stronger password. In fact, it can make it very weak because you have introduced a pattern into your password and patterns are bad.

Dictionary Words
Again, using whole words in a password is not advisable because hacking tools are built to target passwords containing whole words or partial words. You may be tempted to use dictionary words in your longer passphrases, but you should avoid this because dictionary words as part of passphrases may still be crack able.

Password Cracking Explained
A lot of users think that their password is safe because they think that a hacker can only make 3 attempts on their password before the account is locked. What many users don't understand is that password hackers steal the password file and then attempt to crack that file offline. They will only log into the live system after they have obtained a cracked password and know that it is one that is going to work.

B12 - Rationale for using Vitamin B12 in CFS *by Dr. S. Myhill*

Since 1982 a programme of treatment has evolved which I believe all Chronic Fatigue Syndrome patients must do as the foundation before proceeding to other treatments. Vitamin B12 by injection I see as an important part of this programme and it is effective for many, regardless of the cause of their Chronic Fatigue Syndrome.

Those patients who respond to B12 are not obviously deficient in B12; indeed, blood tests usually show normal levels. The "normal" levels of B12 have been set at those levels necessary to prevent pernicious anaemia - this may not be the same as those levels for optimal biochemical function. B12 has a great many other functions as well as the prevention of pernicious anaemia. However, what is interesting is how B12 is beneficial in so many patients with fatigue, including those suffering with CFS, and this suggests that there is a common mechanism of chronic fatigue which B12 is effective at alleviating, regardless of the cause of the fatigue.

General mechanism by which B12 relieves the symptoms of CFS
Professor Martin Pall has looked at the biochemical abnormalities in CFS and shown that sufferers have high levels of nitric oxide and its oxidant product peroxynitrite. These substances may be directly responsible for many of the symptoms of CFS and are released in response to stress, whether that is infectious stress, chemical stress or whatever. B12 is important because it is the most powerful scavenger of nitric oxide and will therefore reduce the symptoms of CFS regardless of the cause.
Nitric oxide is known to have a detrimental effect on brain function and pain sensitivity. Levels are greatly increased by exposure to chemicals such as organophosphates and organic solvents. When sensitive tests of B12 were applied (serum methylmalonic acid and homocysteine) before and after B12 therapy, the following symptoms were noted to be caused by subclinical B12 deficiency: parasthesia, ataxia, muscle weakness, hallucinations, personality and mood changes, fatigue, sore tongue and diarrhoea.

B12 in fatigue syndromes
The "foggy brain" with difficulty thinking clearly, poor short-term memory and multitasking are often much improved by B12. Mood and personality changes, so often a feature of patients with chemical poisoning, can be improved by B12. The physical fatigue and wellbeing are often both improved.

A study
Twenty-eight subjects suffering from non-specific fatigue were evaluated in a double-blind crossover trial of 5 mg of hydroxocobalamin twice weekly for 2 weeks, followed by a 2-week rest period, and then a similar treatment with a matching placebo. The placebo group in the first 2 weeks had a favorable response to the hydroxocobalamin during the second 2 week period with respect to enhanced general wellbeing. Subjects who received hydroxocobalamin in the first 2-week period showed no difference between responses to the active and placebo treatments, which suggests that the effect of vitamin B12 lasted for over 4 weeks. It is noted there was no direct correlation between serum vitamin B12 concentrations and improvement. Whatever the mechanism, the improvement after hydroxocobalamin may be sustained for 4 weeks after stopping the medication.

Practical details
Vitamin B12 has no known toxicity and B12 surplus to requirement is simply passed out in the urine (which may discolour pink). It is theoretically possible to be allergic to B12 but in the thousands of injections that I have sanctioned this has only ever occurred after several injections and caused local itching, redness and swelling (although the commonest cause of redness and swelling is poor injection technique) in a handful of patients. I usually start with 1/2 mg (500 mcg) daily by subcutaneous injection, then adjust the frequency according to response - some patients will respond straight away, some need several doses before they see improvement. I would do at least two months of daily injections (i.e. 60) before giving up. If there is improvement the adjust the frequency of the dose to maintain that. Many people end up injecting at least once a month, often 1-2 times per week, more if stressed, less often if well. Indeed, I suspect we could all benefit from a monthly dose of B12 as we age. It is protective against dementia and heart disease. There is some evidence that vitamin B12 is anti-viral and this may explain its wide application and why so many see benefit. Dr. Patrick Kingsley, in treating his MS patients, found some required 5,000mcgms daily, perhaps more, to really feel well.

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The Local Reality if B12 injections.

1) There is no support in the NIHCE guidelines for B12, nor is there in the British National Formulary (The NHS bible) being used for treating ME/CFS. This means that if an NHS doctor chooses to treat someone was has ME/CFS with B12, it would be outside the official guidelines (sometimes known as off label prescribing). Effectively he or she would not be covered by his insurance and be heavily liable if something went wrong. In a way this is contradictory because TCAD drugs like amitriptyline at low dose have no NIHCE or BNF guidelines. It's just that the consensus of medical opinion supports their use.

2) Oral B12 is available over the counter without restriction. Injectables forms are prescription only.

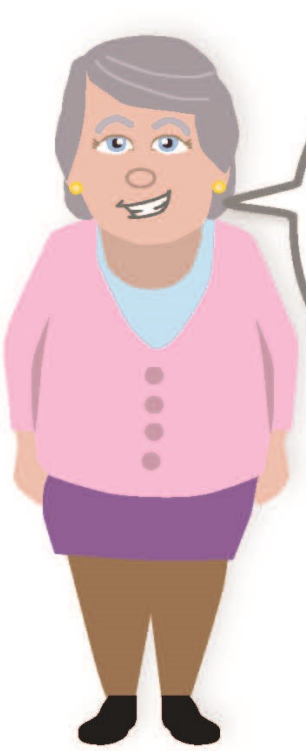
3) Dr. Myhill will prescribe B12 for her patients but is not taking on new patients. Currently I'm not aware of any doctor locally private or NHS who will prescribe B12.

4) Dr. Myhill says "I now have a preparation of B12 which delivers 5,000 mcg (i.e. ten times the dose in a ½ ml injection) as a sublingual spray (Under the tongue). Even with perfect B12 absorption one can only expect 1% to get through the gut wall! So, the idea of the sublingual spray is that some is absorbed under the tongue, the so called "first pass effect" whereby the liver is by-passed. It works well for some people, but many people find that injections are irreplaceable!".
Whether this is psychological or physical is unclear to me. Ed.

5) The most common enquiry I get is when someone's doctor retires who in the past prescribed B12, and the partners will not. I don't really have an answer. I do know that some people import B12 injections from countries where its supply is not a Prescription Only Medicine. I do worry about quality and efficacy of imports. The problem with it is a technical one of safety because there are no guidelines or controls.

I'm sure that it is possible to mimic the anti peroxynitryl by using conventional vitamins and supplements.

The integrated Doncaster Care Record.



What is the Integrated Doncaster Care Record?

It's a new electronic way of storing information about you that can be seen by health and care staff in different locations when you need help.

Being able to see your health and social care records at any time, and in different places in Doncaster means doctors and other professionals can make quicker and safer decisions about your care.

Why do you want to share my information?

Over the years, there's a good chance that you will have received care at a number of places in Doncaster from hospitals to GP surgeries – and the details of your care will be stored on different computer systems. The Integrated Doncaster Care Record (IDCR) has pieced together those computer systems like a jigsaw, to enable those looking after you to see a detailed picture of your health and care history.

It means quicker and more efficient care for you by replacing more traditional forms of information sharing, such as letters and phone calls.

What information will be shared?

Your care record contains key information like:

- Personal details, such as your name, address, date of birth and and next of kin
- Names of the health and care professionals looking after you
- Any medications you are taking
- Any allergies you have
- Any health concerns about you
- Previous referrals to services

- Dates and reasons for any occasions you have been admitted to hospital
- Appointments
- Any assessments you have had
- Care plans and care packages you have
- Emergency contact details

It will not contain sensitive information, such as your sexual health history. Initially, your record will contain information recorded by any of the following organisations, if at any time they have provided care for you:

- Doncaster GPs
- Doncaster and Bassetlaw Teaching Hospitals Foundation NHS Trust
- Rotherham Doncaster and South Humber Foundation NHS Trust
- Fylde Coast Medical Services (Doncaster Urgent Care Service)
- Doncaster Metropolitan Borough Council (Adult Social Care)

This is just the start. In time more service providers will add information, such as ambulance services and community pharmacies.

Who will access my information?

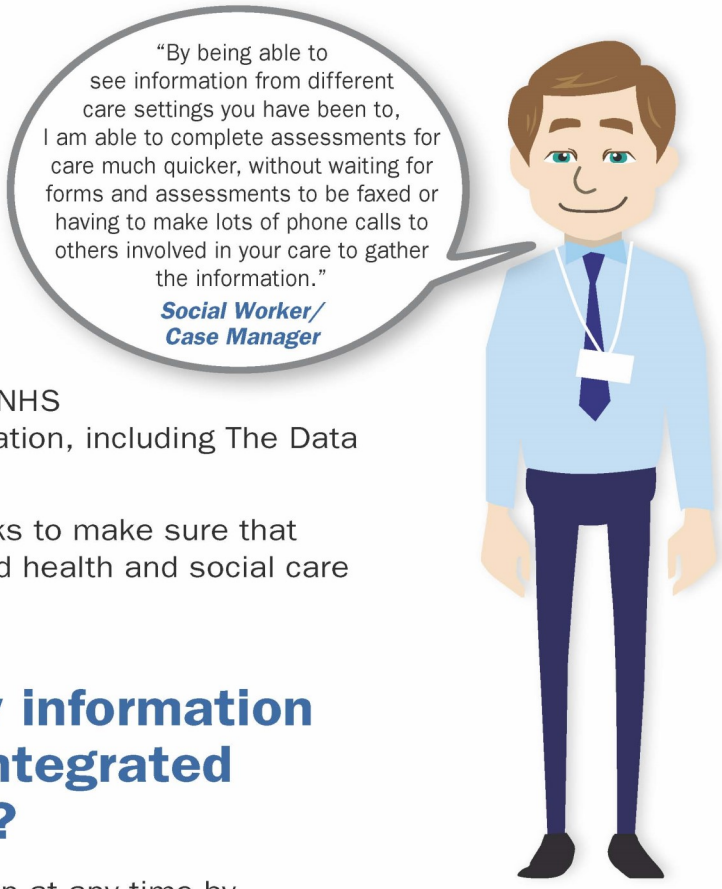
Information about you will only be shared with those Doncaster based health and care professionals who are caring for you. And they will only be able to view information relevant to their job.
So, a social care worker may see different parts of your record to an occupational therapist.

Your information will not be shared with third parties, such as insurance companies.
You can:

- give your consent for a professional to see your record every time you come into contact with care services; or
- give on-going consent to anyone involved in your care so you don't have to be asked again.

If you choose to opt out of sharing your record all together (see over leaf), information about you will not be available via the Integrated Doncaster Care Record.

How do I know my records are kept safe?



By law, everyone working for, or on behalf of,the NHS and social care services must follow confidentiality rules and keep all information about you safe. Information about you will be viewed through a secure, encrypted and audited system that meets stringent NHS security standards and government legislation, including The Data Protection Act.

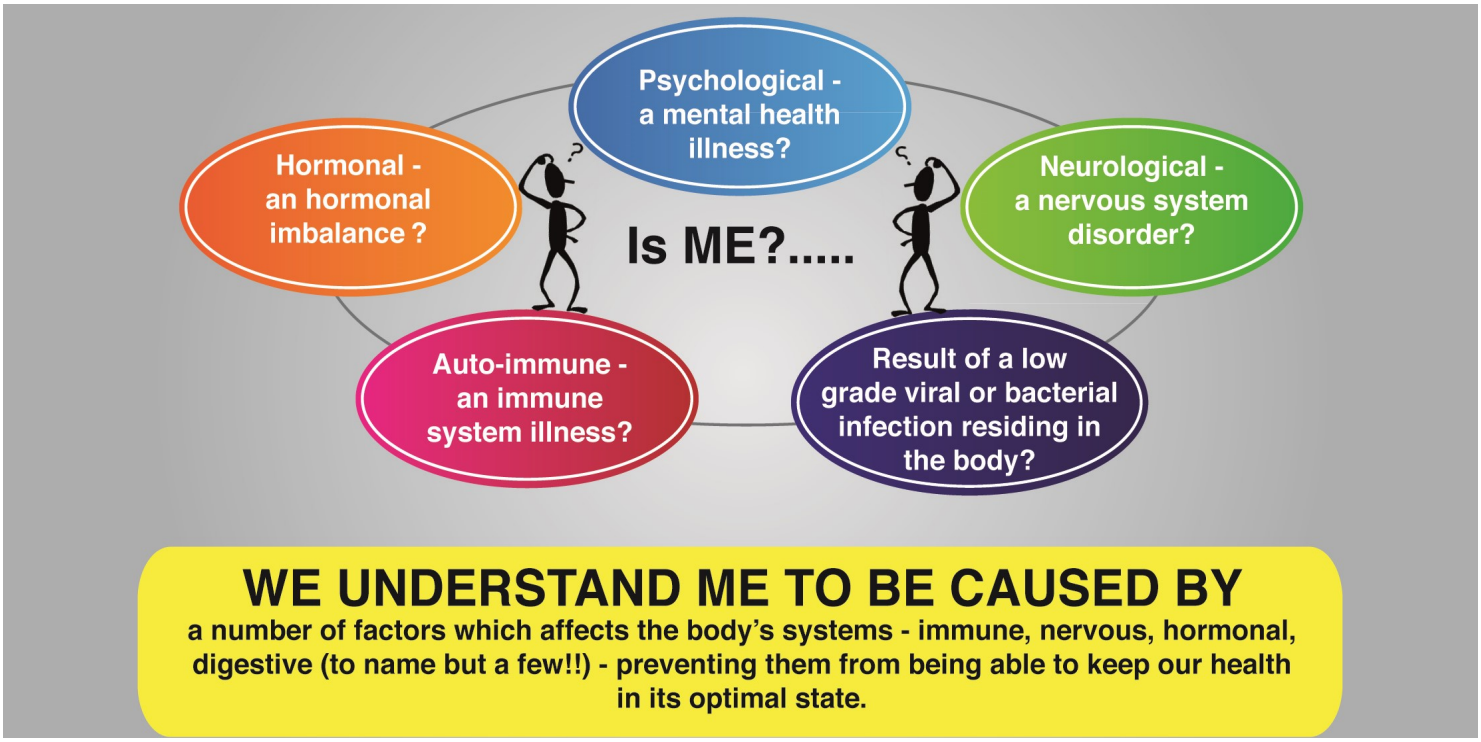
In addition, we will carry out regular checks to make sure that your records are only viewed by authorised health and social care professionals, who have a valid reason.

How can I opt out of my information being shared with the Integrated Doncaster Care Record?

You can opt out of sharing your information at any time by

- completing the online form at www.doncasterccg.nhs.uk/idcr/optout/
- emailing donccg.idcr@nhs.net
- writing to IDCR Co-ordinator, NHS Doncaster CCG, Sovereign House, Heavens Walk, Doncaster, South Yorkshire, DN4 5HZ
- ringing 01302 566050

However, we ask you to think carefully before making this decision as sharing your health and social care information will make it easier for Doncaster services to provide the best treatment and care for you.



Wilson's Temperature Syndrome: Real or Imaginary?

From Wikipedia

Please do not confuse this with Wilson's disease, a medically recognized condition caused by a defect in copper metabolism.

Wilson's (temperature) syndrome, also called Wilson's thyroid syndrome or WTS, is an alternative medicine concept which is not recognized as a medical condition by evidence-based medicine. Its supporters describe Wilson's syndrome as a mix of various common and non-specific symptoms which they attribute to low body temperature and impaired conversion of thyroxine (T4) to triiodothyronine (T3), despite normal thyroid function tests. Denis Wilson, a physician who named the syndrome after himself, advocates treating these symptoms with sustained-release triiodothyronine.

The American Thyroid Association (ATA) describes Wilson's syndrome as at odds with established knowledge of thyroid function. The ATA described the diagnostic criteria for Wilson's syndrome as imprecise and non-specific, and found a lack of any scientific evidence supporting Wilson's claims. The ATA further raised concern that the proposed treatments were potentially harmful. Florida State Medical Board members described Wilson's syndrome as a "phony syndrome" and a scam during disciplinary action against Wilson.

Origins and claims: The term "Wilson's syndrome" was coined in 1990 by E. Denis Wilson, a physician practicing in Longwood, Florida. Wilson said that the syndrome's manifestations included fatigue, headaches, PMS, hair loss, irritability, fluid retention, depression, decreased memory, low sex drive, unhealthy nails, easy weight gain, and about 60 other symptoms. Wilson wrote that the syndrome can manifest itself as "virtually every symptom known to man." He also says that it is "the most common of all chronic ailments and probably takes a greater toll on society than any other medical condition."
Wilson says that low thyroid symptoms and low temperatures in the presence of normal thyroid function tests are not due to hypothyroidism and might be reversed with a few months of treatment. To distinguish this condition from hypothyroidism, he named it Wilson's (temperature) syndrome. He states that it is "especially brought on by stress" and can persist after the stress has passed. He says that the main diagnostic sign is a body temperature that averages below 98.6°F (37.0°C) (oral), and that the diagnosis is confirmed if the patient responds to treatment with a "special thyroid hormone treatment". He says that certain herbs can also help support normal body temperatures.

Patient death and medical license suspension: In 1988 a 50-year-old woman died of an arrhythmia and heart attack while taking excessive amounts of thyroid hormone prescribed by Wilson; around that time, she confessed to not taking the medicine as regularly as prescribed. Four years later, in 1992, the Florida Board of Medicine took disciplinary action against Wilson, accusing him of "fleecing" patients with a "phony diagnosis". The Board of Medicine and Wilson settled the disciplinary action, agreeing to a 6-month suspension of Wilson's medical license, after which Wilson would need to attend 100 hours of continuing medical education, submit to psychological testing, and pay a $10,000 fine before resuming practice. Wilson also agreed not to prescribe thyroid medication to anyone unless the Board of Medicine determined that the medical community had accepted "Wilson's Temperature Syndrome" and Wilson's methods and modalities of treatment.

Evaluations: During disciplinary action against Wilson, members of the Florida Board of Medicine stated that there was no evidence [Wilson's] theory is valid. They described Wilson's treatments as dangerous and a scam, stating that Wilson was fleecing insurance companies and patients with treatments for "a phony syndrome". The American Thyroid Association (ATA), a professional association dedicated to promoting thyroid health, disavows Wilson's Temperature Syndrome. The ATA stated in 2005 that a "thorough review of the biomedical literature has found no scientific evidence supporting the existence of 'Wilson's Temperature Syndrome'." The statement added that the mean temperature of normal persons in the AM on waking is 97.5°F, not 98.5°F, and that many of the symptoms described by Wilson are nonspecific and typical of depression, anxiety, and psychological and social stress. It also notes that a similar set of symptoms occurs in the alternative diagnoses of neurasthenia, chronic fatigue syndrome, fibromyalgia, multiple chemical sensitivity, chronic Epstein-Barr virus syndrome, and chronic candidiasis. Finally, the Association notes that chronic supplementation with triiodothyronine (T3) is particularly difficult and problematic, since various tissues set their own cellular levels of this hormone by making it individually from thyroxine, and supplementation of T3 may overwhelm this normal regulatory mechanism in some of these tissues.

ME/CFS and Thyroid matters
By Dr. Sarah Myhill and Craig Robinson

There are four reasons why UK citizens are not subject to "best practice" with respect to prescribing thyroid hormones. All relate to the prescribing of thyroid hormone for under-active thyroid glands (hypothyroidism).

The threshold for thyroid stimulating hormone (TSH) is set too high.

When levels of thyroid hormones in the blood start to fall, the pituitary gland increases its output of thyroid stimulating hormone (TSH), which kicks the thyroid into life and increases output of thyroid hormones. If the thyroid gland starts to fail, this is reflected by levels of TSH rising. The question is at what point should the prescription of thyroid hormones begin?

The normal range for TSH in this country varies enormously from one laboratory to another. This means in some locations in the UK a thyroid prescription would not be given until the TSH rose above 5.0mIU/l.
Because of research, the normal range for TSH in America has now been reduced so that anybody with a TSH above 3.0mIU/l is now prescribed thyroid hormones. This research has shown that people with a TSH above 3.0mIU/l are at increased risk of arterial disease (a major cause of death in Western culture), insulin resistance (and therefore diabetes), inflammation and hypercoagulability (sticky blood). Indeed, there is a recommendation afoot in America to further reduce the threshold for prescribing to 2.5mIU/l.

What is completely illogical is that in UK the target TSH level for patients on thyroid replacement therapy is often stated as being less than 2mIU/l or even less than 1.5mIU/l. This is a ridiculous anachronism given that prescription is not recommended until levels exceed, say, 5.0mIU/l! So, someone could have a level of 4.0mIU/l and not be receiving thyroid replacement therapy (because their level is not above 5.0mIU/l), whereas if someone was on thyroid replacement therapy, a level of 4.0mIU/l would be considered much too high and would need to be brought down to below 2.0mIU/l or even 1.5mIU/l!

We should amend the threshold for prescribing thyroid hormones to 3.0mIU/L or better still 2.5mIU/l. There is a further inconsistency in BTA (British Thyroid Association) guidelines. The level of thyroid hormones in pregnancy is critical for foetal development. For pregnancy the target for TSH is a level below 2.5mIU/L. Furthermore, requirements during pregnancy increase, so thyroid function should be checked every three months. What is the logic of only prescribing thyroid hormones to a non-pregnant woman with a TSH of above 5.0mIU/l but if pregnant the prescribing of thyroid hormones would start when levels exceed 2.5mIU/l?

Dr. Kenneth Blanchard states that reliance on a TSH to diagnose hypothyroidism is the biggest single medical error of modern times. It has resulted in millions of people missing out on this safe, life transforming, disease preventing treatment.

Population normal range versus individual normal range - they are not the same
The population normal range for levels of thyroid hormone in the blood is not the same as the individual normal range. We differ as individuals in our biochemistry as we differ in our looks, intelligence and morphology. This biochemical variation should be considered when it comes to prescribing thyroid hormones.

The population normal range of a Free T4 is 12 - 24pmol/L. A patient, therefore, with blood levels of 12.1 would be told they were normal because they are within the population reference range. But, that person's personal normal range may be high. They may feel much better running a high T4 of say 22, i.e. nearly twice as much but still within the population reference range. Research done originally in UK, and now repeated in America, clearly shows that the individual normal range of thyroid hormones is not the same as the population reference range. To find out who these individuals are, patients must be assessed clinically as well as biochemically. In actual UK clinical practice this is rarely done except by a few physicians conversant with this issue.

Some people feel better on different preparations of thyroid hormones
In theory, if the patient has been shown to be hypothyroid, then all their symptoms should be improved with synthetic sodium thyroxine. In practice, this is not always the case - there is no doubt that clinically some patients feel very much better taking biologically identical hormones such as natural thyroid (a dried extract of pig thyroid gland which is a mix of T4 and T3). Indeed, before synthetic thyroid hormones became available, all patients were routinely treated with natural thyroid. The purity and stability of these preparations has been long established, indeed much longer established than synthetic thyroxine!

Part of the reason why people feel better taking natural bio-identical hormones is that some people are not good at converting T4 (which is relatively inactive) to T3 (which is biologically active). However, this does not explain the improvement in every case. It is difficult to explain why there should be an additional effect, but for many people it is the difference between drinking cheap French plonk (Plonk) and good quality Spanish Rioja. The alcohol content is the same, but the experience completely different!

According to Dr. A Toft, Consultant Endocrinologist, Edinburgh, “It would appear that the treatment of hypothyroidism is about to come full circle.”

“In patients in whom long-term T4 therapy was substituted by the equivalent combination of T3 and T4 scored better in a variety of neuropsychological tests. The treatment of hypothyroidism is about to come full circle”.
Ref: Endocrine Abstracts 3 S40, T3/T4 combination therapy. AD Toft, Endocrine Clinic, Royal Infirmary, Edinburgh, UK. Please see Endocrine Abstracts

Timing of dosing
I have learned much more from consultant endocrinologist Dr. Kenneth Blanchard's book "The functional approach to treating hypothyroidism". (Amazon.co.uk link to "The Functional Approach to Hypothyroidism". He makes many useful clinical points:

- Thyroid hormones should be taken with food - he observes that cravings can be triggered by thyroid hormones on an empty stomach
- T4 (thyroxin) is slow acting and "base loads". It is the night hormone - we should split our daily dose into two. The evening dose should be taken with supper which should be at least 4 hours before bed time. By contrast T3 is the day hormone that wakes us up.

Our requirements change with the seasons - in Nature TSH falls in winter so levels of T4 and T3 fall - this puts us into semi-hibernation and allows energy conservation by causing mild fatigue and depression with greater need for sleep. The reverse is true for the summer. In modern times with food and warmth aplenty the imperative to do this declines. However, some people need more thyroid hormones in winter to prevent severe fatigue and depression. In this event Dr. Blanchard suggests "jump starting" followed by a different maintenance dose - so for example in the Autumn someone taking 100mcgms of T4 would have a jump start of 150mcgms for 3 days then maintenance dose of 110mcgms. In the Spring one would do the reverse - stop T4 for 3 days then return to the usual 100mcgms per day.

Some people only feel well using pure T3
At present we do not have biochemical tests to predict who these people are! A reverse T3 test may help but may not. If symptoms are typical of hypothyroidism but not responding to T4 or T4/T3 mixes, then a trial of pure T3 may be in order. T3 is short acting and must be taken at least 3, possibly 5 times daily. The smallest size tablet is prescription only tertroxin 20mcgms (equivalent to 100mcgms of T4). A starting dose would be 10mcgms split into 3 doses - tricky! I suggest crushing half a tablet and using a wet fingertip to take a third of the powder three times daily. One may know within a few days if this was making a difference, but a proper trial would be a few weeks. For details, see Paul Robinson's excellent book on the subject - Recovering with T3: My Journey from Hypothyroidism to Good Health Using the T3 Thyroid Hormone. (Amazon.co.uk link to "Recovering with T3: My Journey from Hypothyroidism to Good Health Using the T3 Thyroid Hormone").

For more detailed discussion see Thyroid Hormone Transport where the importance of pure T3 is explained in terms of transport of T3 across cell membranes.

The correct proportion of T4 to T3

Dr. Kenneth Blanchard maintains that the correct proportion is 98.5% T4 to 1.5% T3, for some a bit more, others less. Achieving this is difficult because Armour thyroid is 80% T4 and 20% T3. He uses slow release T3 - not available in UK. However transdermal T3 could be an option - watch this space!
The timing of dosing with T3 may be critical

Paul Robinson, in his excellent book on T3, hypothyroidism and the Circadian rhythm (Amazon.co.uk link to The Ct3m Handbook) has made the interesting observation that our circadian rhythms, essential to health, are determined by when hormones are produced. Since they work synergistically we need them to be produced at the same time. Timing is triggered by the pituitary gland, the conductor of the endocrine orchestra! It starts with TSH levels rising sharply at midnight and is followed by increases in T4, T3 and cortisol later in the night. As they come together they trigger wakefulness. Paul found out for himself, and proved it to his satisfaction through blood tests, that his health was further improved by taking his morning dose of T3 at 5.30am. See his website Recovering with T3 for his account of this.
Initial improvement followed by decline

Dr. Blanchard observed that some patients improved on thyroxin and then worsened. He describes a "sweet spot" of optimal levels of T4. He believes the reason for this is that TSH is partly responsible for converting T4 to T3 - so if levels of these hormones are too high, TSH is switched off and with that comes a switching off T4 to T3 conversion. T3 is the day hormone that fires us up and because T4 is slow acting there may be a delay in noticing this "switch off" of T4 converting to T3 and this can be clinically very confusing.

Monitoring treatment just by using a TSH can be misleading

In his article (follow the link below in External Links) Peter Warmingham cogently explains how just a TSH is not a good way to monitor replacement therapy. It is vital to measure levels of free T4, ideally free T3 as well, and assess the patient clinically - i.e. how do they feel? Are there any clinical symptoms of under or over dosing?

Finally, anyone who is hypothyroid for reasons other than autoimmunity, is likely to be iodine deficient.

Why are we seeing an epidemic of thyroid disease?

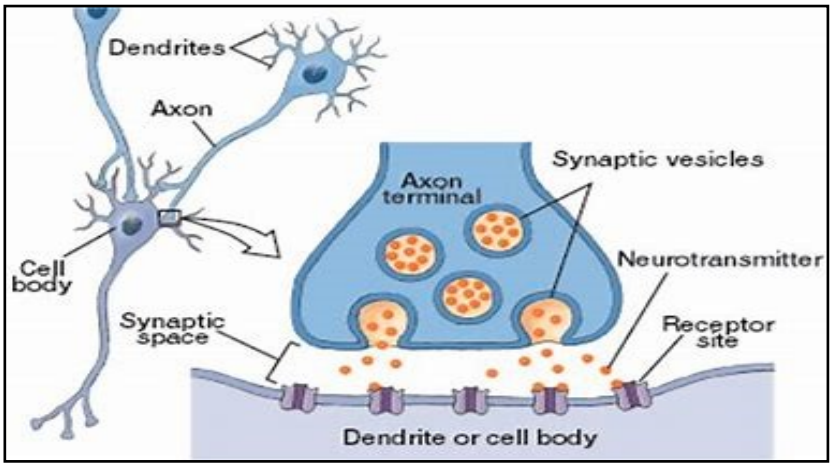
A whole range of chemicals have been shown to be goitrogenic (substances that suppress the function of the thyroid gland by interfering with iodine uptake) and/or suppressors of the HPA axis and/or suppressors of thyroid hormones uptake and/or suppressors of T3 uptake. These include perchlorates (washing powder), phthalates (added to plastics to increase their flexibility, transparency, durability, and longevity) and bisphenol A (in plastic wrappings), pyridines (cigarette smoke), PCBs and PBBS (fire retardants in soft furnishing), UV screens (sunblocks and cosmetics), and many others.

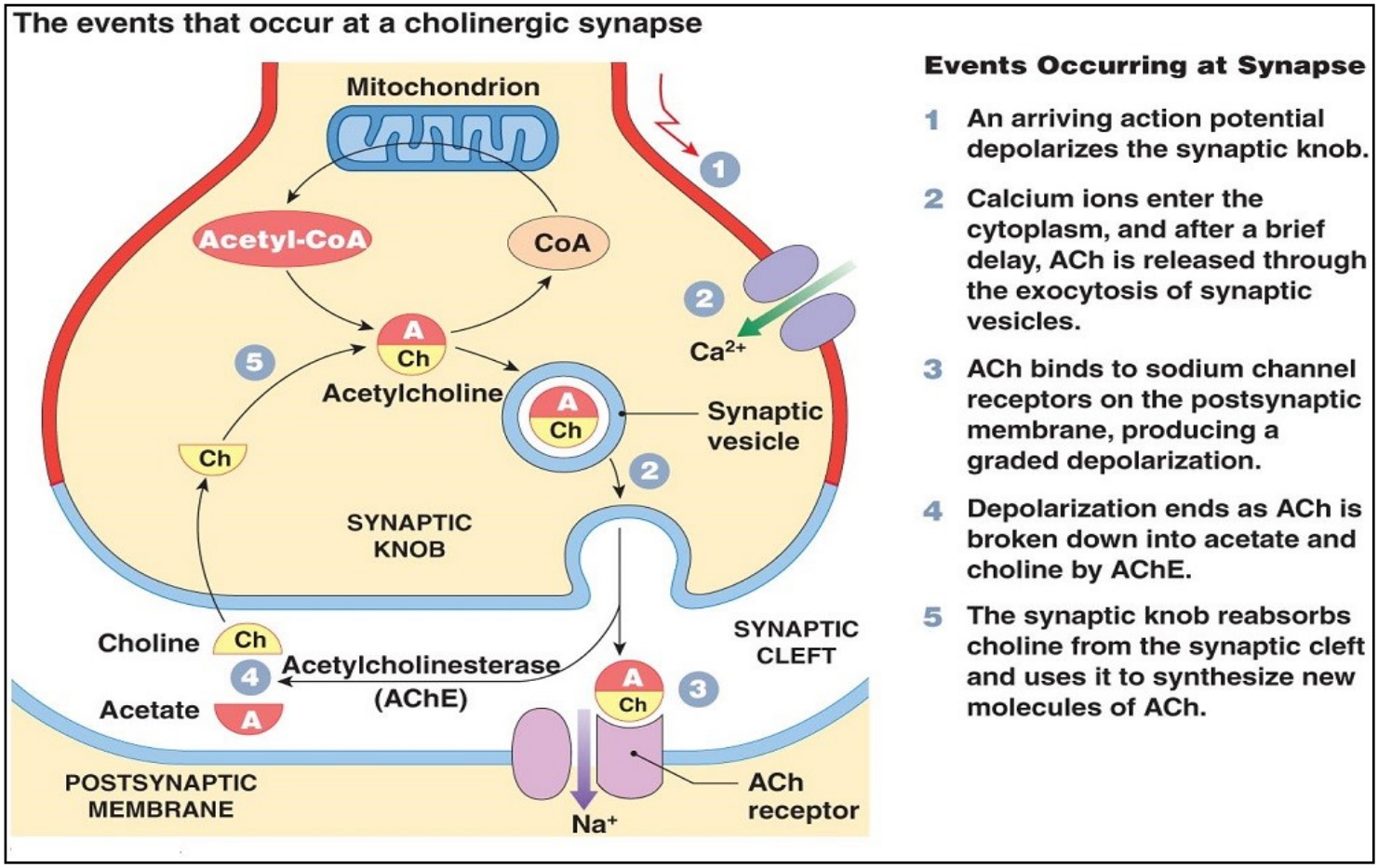
Photo corner *Mute swans at Potteric Car Doncaster with thanks to Amy Thompson*

Choline Esterase Inhibitors

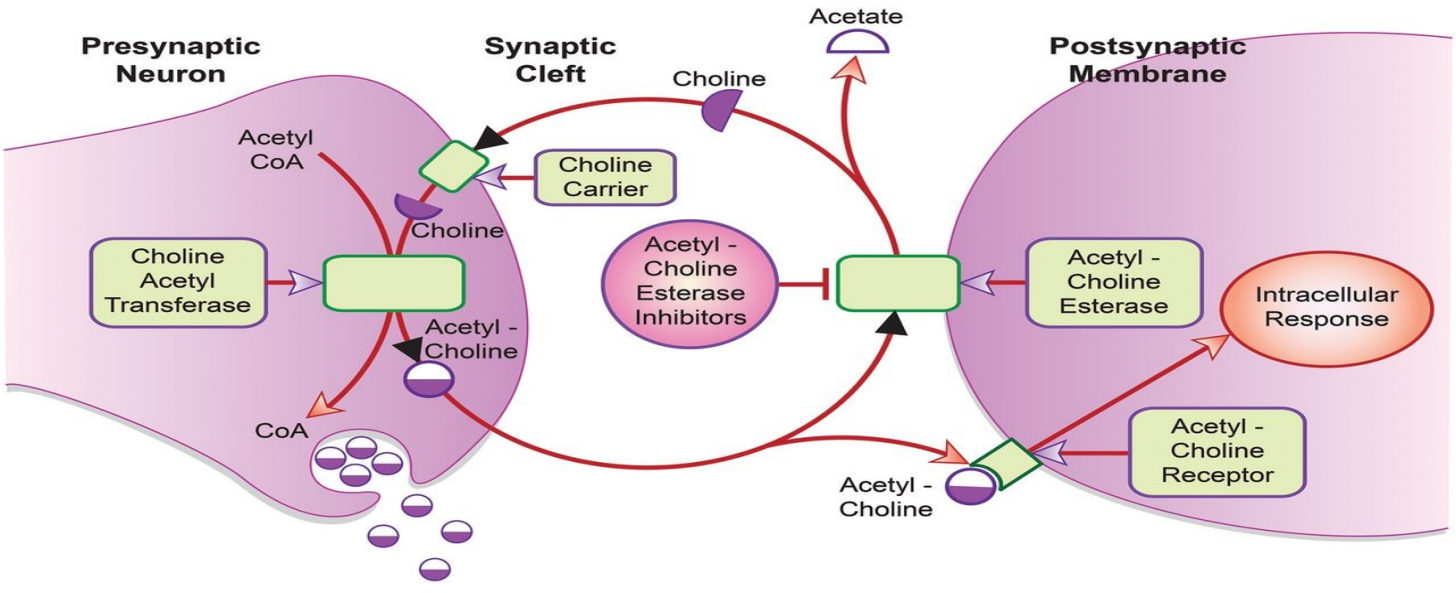
This group of substances has occupied the news headlines at the time of writing. I'm going to try and explain the issues involved and the implications and, this group of substances implications in ME/CFS

A synapse is a junction of two nerve cells which is like a connection plug in wiring. The following graphic explains the process which is standard teaching in schools.





Choline esterase inhibitors block the clearance of acetyl choline, which accumulates within the synapse. In effect this is like a tap being left open or the accelerator of a car jamming a full throttle.



An **acetylcholinesterase inhibitor** (often abbreviated **AChE**) or **anti-cholinesterase** is a chemical or a drug that inhibits the acetylcholinesterase enzyme from breaking down acetylcholine, thereby increasing both the level and duration of action of the neurotransmitter acetylcholine. Acetylcholinesterase inhibitors are classified as reversible, irreversible, or quasi-irreversible (also called pseudo-irreversible).

Uses

- Occur naturally as venoms and poisons
- Are used as weapons in the form of nerve agents
- Are used as insecticides
- Are used medicinally

Potential side effects of acetylcholinesterase inhibitors

Titration phase
When used in the central nervous system to alleviate neurological symptoms, such as rivastigmine in Alzheimer's disease, all cholinesterase inhibitors require doses to be increased gradually over several weeks, and this is usually referred to as the titration phase. Many other types drug treatments may require a titration or stepping up phase. This strategy is used to build tolerance to adverse events or to reach a desired clinical effect. This also prevents accidental overdose and is therefore recommended when initiating treatment with drugs that are extremely potent and/or toxic (drugs with a low therapeutic index).

mild – usually goes away	potentially serious
Diarrhoea Headache Insomnia Nausea Vomiting	Abdominal pain Lack of appetite Yellowed skin Dizziness Slow heartbeat Sudden or substantialweight loss Weakness

Reversible inhibitor
Compounds which function as reversible competitive or noncompetitive inhibitors of cholinesterase are those most likely to have therapeutic uses. These include:

Irreversible Inhibitors
Physostigmine, Neostigmine, Pyridostigmine, Rivastigmine, Galantamine, Caffeine, Donepezil, Edrophonium

Quasi-irreversible inhibitor
Compounds which function as quasi-irreversible inhibitors of cholinesterase are those most likely to have use as chemical weapons or pesticides. These include:
Organophosphates: Echothiophate, Cyclosarin, Dichlorvos, Sarin, VX , VE, VG, VM, Diazinon,

Medical uses

- To treat glaucoma
- To treat postural tachycardia syndrome
- As an antidote to anticholinergic poisoning
- To reverse the effect of non-depolarising muscle relaxants
- To treat neuropsychiatric symptoms of diseases such as Alzheimer's disease, particularly apathy
- To increase chances of lucid dreaming (by prolonging REM sleep).
- To treat Alzheimer's disease, Lewy Body Dementia and Parkinson's disease. In these neurodegenerative conditions AChEIs are primarily used to treat the cognitive (memory and learning deficits mostly) symptoms of dementia. These symptoms are attenuated due to the role of acetylcholine in cognition in the CNS. There is some evidence to suggest that AChEIs may attenuate psychotic symptoms (especially visual hallucinations) in Parkinson's disease.
- To treat cognitive impairments in patients with schizophrenia. There is some evidence to suggest efficacy in treating positive, negative and affective symptoms.
- As a treatment for autism and to increase the percentage of Rapid eye movement sleep in autistic children, in line with the mechanism by which they encourage lucid dreaming.
- In Myasthenia Gravis to compensate the antibody destruction of acetyl choline receptors.

UK Attack Salisbury. Novichok

Update:
Early April 2018
At the time of writing Sergei Skripal remains in hospital recovering. Yulia has regained consciousness and has been discharged. The policemen as been discharged from hospital..

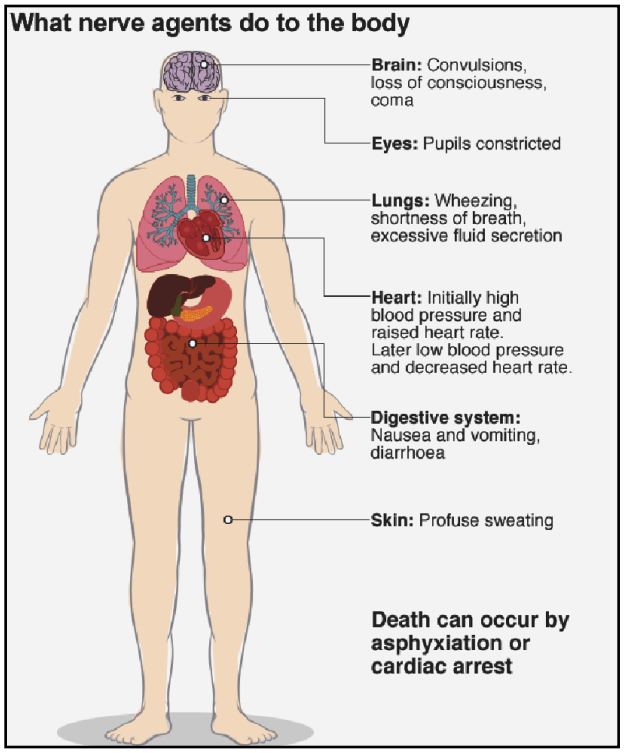
On 12th March 2018, the UK government stated that a Novichok agent had been used in an attack in Salisbury on 4th March 2018 to kill former GRU officer Sergei Skripal and his daughter Yulia. British Prime Minister Theresa May said in Parliament: "Either this was a direct action by the Russian state against our country, or the Russian government lost control of its potentially catastrophically damaging nerve agent and allowed it to get into the hands of others. On 14th March 2018, the UK expelled 23 Russian diplomats after refusing to meet the UK's deadline of midnight on 13th March 2018 to explain the use of the weapon.

After the attack, 21 members of the emergency services and public were checked for possible exposure, and three were hospitalised. Up to 12th March, one police officer remained in hospital. Five hundred members of the public were advised to decontaminate their possessions to prevent possible long-term exposure, and 180 members of the military and 18 vehicles were deployed to assist with decontamination at multiple locations in and around Salisbury. The exact location of the attack has not been released.

Daniel Gerstein, a former senior official at the U.S. Department of Homeland Security, said it was possible that Novichok nerve agents had been used before in Britain to assassinate Kremlin targets, but had not been detected: "It's entirely likely that we have seen someone expire from this and not realized it. We realized in this case because they were found unresponsive on a park bench. Had it been a higher dose, maybe they would have died, and we would have thought it was natural causes.

Effects

As nerve agents, the Novichok agents belong to the class of organophosphate acetylcholinesterase inhibitors. These chemical compounds inhibit the enzyme acetylcholinesterase, preventing the normal breakdown of the neurotransmitter acetylcholine. Acetylcholine concentrations then increase at neuromuscular junctions to cause involuntary contraction of all muscles. This then leads to respiratory and cardiac arrest (as the victim's heart and diaphragm muscles no longer function normally) and finally death from heart failure or suffocation as copious fluid secretions fill the victim's lungs.



The use of a fast-acting peripheral anticholinergic drug such as atropine can block the receptors where acetylcholine acts to prevent poisoning (as in the treatment for poisoning by other acetylcholinesterase inhibitors). Atropine, however, is difficult to administer safely, because its effective dose for nerve agent poisoning is close to the dose at which patients suffer severe side effects such as changes in heart rate and thickening of the bronchial secretions which fill the lungs of someone suffering nerve agent poisoning, so that suctioning of these secretions and other advanced life support techniques may be necessary in addition to administration of atropine to treat nerve agent poisoning.

In the treatment of nerve agent poisoning, atropine is most often administered along with pralidoxime, which reactivates acetylcholinesterase which has been inactivated by phosphorylation by an organophosphorus nerve agent and relieves the respiratory muscle paralysis caused by some nerve agents. Pralidoxime is not effective in reactivating acetylcholinesterase inhibited by some older nerve agents such as soman or the Novichok nerve agents, described in the literature as being up to 8 times more toxic than nerve agent VX.

The agents may cause lasting nerve damage, resulting in permanent disablement of victims, according to Russian scientists. Their effect on humans was demonstrated by the accidental exposure of Andrei Zheleznyakov, one of the scientists involved in their development, to the residue of an unspecified Novichok agent while working in a Moscow laboratory in May 1987. He was critically injured and took ten days to recover consciousness after the incident. He lost the ability to walk and was treated at a secret clinic in Leningrad for three months afterwards. The agent caused permanent harm, with effects that included "chronic weakness in his arms, a toxic hepatitis that gave rise to cirrhosis of the liver, epilepsy, spells of severe depression, and an inability to read or concentrate that left him totally disabled and unable to work." He never recovered and died in July 1992 after five years of deteriorating health.

Relevance to ME/CFS

Research by Dr. Puri and others suggest that there is depletion of acetyl choline levels in ME/CFS of cellular membranes. As choline esterase inhibitors increase acetyl choline levels, it was though that a mild drug of this type could help relieve fatigue. Galantamine has been trialed, and so far, does not seem to have made a significant impact.
.
Some sheep dips used to kill parasites and based on choline esterase inhibitors. Historically contact of agricultural workers with one of these agents has resulted in in case of ME/CFS. Historically some domestic insecticides used choline esterase inhibitors.

Choline esterase inhibitors have been tried to treat POTs, although alternatives medicines are now used.

Recipe Corner *by Carolyn*

Spring chicken in a pot

Casseroles aren`t just for winter, here is a light one filled with spring veggies to enjoy:-

Prep: 20 mins
Cook: 45 mins
Serves: 4
Freezable

Nutrition per serving:
Kcal 339, fat 9g, saturates 3g, carbs 27g, sugars 12g, fibre 8g, protein 36g, salt 0.5g

Cooking Method-

Heat the oil in a large, heavy pan. Add the onion, gently fry for 5 minutes until softened, add the chicken, then fry until lightly coloured. Add the potatoes, stock and plenty of freshly ground black pepper, then bring to the boil. Cover, then simmer for 30 minutes until the potatoes are tender and the chicken is cooked. (Can be frozen at this point if you wish).

Add the broccoli, spring greens, petit pois and spring onions, stir well, then return to the boil. Cover, then cook for 5 minutes more, stir in the pesto and heat through.

<u>Ingredients:-</u>

1 tablespoonful olive oil
1 onion, chopped
500g boneless, skinless chicken thigh
300g small new potatoes
425ml low-salt vegetable stock
such as found in good stock cubes)
350g broccoli, cut into small florets
350g spring green, shredded
140g petit pois (peas)
Bunch spring onions, sliced
2 tablespoonful pesto

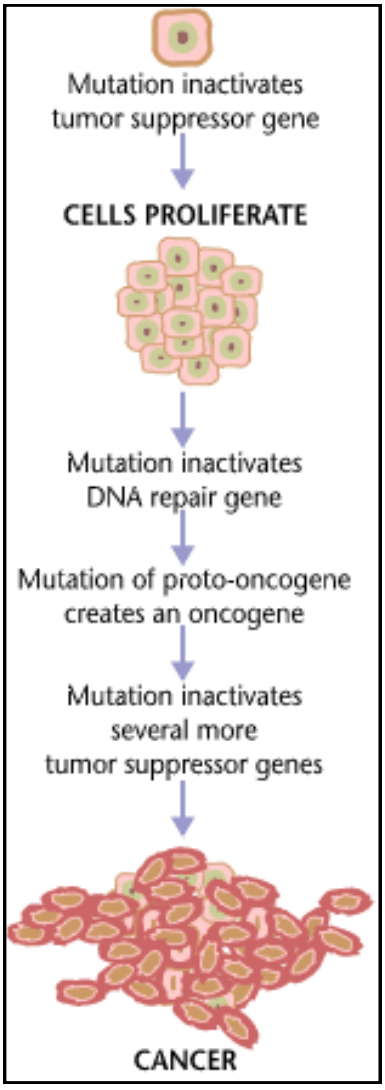
The Basics of Cancer. *with Thanks to Patient UK*

In 1971 President Nixon declared war on Cancer. The strategy involved efforts to find a cure for cancer by increased research to improve the understanding of cancer biology and the development of more effective cancer treatments, such as targeted drug therapies. The aim of such efforts is to eradicate cancer as a major cause of death. Despite significant progress in the treatment of certain forms of cancer, cancer in general remains a major cause of death 40+ years after this war on cancer began. New research directions, in part based on the results of the Human Genome Project, hold promise for a better understanding of the genetic factors underlying cancer, and the development of new diagnostics, therapies, preventive measures, and early detection ability.

What is cancer?

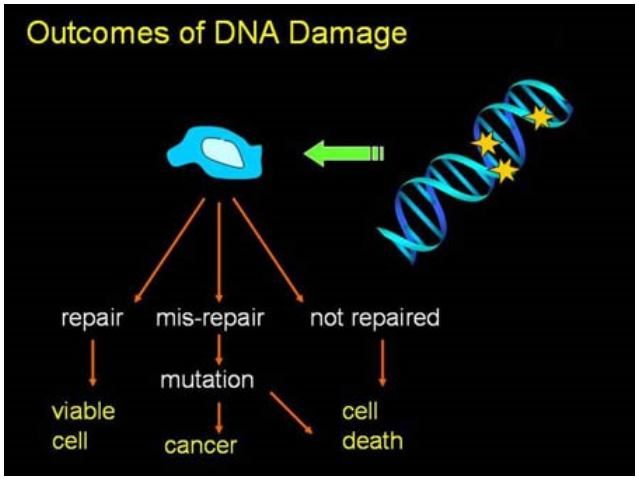
Cancer is a disease of the cells in the body. There are many different types of cell in the body, and many different types of cancer which arise from different types of cell of at least 200 types. What all types of cancer have in common is that the cancer cells are abnormal and multiply out of control. However, there are often great differences between different types of cancer. Some grow and spread more quickly than others and some are easier to treat than others, particularly if diagnosed at an early stage. Some respond much better than others to chemotherapy, radiotherapy, or other treatments, while some have a better outlook (prognosis) than others. For some types of cancer there is a very good chance of being cured. For some types of cancer, the outlook is poor. So, cancer is not just one condition. In each case it is important to know exactly what type of cancer has developed, how large it has become, whether it has spread and how well the particular type of cancer responds to various treatments. This will enable you to get reliable information on treatment options and outlook.

How do cancer cells come about?



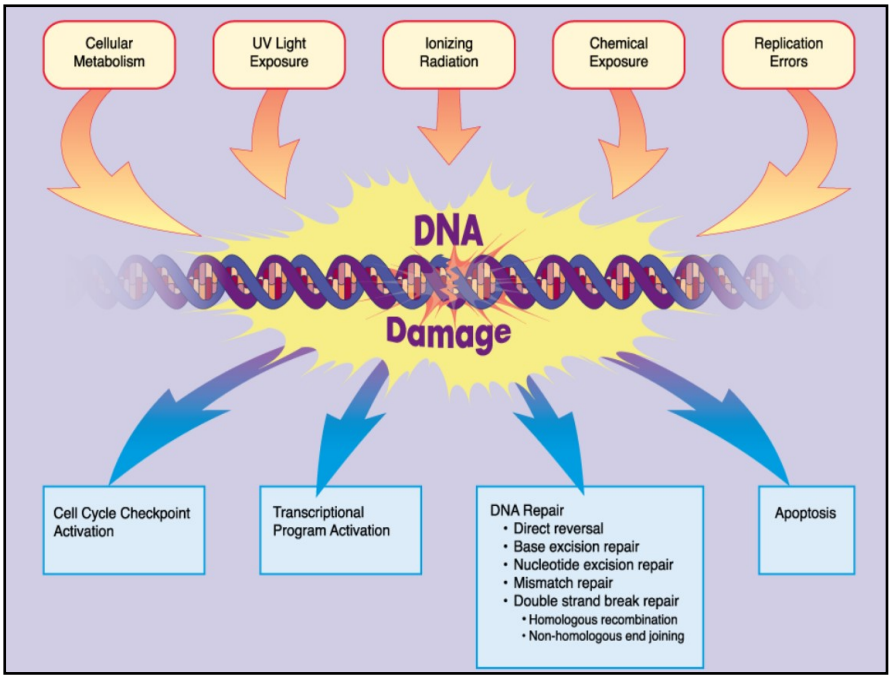
The body is made up from millions of tiny cells. Different parts of the body such as organs, bones, muscles, skin and blood are made up from different specialised cells. Most cells have a centre called a nucleus. The nucleus in each cell contains thousands of genes which are made up from a chemical called DNA. The genes are like codes which control the functions of the cell. For example, different genes control how the cell makes proteins, or hormones, or other chemicals. Certain genes control when the cell should multiply, and certain genes even control when the cell should die.

Most types of cell in the body divide and multiply from time to time. As old cells wear out or become damaged, new cells are formed to replace them. Some cells normally multiply quickly. For example, you make millions of red blood cells each day as old ones become worn out and are broken down. Some cells do not multiply at all once they are mature - for example, brain cells. Normally, your body only makes the right number of cells that are needed.



Sometimes a cell becomes abnormal. This occurs because one gene (or more) in the cell becomes damaged or altered. The abnormal cell may then divide into two, then four, then eight, and so on. Lots of abnormal cells may then develop from the original abnormal cell. These cells do not know when to stop multiplying. A group of abnormal cells may then form. If this group of cells gets bigger, it becomes a large clump of abnormal cells called a tumor.

What causes cancer?
We all have a risk of developing cancer. Many cancers seem to develop for no apparent reason. However, certain risk factors are known to increase the chance that one or more of your cells will become abnormal and lead to cancer. Risk factors fall into two groups.



Unmodifiable Risk Factors

Age
The older you become, the more likely it is that you will develop a cancer. This is probably due to an accumulation of damage to cells over time. Also, the body's defences and resistance against abnormal cells may become less good as you become older. For example, the ability to repair damaged cells, and the immune system which may destroy abnormal cells, may become less efficient with age. So, eventually one damaged cell may manage to survive and multiply out of control into a cancer. Most cancers develop in older people.

Infections
Some germs (viruses and bacteria) are linked to certain cancers. For example, people with persistent infection with the hepatitis B virus or the hepatitis C virus have an increased risk of developing cancer of the liver. Another example is the link between the human papillomavirus (HPV) and cervical cancer. Most (possibly all) women who develop cervical cancer have been infected with a strain (subtype) of HPV at some point in their lives. Another example is that a germ (bacterium) called Helicobacter pylori is linked to stomach cancer. One research study estimated that about one in six cancers - two million a year globally - are caused by largely treatable or preventable infections. They estimated that four infections - HPV, H. pylori, and hepatitis B and C viruses - accounted for 1.9 million cases of cervical, stomach and liver cancers in 2008. Most of these were in the developing world. Initiatives such as immunisation against HPV and hepatitis B are helping to combat these infections.
But, most viruses and viral infections are not linked to cancer.

Immune system
People with a poor immune system have an increased risk of developing certain cancers. For example, people with AIDS, or people on immunosuppressive therapy, e.g. transplant patients.

Genetic makeup
Some cancers have a strong genetic link. For example, in certain childhood cancers the abnormal gene or genes which may trigger a cell to become abnormal and cancerous are inherited. Other types of cancer may have some genetic factor which is less clear-cut. It may be that in some people their genetic makeup means that they are less resistant to the effect of carcinogens or other factors such as diet. Another example is BRCA gene mutation is found in families with a multiple history of breast cancer.

Modifiable Risk Factors

Chemical carcinogens
A carcinogen is something (chemical, radiation, etc.) which can damage a cell and make it more likely to turn into a cancerous (malignant) cell. As a rule, the more the exposure to a carcinogen, the greater the risk. Well-known examples include:

Tobacco. If you smoke, you are more likely to develop cancer of the lung, mouth, throat, oesophagus, bladder and pancreas. Smoking is thought to cause about 1 in 4 of all cancers. About 1 in 10 smokers die from lung cancer.

The heavier you smoke, the greater the risk. If you stop smoking, your risk goes down considerably.

Workplace chemicals such as asbestos, benzene, formaldehyde, etc. If you have worked with these without protection you have an increased risk of developing certain cancers.

Lifestyle factors:
Diet and other lifestyle factors (and, as mentioned, smoking) can increase or decrease the risk of developing cancer. For example: If you eat a lot of fruit and vegetables you have a reduced risk of developing certain cancers. The exact way in which they protect against cancer is not fully understood. These foods are rich in vitamins and minerals, and contain chemicals called antioxidants. They may protect against damaging chemicals that get into the body. We should all eat at least five portions of fruit and vegetables per day.

Red meat:
There is strong evidence that eating a lot of red meat (such as beef, pork and lamb) increases your risk of bowel cancer and stomach cancer.

Processed meat:
Processed meat also increases your risk of cancers, especially bowel cancer. Processed meat means meat that has been transformed through salting, curing, fermentation, smoking or other processes, e.g. bacon, salami, chorizo, pepperoni and all types of ham.

Alcohol
The risk of developing certain cancers is increased by lack of regular exercise or drinking too much alcohol.

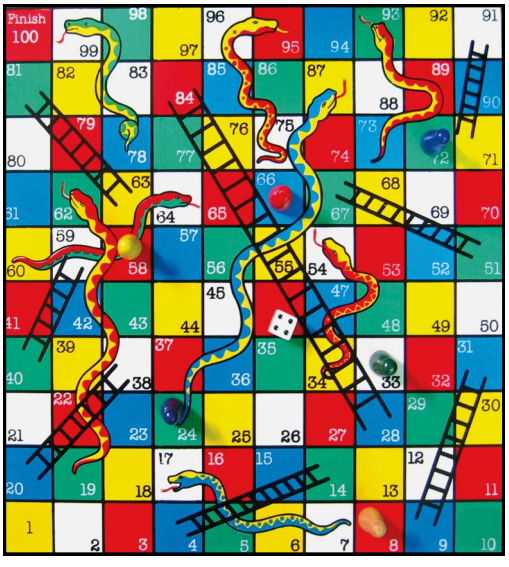
Obesity:
Research has shown that many types of cancer are more common in people who are overweight or obese, including cancers of the breast, bowel, lining of the womb (endometrium), oesophagus, pancreas, kidney, liver, stomach, ovary, thyroid, myeloma, and brain (meningioma).

Radiation:
Radiation is a carcinogen. For example, exposure to radioactive materials and nuclear fallout can increase the risk of leukaemia and other cancers. Too much sun exposure and sunburn (radiation from UVA and UVB) increase your risk of developing skin cancer. The larger the dose of radiation, the greater the risk of developing cancer. But note, the risk from small doses, such as from a single X-ray test, is very small.

Most cancers are probably due to a combination of factors.

Not everybody who has contact with a potential cancer-causing substance (carcinogen) or has an unhealthy lifestyle will develop cancer. For example, not all smokers develop cancer of the lung. In fact, we are all probably exposed to low doses of carcinogens a lot of the time.

The body has certain mechanisms which may protect us from developing cancer. For example, it is thought that many cells which are damaged by carcinogens can repair themselves. Also, the body's immune system may be able to destroy some types of abnormal cells before they multiply into a tumor. Perhaps one carcinogen may only damage one gene, and two or more genes may need to be damaged or altered to trigger the cells to multiply out of control. In many cases it is likely that there is a combination of factors (such as genetic makeup, exposure to a carcinogen, age, diet, the state of your immune system, etc.). These may play a part in triggering a cell to become abnormal and allowing it to multiply out of control into a cancer. One expert described the development of cancer as like a game of snakes and ladders. The higher the risk factors, the more ladders, where as you can add more snakes by reducing the risk factor.

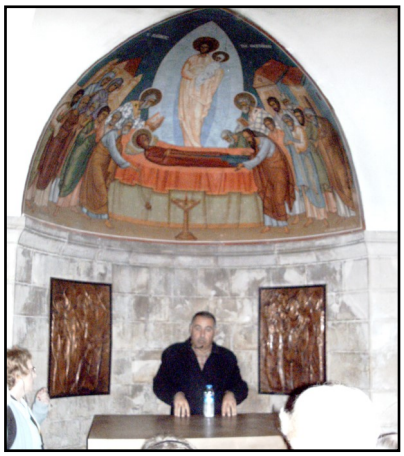


North of Doncaster. *Personal comment by Trevor Wainwright*

Travel Diary to the Holy Land Part 5:

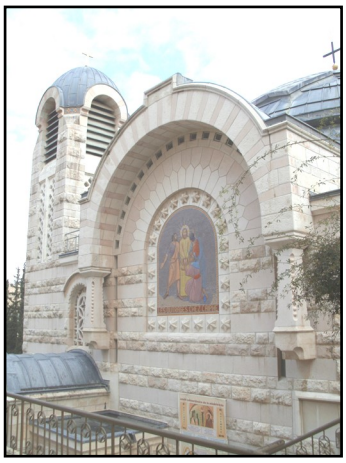
The day after visiting Shepherds Field we were starting a bit later, so I used the time for a bit of local sightseeing, taking first a view of Jerusalem outside the old city walls, followed by pictures of the imposing Tower of David Museum. My attention was drawn to an old British style post box and what the notice said: Christ Church the oldest Protestant Church in the Middle East. It was a complex not only with church but with a museum, accommodation and study centre. I visited the church; on the walls was an art exhibition by the group 'Jews for Jesus' a Messianic Jewish non-profit organisation founded in 1973. After a walk in the rear garden I headed back to join my fellow pilgrims and once more follow in the footsteps of Jesus.

Our journey took us to The Zion Gate on the Old City Wall, we dismounted and looked at the damage caused by gun fire in the Six Day War, so reminiscent of the times I was Karlovac, Croatia 1994 to Bihac, Bosnia 1995 and Sarajevo, Bosnia 1996, the memories came flooding back. Then it was to Mount Zion where the earliest Christian Community lived following the resurrection. Our first port of call was The Abbey of the Dormition of the Virgin Mary, now called Hagia Maria Sion Abbey the name was changed in 1998 in reference to the church of Hagia Sion (Holy Zion) that formerly stood on this spot in the 5th Century which was destroyed by the Persians in 614. We passed its high bell tower before reaching the main body of the Dormition.

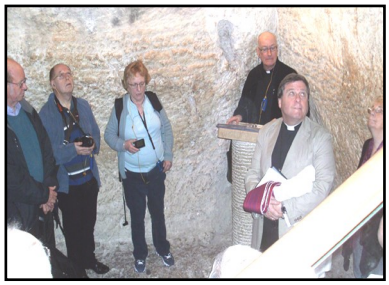


The inside was circular and remarkable for its simplicity and beauty. At the centre of its semi-circular apse is a mosaic of Mary and of the child Jesus, with the figures of twelve prophets below them. Around the church are six side chapels decorated by beautiful mosaics depicting scenes such as Mary and the infant Jesus receiving pilgrims, Jesus' family tree, John the Baptist on the shore of the Jordan River, St. Benedict – the founder of the Benedictine order, and other saints. We made our way down to the crypt, a round pillared room with a sculpture of Mary "asleep" in the centre, it was within touching distance, I laid my hand gently on her head as a mark of respect. On the ceiling above her was the figure of Jesus, as if watching over her, surrounded by the great women of the Bible: Beyond this main room, are several other chapels and altars donated by various countries. One mosaic was the assumption of Mary where she is carried to heaven in the arms of Jesus as a child but with a woman's face, anther was of the 12 Apostles again this time the12th being co-opted his name was either Paul or Mathias.

Leaving the Dormition we made our way to The Cenacle also known as "Upper Room", a room in the David's Tomb Compound, traditionally held to be the room of the Last Supper. So, called because it is the upper floor of a two-story building, it is the minaret of a Muslim mosque. Immediately beneath it is the Jewish shrine venerated as the shrine of King David (though he is not buried there). A plain room but the architecture tells the story. The Crusader period is depicted by a slender marble column supporting a stone canopy in the south-west corner. Carved into the capital at the top of the column are two young on the blood their mother has drawn from her breast symbolising Christ giving his blood for the salvation of humankind. In the 16th century, after the Turks captured Jerusalem, the room was transformed into a shrine to the memory of the prophet David. Its mihrab (a niche indicating the direction of Mecca) and stained-glass windows with Arabic inscriptions remain. A more modern gift, a bronze olive tree from Pope John Paul II during his pilgrimage there stood in a quiet corner not looking at all out of place.



Then it was to The Church of St Peter in Gallicantu, (Cock crow in Latin), A Byzantine shrine dedicated to Peter's repentance, said to be built on the site of the House of Caiaphas the High Priest, where Jesus spent his last night on earth. Just above the main entrance was an image of Jesus being mocked. In the courtyard a statue depicts the events of the denial and include the main figures; the cock, the woman, and the Roman soldier. The inscription includes the biblical passage; But he denied him, saying "Woman, I know him not" Further images showed Peter facing Jesus after denying him three times, Peter full of remorse after his denial and Peter's commissioning where he is asked by Jesus three times if he loves him, due to his three denials, each time Peter answers "Yes".



The church is cut into the stone under which are prison cells one of which is said to have held Jesus the night he was arrested. We made our way down to the cells, me little knowing what an emotional moment it would be. We made our way down the steps, Jesus went down lowered by a rope through a hole in the floor where he would have been untied, then most likely fastened to the walls. The straps were still there. We held mass in the prison room beneath the entry hole. Then I was beckoned by Bishop Tony, I introduced my poem “Denial” written in the 1990’s based on Peters’ perspective and his guilt part of a trilogy. I began slowly as the poem progressed my voice resounded off the walls as if being gently coaxed from me, and being given meaning, the emotion of the moment slipped quietly in to my recital it was almost as if I had done the denial myself, tears were welling in my eyes as I finished, it had been a truly emotional recital.

Back in the main body of the church, I stood briefly once more before the main altar looking up at what is perhaps the most striking feature of the interior, the ceiling, which is dominated by a huge cross-shaped window designed in a variety of colours. Walking out I admired the Stations of the Cross also lining the walls, marked with simple crosses. Outside, we saw all around the yard of the church many of the ancient buildings from the different periods in Jerusalem's history. The excavations, conducted in 1888 revealed the Byzantine church, Byzantine roads and houses, a rock-hewn bath house, and parts of the aqueduct. They also revealed a stepped road, which could well have been the steps down which Jesus went with his disciples and crossed the Kidron valley to the Garden of Gethsemane and the same ones up which he was taken to the house of Ciaphas the High Priest. These were shown on carved murals in the grounds at the top of the stairs.

Re-entering the Old City through the Damascus Gate and heading on through the narrow streets passing a white car with the initials UN on its bonnet and front doors, a reminder that things are far from settled. After a service at the 1st Station the Church of Condemnation where Jesus was condemned to be crucified and given his cross before then it was along the narrow streets following the way of the cross, something I had wanted to do for a long time and now it was happening, I was trying to imagine what it would have been like had they been crowded with jeering onlookers at the time. We passed street traders I imagined them selling souvenirs of the event. We stopped to commemorate with a prayer, each one a different topic but with the same aim, each station depicting what happened there.
We passed a group of tourists taking pictures, with cameras on tripods reminding me of the media,

what they would have made of the event had it been happening today. I then realised there would have been Roman soldiers pushing the crowds back, and the buildings that made the street narrow might not have been built then. We came out into the courtyard at the Church of the Holy Sepulcher. A crowd had gathered as there had been a dignitary arriving. They were looking towards the church as if looking at Jesus on the cross a variety of expressions each with its own thoughts as if they were watching him die. One woman was knelt taking a picture. For a moment I imagined her on the day knelt in prayer; others seemed to be milling about as if waiting for something to happen.
Just before we went into the church Joseph told us the story of the keys. *To be continued....*