The Heart of the Matter – Beyond the Cholesterol Myth

By Angela Frieswyk, BSc

Awareness of the 'cholesterol myth' has grown, not only within the 'alternative health' sector, but now amongst the general public. While mainstream healthcare has been slow to look beyond the cholesterol agenda, the drug industry has enjoyed immense profits from pharmaceutical statins. Prescription rates for cholesterol lowering drugs in New Zealand and Australia have soared over the past decade and are now estimated to be prescribed to three in ten people over the age of 45 years in Australia[6], higher than European countries.

The aim of this article is to briefly recap on the 'cholesterol myth', delve into other markers of cardiovascular disease risk beyond elevated cholesterol levels and consider a better model for treatment. In doing this I hope to put cholesterol's rightful place in human physiology back in context.

Cholesterol – essential for life

Cholesterol is a natural substance that can be either obtained from our diet or produced in the liver. Cholesterol is required to help provide structure, strength and flexibility to cell membranes. It forms the backbone of bile (which emulsifies fats during digestion) and a myriad of hormones. It is required for the synthesis of vitamin D, a deficiency of which is linked to many chronic health complaints, including heart disease. Immune function is also dependent on vitamin D. We start life with it by being breastfed on our mother's milk, a rich source of both saturated fat and cholesterol that are necessary for normal growth, particularly the development of the brain.

We have all heard about 'good cholesterol' and 'bad cholesterol', the same cholesterol but with differences in density. High density lipoprotein (HDL or 'good cholesterol') acts like a sponge taking excess cholesterol from the cells back to the liver, where it is either recycled or passed out via bile through the digestive system. Low density lipoprotein (LDL or 'bad cholesterol') takes cholesterol from the liver into the blood stream for delivery to cells for repair and hormone synthesis. While we don't want too much cholesterol being recycled into the blood stream where plaque formation may occur (given the initiators to do so), neither do we want too little cholesterol available for cell repair, vitamin D and hormone synthesis. Hence we can see that both HDL and LDL are each essential in their roles, so the monikers "bad" versus "good" are more about the appropriate balance (which a cholesterol ratio test helps identify).

Initiators of arteriosclerosis

Plaque formation does not occur simply due to having elevated cholesterol. Current understanding suggests the process begins with damage to the artery wall that triggers a rise in a type of modified LDL (oxidised sticky cholesterol), an inflammatory immune response and an increase in clotting around the area. In the immediate term this response is essential to protecting the damaged area. However, should the driver of inflammation continue, then the very process that protects the damage will compound, resulting in a thicker and thicker disposition of cholesterol and increases the risk of clotting episodes. Simply lowering cholesterol does not therefore address the underlying driver; inflammation and oxidative stress.

Inflammation and oxidative stress

Many processes will drive inflammation and oxidative stress, but the most concerning are smoking, high blood pressure (familial, plus stress, fluid retention and other secondary drivers), elevated blood sugar levels and elevated triglycerides (high sugar/carbohydrate diets, alcohol, plus lack of exercise and obesity), nutritional deficiencies, chemical exposure and unresolved infection[1]. Smoking can be avoided, nutritional deficiencies can be identified and corrected, chemical exposure limited (and treated); plus elevated blood sugar, triglycerides, stress and blood pressure can all be somewhat managed. But what about the more stealth like processes such as oxidative chemical damage and unresolved infection, particularly when symptoms are not significant enough to be noticed or are sufficiently ignored. It's worth pointing out here that infections thought to be linked to cardiovascular damage include infected teeth (including root canals), Chlamydia pneumonia[1], herpes viruses[1], peptic ulcers[3] and gastritis[3].

A blood test that can prove useful in this context (and in conjunction with a proper clinical assessment) is 'highly sensitive C-reactive protein' (hs-CRP). This marker is elevated when there is an inflammatory response in the body, either due to infection or injury. This is recognised by the American Heart Association as a risk marker for heart disease but unfortunately, unlike cholesterol testing, this test is not offered as a general public screening test and is typically only checked for those already known to be at risk. Highly sensitive-CRP does have limitations in that it does not pinpoint where in the body inflammation is occurring. However, raised levels of hs-CRP (particularly levels above 3.0 mg/L) have been found to be more significant risk indicators of cardiovascular disease than cholesterol alone, particularly when coupled with unfavourable cholesterol ratios[3]. The later stage of the clotting cascade that is driven by ongoing inflammation can also be identified with a fibrinogen blood test, elevated levels of which can also show an elevated risk of developing cardiovascular disease.

Outside of mainstream diagnostics, traditional naturopathic diagnostics (clinical assessment, iridology, kinesiology, etc) and certain electronic diagnostic equipment (eg EAV, Vega and electro-dermal computer screening) may help identify sources of infection, inflammation or other stealth factors that maybe driving the underlying pathology. Accuracy of information obtained from diagnostic tools, is of course, dependent on the skill, insight and clinical experience of the practitioner interpreting the information.

What about homocysteine?

Homocysteine is an amino acid produced by the body, typically as a by-product of consuming meat. In the 1990s it was recognised that elevated homocysteine levels were linked to an increased risk of stroke, atherosclerosis, DVT and peripheral artery disease. Normally homocysteine is metabolised into amino acids cysteine and methionine, but some people have genetic factors that can limit this conversion. The conversion of homocysteine is also limited when there are deficiencies of vitamin B6, B12 and folic acid. Low levels of these vitamins are common in those with poor diets, alcoholics and chronic kidney disease. Supplementation with these nutrients (particularly folic acid) has been shown to lower homocysteine levels, though it is as yet unclear whether this has a flow-on effect of actually lowering cardiovascular disease. 5%-12% of the general population has elevated homocysteine levels. While elevated homocysteine is not ranked as high a risk factor as elevated hs-CRP with a raised cholesterol ratio, given the high prevalence of nutritional deficiencies it is worth screening for.

Healthy dietary cholesterol versus oxidised cholesterol

While the cholesterol myth has exposed the erroneous beliefs about dietary cholesterol, it is important that we discern between sources of normal dietary cholesterol and that of highly toxic oxidised cholesterol. Upon exposure to oxygen and heat, particularly high heat, cholesterol will oxidise, creating a toxic food that needs to be ranked amongst other well known toxic fats such as hydrogenated fats (eg margarines and shortening), excess intake of polyunsaturated oils (eg particularly corn oil and soybean oil) and trans fats. Oxidised cholesterol is much more sticky, which not only encourages atherosclerotic plaque formation, it is also linked to destructive immune reactions that can damage the arterial lining (a "catch-22" situation). In fact early researchers flawed many dietary choles-

terol experiments by using oxidised cholesterol. The researchers used purified pharmaceutical grade cholesterol in many animal studies, which was thought to be reflective of dietary cholesterol, but was in fact highly oxidised from being purified and then stored in jars on the laboratory shelf. When this type of cholesterol was mixed into the diet of the unfortunate laboratory animals it was shown to induce lesions in the arteries within 24 hours of being administered.[2]

While there will always be some degree of oxidation during cooking and storage, it is the degree of heat and oxygen exposure that is significant, along with the amount of oxidised food that is regularly consumed in our diets. Fried foods, fast foods and processed foods are obvious culprits. Consider the amount of processing that occurs in the mass production of a fast food burger pattie, which is then cooked over a high heat in poor oils. Less obvious examples are dried milk powder and powdered eggs yolks. These are created using a spray drying process that forces the milk or egg yolk through very tiny holes at high temperatures and pressure. Sadly these commercial ingredients end up in foods that are often fed to our very youngest.

It is important to minimise your intake of processed foods, maximise your intake of fresh antioxidant rich produce and balance your diet with healthy proteins and fats (eg slow cooked meals made from pasture fed animals, organic free range poultry and deep sea / cold water fish). For more sound dietary wisdom try reading *Nourishing Traditions* by Sally Falloon, *Know Your Fats* by Mary Enig, PhD or refer to the Weston A. Price Foundation website; www.westonaprice.org.

Is there a case for lowering cholesterol blood levels?

Not without good reason to do so (e.g evaluate your risk factors) and not without first addressing and treating sources of infection, oxidation, inflammation and making appropriate dietary changes, particularly avoiding oxidised cholesterol and lowering sugar, alcohol and excessive intake of grains. The aim of directly treating cholesterol should always be to correct the cholesterol ratio (total cholesterol: HDL), not to aggressively lower cholesterol to levels where normal physiology is hindered (such as reducing your synthesis of vitamin D and hormones). Lowering cholesterol to unnaturally low levels is usually only achieved by using pharmaceutical statin medication, which comes with a notorious list of potential side effects, including (but not limited to): muscle, nerve, kidney and liver damage, plus an increased risk of diabetes and memory loss.

There are, however, many natural options that can be trialed for lowering cholesterol, including garlic, lecithin, guggul extract, policosanol, plant phytosterols or herbs that encourage bile flow and gallbladder/liver function such as globe artichoke. I have used several treatments over the years and have seen success as well as failure, but to date the most effective treatment that I prescribe is certainly red yeast rice.

Red yeast rice – A gentler statin

Red yeast rice is a fermented product of rice in which red yeast (*Monascus purpureus*) has been grown. It has been used in Chinese cuisine (it is an ingredient in the world famous dish "Peking duck") and as a medicinal food to promote "blood circulation" for centuries and is still used in China and worldwide today. Several trials exist that confirm the cholesterol lowering effect of red yeast rice, the first coming from China in 1997. This trial used 324 patients with high total cholesterol, high LDL and low HDL using 1.2gm of red yeast rice per day over eight weeks, versus a control group. The red yeast rice group reduced total cholesterol by 23%, LDL by 31%, triglycerides by 34% and increased HDL levels by 20%.[4] Later in 2002 it was first documented that the mode of action for the cholesterol lowering effect was due to a family of naturally occurring substances produced by red yeast activity. These substances were in fact monacolins, naturally occurring statins that are known to inhibit cholesterol synthesis in the liver cells. This put red yeast rice manufacturers in the spotlight for a legal battle with the FDA and Merck & Co who manufacture Mevacor ('Lovastatin'), the purified and concentrated statin, monacolin K. Mevacor asserts that monacolin is a patented pharmaceutical. It's that common theme, when something natural works well enough, it must be isolated, owned and made profitable.

But is red yeast rice therefore any different to a pharmaceutical statin? Yes, it is more aptly known as the 'gentler statin'. Red yeast rice contains a family of nine different monacolins that naturally occur at much lower doses than purified monacolin K alone. A trial that used 2.4gm of red yeast rice was found to provide less than ¼ of the corresponding lowest prescribed dose of monacolin K (4.8mg, versus 20-40mg/day doses of monocolin K or 'Lovastatin'). Red yeast rice also contains other cholesterol balancing substances including plant phytosterols, isoflavones and monounsaturated fatty acids. It is thought that the effectiveness of the low dose of monacolins within red yeast rice is by virtue of a synergy of all of these ingredients.

Despite the fact that red yeast rice has been proven to be an effective alternative to pharmaceutical statins, the natural health industry has been muzzled due to the above legal battle. In New Zealand this still remains somewhat of a grey area, while in Australia the TGA has declared it illegal to make any claims or give instruction on using red yeast rice to lower cholesterol. In the US it is a violation of the Federal Food, Drug and Cosmetic Act to market red yeast rice as a cholesterol lowering agent. The FDA's rationale is that it is an unapproved drug, yet is happy to approve statin medication despite a trail of physiological havoc.

Red yeast rice is unlikely to ever be an approved 'drug' as monopoly profits cannot be gained from a non-patentable food substance. However, as it has always been a traditional food, current law allows it to continue to be available on the market providing natural health industry does not make any cholesterol lowering claims. Very few side effects from red yeast rice occur, including no evidence of liver or kidney problems[5]. However, red yeast rice may potentially reduce the production of co-enzyme Q10 with long term use, albeit not as aggressively as statin medication (I would question the need for long-term use except in a very few cases of familial high cholesterol production). It is therefore prudent to supplement with CoQ10 (or more ideally ubiquinol) when using red yeast rice. Red yeast rice should never be used alongside pharmaceutical statins medication and certain other medications. I always recommend that people use red yeast rice extract with advice from an experienced practitioner and monitor their cholesterol tests. Although red yeast rice has proven to be consistent in effectiveness, there are many other natural cholesterol lowering agents that could be trialed first.

There is so much more on the topic of cholesterol metabolism and cardiovascular disease that hasn't been included in this article, including magnesium therapy for preventing arterial calcification (calcium deposits) and for regulating cardiac muscle function; and the emotional-physical link to heart disease. This topic can be overwhelming, but a good place to start is to seek an experienced natural health practitioner to help you identify your key weaknesses and prescribe the most appropriate treatment, diet and lifestyle changes. Change rarely occurs without effort and most importantly, in the words of Confucius ... "Wherever you go, go with all your heart".

About the Author

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