

C-Reactive Protein: Turning a Symptom into a Disease

*“August 4, 2002, heralded and trumpeted across the headlines of American newspapers that medicine has “discovered” that inflammation of the blood vessels, marked by a high **C-Reactive Protein** level in the blood, is the major cause of heart disease.”*

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In my article, “Medicine ‘Discovers’ C-Reactive Protein,” I noted that holistic practitioners have long (decades) recognized that a high C-Reactive Protein (CRP) level is an indicator of blood vessel inflammation. Further, this is indicative of a severe bioflavonoid deficiency. In thousands of CRP cases, we have successfully eliminated the **bioflavonoid deficiency** with a **buckwheat phytochemical complex**. This routinely quells viral and other infections and inflammations in blood vessels and brings high CRP levels down to normal—lowering the risk of stroke, heart disease, and heart attack.

But mark my words—now that medicine has ‘discovered’ this symptom, it will be turned into a disease for which pharmaceutical companies are already gearing up. Rather than treat the underlying cause of the problem by supplying whole, natural, bioflavonoid phytochemical complexes like buckwheat, the medical profession will turn to powerful drugs to treat the “new disease”.

Even now, without an understanding of how to resolve nutritional—deficiency disease, doctors are stymied about what to do for a patient with this newly discovered high CRP level. Most physicians turn to statin (cholesterol-lowering) and blood pressure drugs. This can compound the problem, does not resolve the inflammation in the blood vessels, and leaves the underlying problem fermenting and still in place. The only difference is that the body now has to deal with the underlying problem plus the new problems that are caused by the drugs.

But this will just be the tip of the iceberg. Once the pharmaceutical houses get into high gear, CRP will take the place of cholesterol as a household word. This new “disease” will be discussed with buzzwords like hyper(c) proteinaemia. And the new high-tech drug buzz will be about things like IL-6. So be forewarned. None of these things will work, but they will make billions of dollars for the drug companies.

Instead, treat the underlying cause of the problem with **Cyruta-Plus** from Standard Process at a dose of six daily for six to twelve weeks. This product is made directly from organic buckwheat plants, processed perfectly without heat to retain the entire live vitamin C and bioflavonoid complexes.

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Syndrome X and C-reactive Protein

By: David Seaman

Syndrome X is most easily viewed as a pre-diabetic state, as it is characterized by hyperinsulinemia and hyperglycemia. Syndrome X can initially exist without symptoms and, as it progresses, symptoms such as fatigue can develop, which are considered a “normal” part of aging, rather than the outcome of a pathological state.

Metabolic features of Syndrome X:

1. Increased production of pro-inflammatory mediators: C-reactive protein (CRP), nuclear factor kappa-B (NF- κ B), tumor necrosis factor (TNF), and free radicals
2. Increased platelet aggregation—platelet hyperaggregability
3. Reduced degradation of fibrin
4. tonic vasoconstriction; reduced vasomotor regulation
5. Promotes a pro-thrombotic state
6. Promotes greater infarct size
7. Leads to heart disease and stroke

As outlined above, there are numerous pro-inflammatory factors involved in syndrome X. Perhaps the most well studied is C-reactive protein. CRP was discovered seventy years ago when scientists were studying the human inflammatory response. It was initially characterized as an acute-phase mediator that was released chronically as a part of an ongoing sub-clinical pro-inflammatory state. Consider the diverse functions of CRP in the pathogenesis of heart disease.

Pro-inflammatory aspects of CRP:

1. Localizes in atherosclerotic intima, but not normal intima
2. Induces the production of adhesion molecules
3. Reduces endothelial derived relaxing factor, which is also known as endothelial-nitric oxide
4. Induces the production of plasmin inhibitors which lead to increased fibrin deposition
5. Triggers the oxidation of LDL-cholesterol
6. Mediates the uptake of LDL by macrophages
7. Blunts normal endothelial vasoreactivity
8. Recruits circulating monocytes into the arterial wall, and their subsequent activation
9. Stimulates complement activation

When I was in Chiropractic College, as part of our training, we did blood test on our patients, and CRP and ESR were part of the panel. Back in those days (1986) the CRP we tested for was never elevated unless there were overt trauma or infection or, perhaps, rheumatoid arthritis. Otherwise CRP was never elevated.

In recent years, a new high sensitivity CRP (hsCRP) has been developed, which can be used as a screening test for patients. Ridker indicates that many physicians screen for hsCRP, because it is associated with a markedly increased risk of myocardial infarction, stroke, peripheral arterial disease, and sudden cardiac death, even among apparently healthy individuals with low levels of LDL-cholesterol.

What about musculoskeletal conditions? Most medical and chiropractic physicians have been taught that osteoarthritis (OA) is a non-inflammatory condition caused by mechanical wear and tear. Recent research would suggest otherwise, and is consistent with most clinical presentations of osteoarthritis; that is, several joints hurt, but one may be most pronounced and perhaps associated with previous injury. We tend to forget that other joints also hurt, which takes our mind away from viewing research suggests that increasing levels of CRP can predict the emergence and severity of osteoarthritis.

If you decide to measure hsCRP in your office, the typical interpretation is as follows: less than 1mg/L is low risk for heart disease prediction/expression; between 1/3 mg/L reflects moderate risk; and between 3-10 mg/L suggests high risk. Greater than 10 suggests an acute phase response and, thus, a re-test in 3 weeks. In terms of diet, it appears that the following are associated with elevated CRP: syndrome X, low fiber diet, low magnesium intake, low omega-3 intake, trans-fat intake, low micronutrient intake, and reduced sleep.

The key to understanding elevated CRP is that it is a marker for dietary imbalances that create a pro-inflammatory state. Syndrome X is a significant metabolic driver of elevated CRP and numerous other pro-inflammatory mediators. The dietary approach to syndrome X focuses on foods with a low glycemic index and low glycemic load, which means vegetables, lean animal products, fruits, and nuts.

Patients need to understand that dietary changes are the most important component for reducing the expression of syndrome X. In addition, supplements to help reduce the pro-inflammatory state include a multivitamin, magnesium, fish oil, vitamin D, and botanicals such as ginger, turmeric, and garlic.

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