To our knowledge, the causes of cellular inflammation and the functional role of CRP have not been previously elucidated.

When elements are recognized as the foundation for cytokines and cellular signaling (aka epigenetics and DNA repair), nearly all aspects of nutritional science and the needed to establish cellular health will become a primary scientific discipline for biomedical activities.

With that said, the findings included in the attachment to this article are provided for discussion purposes for use as a foundation from which biomedical research can develop an understanding for these crucial universal cellular activities (inflammation, CRP and IL-1B activities); i.e., not merely for cardiac issues.

The following is an example of many studies that link CRP to other diseases that include but are not limited to cancer.
https://www.ncbi.nlm.nih.gov/pubmed/22035340

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https://www.aol.com/article/news/2017/08/27/a-new-way-to-fight-heart-disease-may-also-tackle-cancer/23187280/

Refer to the following for another version of the NBC News article.
https://medicalxpress.com/news/2017-08-drug-lowers-heart-curbing-inflammation.html

A new way to fight heart disease may also tackle cancer

NBC NEWS

MAGGIE FOX AND FELIX GUSSONE, MD

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Researchers say they've proven a long-held theory about heart disease: that lowering inflammation may be nearly as important as cutting cholesterol levels.

They showed that using a targeted drug to reduce inflammation cut the risk of heart attacks, strokes and other "events" in patients who had already suffered one heart attack – independent of any other treatment they got.

A bonus side-effect – the treatment also appeared to have reduced rates of lung cancer diagnosis and death.

The studies, being presented at a meeting in Barcelona this weekend, are just a first step and do not yet open a door to a new way of treating heart patients.

And they don't necessarily apply to everybody. But Dr. Paul Ridker of Brigham and Women's Hospital and Harvard Medical School, who led the research team, thinks the findings will lead to ways to help people most at risk of dying of heart disease and stroke.

"This plays beautifully into the whole idea of personalized medicine and trying to get the right drug to the right patient," Ridker said.

Novartis, which makes the drug, said it would ask the Food and Drug Administration for permission to market the drug as a way to prevent heart attacks and would start further tests on its effect in lung cancer.

Ridker's team tested 10,000 patients who had suffered one heart attack already and thus were at very high risk of having a second one. The patients all had high levels of high sensitivity C-reactive protein or CRP, a measure of inflammation in the body.

They were already taking a basket of medications for their heart disease, from cholesterol-lowering statins to blood pressure drugs.
On top of that, the team added a drug called canakinumab, a monoclonal antibody or magic bullet agent that targets a specific cause of inflammation called interleukin 1 beta.
Volunteers got either a placebo, or injections every three months of low, medium or high doses of canakinumab.

After three to four years, people who got the highest dose of the drug were the least likely to have had another heart attack, stroke or to have died of heart disease.

Those who got the two highest doses of canakinumab had a 15 percent lower chance of having a heart attack, stroke or other major cardiovascular event, the team found. Patients were also less likely to need a heart bypass or angioplasty to clear out clogged arteries.

"For the first time, we've been able to definitively show that lowering inflammation independent of cholesterol reduces cardiovascular risk," Ridker said.

Dr. Steven Nissen, chairman of the Department of Cardiovascular Medicine at the Cleveland Clinic, who was not involved in the study, said the results were impressive. "It shows us that people with high levels of inflammation - if you target the inflammation - you can reduce the risk of heart attack stroke and death," Nissen said.

The results are being presented at the European Society of Cardiology meeting in Barcelona, and also published in the New England Journal of Medicine and the Lancet medical journal. It's been long known that both inflammation and cholesterol buildup are involved in heart and artery disease.

Inflammation is part of the body's immune process, and the patients in the trial were more likely to suffer serious infections, including pneumonia. The same thing happens to people taking immune-suppressing drugs to fight rheumatoid arthritis.

"Physicians would have to be cautious," Ridker said.

But the researchers found some other side-effects. People taking the higher doses of canakinumab had lower rates of cancer, especially lung cancer, as well as lower rates of arthritis and gout.

This makes sense to Ridker.

"If you smoke a pack of cigarettes, you chronically inflame the lung. If you are a long-haul truck driver breathing in diesel, you are chronically inflaming the lung," he said. Inflammation can drive cancer as well as heart disease, he said.

"These are fascinating, human findings that open a potential new class of therapies for cancer," said Dr. Laurie Glimcher, president and CEO of the Dana-Farber Cancer Institute.

Ridker does not believe the drug prevents cancer. He thinks inflammation may fuel the growth of some tumors.

"The tumors were obviously already there. They were just small and undiagnosed," he said. The findings will not immediately mean new treatments for heart disease patients. For one thing, like any medical finding, they'll have to be replicated by other researchers. Ridker's testing another drug, methotrexate, that's also used to treat rheumatoid arthritis.

While canakinumab has already been approved by the FDA, it is a so-called orphan drug used to treat a very rare genetic condition. Sold under the brand name Ilaris, it costs about $200,000 a year.

"We look forward to submitting the ... data to regulatory authorities for approval in cardiovascular and initiating additional phase III studies in lung cancer," said Vas Narasimhan, who heads drug development for Novartis.

Ridker says he is pressing Novartis to try something different, perhaps offering the first dose of the drug free. People whose CRP levels fell more after their first dose also tended to be those who had lower rates of heart attacks and strokes years later.

"It might be worth taking one dose and see if you respond. If you don't, well, there is no reason to be on the drug," he said.

"This is the way to really focus these treatments on the patients on whom it really works. I think that's just good medicine."

In the end, Ridker believes, some extreme heart disease patients will be helped more by the newest cholesterol-lowering drugs, called PCSK9 inhibitors, while others may be better helped by targeted anti-inflammatory drugs.

"Half of heart attacks occur in people who do not have high cholesterol," he said. "For the first time, we've been able to definitively show that lowering inflammation independent of cholesterol reduces cardiovascular risk.

Nissen agrees.

"I think it's a game changer. The only good therapies we've had so far were statins. But now it seems like we have something new in the future," he said.

"It opens up pathways to new research and new treatments in the future. There are many other anti-inflammatory activities going on in our body, not just the one that's tackled by canakinumab. There will be so many more studies now to see if other therapies that tackle other pathways will also reduce the risk."

And the findings may offer some common-sense advice to everyone about lowering inflammation, Ridker said.

"There's a lot you can do about it right now," he said.

"If your high sensitivity C-reactive protein is elevated, you are a high-risk patient. This is overwhelming evidence that you should go to the gym, throw out the cigarettes, eat a healthier diet," he said.

"Because all three of those well-known interventions lower your inflammatory burden."

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MCFIP – Our explicit and replicable epigenetic modeling based on the fundamentals of physical chemistry and quantum mechanics identifies the following relative to IL-1B:

- It is a cell surface signaling byproduct of iron - aluminum based IL-1 with the amino acid cascade of threonine – serine – cysteine.
- Formation of cell surface signaling takes place through the following mechanism:

http://www.mcfip.net/upload/Cell%20Surface%20Signaling%20Molecule%20Formation%207-2017.pdf

- The signaling molecule is also linked to nearly all forms of leukemia. http://www.mcfip.net/upload/Leukemia%20Biomarkers%20-%20o.pdf

Epigenetic modeling of CRP identifies the following:

- It is IL-6 that is an electrolyte with calcium - magnesium and chloride as constituents.
- Along with IL-2 and IL-9 it constitutes the three electrolytes and they all exist as mitochondria for cardiomyocytes.
- The three cell surface counterparts to these cytokines are IL-32, IL-33 and IL-3 respectively. They are formed when a gasotransmitter (e.g. nitric oxide in the case of Interleukins) is substituted for chloride and the gasotransmitter is subjected to catabolic activity using the following mechanism: http://www.mcfip.net/upload/Cell%20Surface%20Signaling%20Molecule%20Formation%207-2017.pdf
- Collectively, these epigenetic signaling molecules constitute the electrolytic activity known as TITIN.

Biomedical research has been unable to elucidate the specific mechanism that causes cellular inflammation. We assert that it is endocytosis and the volume of activity of the movement of signaling from the surface of the cell into the cytoplasm as a means of supporting cellular health is the source of inflammation. Refer to the following to understand endocytosis.
http://www.mcfip.net/upload/Endocytosis%20Modeling%204-30-17.pdf

In terms of cardiac activity, with near certainty, CRP (IL-6) is signaling of mitochondrial activity along with IL-2 that is a pair for symbiotic activity supported by IL-9. The elements of the three cytokines are calcium - magnesium, sodium - potassium and calcium - zinc

respectively with chloride being the common factor. IL-9 functions as a "modulator" to maintain homeostasis between IL-6 (calcium - magnesium) and IL-2 (sodium - potassium). Furthermore, we assert the byproducts of catabolic activity of the gasotransmitter iteration, with near certainty, provide the same cell surface role for the support of bioelectric energy required to support cellular health.

Note: All facets of the epigenetic activities outlined thus far can be independently verified as being factual. However, anecdotal evidence supports the likelihood that byproducts of the gasotransmitter driven cytokines (e.g. MYC) interface and resulting imbalances can be the primary cause of chronic diseases such as A-Fib, cancers, etc.

Obviously, because this assertion is hypothetical, it must be subjected to through review by an integrated team of researchers that utilize the tenets of physical science based epigenetic modeling.

As part of the research we recommend, one of the byproducts of IL-3 with the amino acid cascade of threonine – serine – cysteine must be pursued. It is the family of MYC that regulates cellular alignment and can be verified as the primary epigenetic marker of many cancers.

For use as part of discussions, the role of MYC is depicted below: