



California

CHAPTER

**California Chapter of the
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C-Reactive Protein for Cardiac Risk Stratification and for Determining the Use of Statins

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C-Reactive Protein, (CRP) is primarily produced in the liver under the control by the pro-inflammatory cytokine, IL-6. It is established that atherosclerosis is an inflammatory disease, and the magnitude of inflammation greatly increases the risk of heart attack and stroke. As CRP has been shown to be a readily available and reliable marker of inflammation, it may be used to identify people who are at risk for heart attack and stroke, and in addition, to serve as a guide to further enhance cardiovascular risk reduction for those who are currently under medical treatment for established cardiovascular disease.

Multiple studies have established that ostensibly “healthy” individuals with a high CRP were at significantly elevated risk of coronary heart disease (CHD). Moreover, this elevated risk was independent of traditional cardiovascular risk factors including age, smoking, LDL cholesterol, blood pressure and diabetes. This was most evident in the Women’s Health Study. In addition, CRP was found to be a stronger predictor of 8-yr cardiovascular risk than was LDL-C levels. Moreover, Its prognostic value was found to be greatest in the patients who had been previously considered to be at only “intermediate” risk by Framingham risk scoring.

Many factors can raise CRP and may contribute to a patient’s inflammatory burden. They include hypertension, obesity, cigarette smoking, diabetes and metabolic syndrome, as well as sleep apnea. The recently published risk of hormonal replacement therapy is in part due to its ability to increase CRP levels. CRP has been found to be more than just a marker of inflammation.

Although initially considered as an innocent bystander in the atherosclerotic process, CRP may have direct proinflammatory effects and contribute to the initiation and progression of atherosclerotic lesions. Mechanisms include activation of circulating monocytes(white blood cells) , mediation of endothelial (blood vessel lining) dysfunction, and the induction of a prothrombotic state (clot formation).

CRP has also been shown to be a marker of residual risk for those already on statins. In the “PROVE-IT” trial patients with a very low achieved LDL of < 70mg/dl but with a CRP >2 md/dl, had the equivalent risk of patients with an LDL > 70mg/dl but a CRP < 2mg/dl. In the “REVERSAL” trial, plaque volume within the coronary arteries was actually measured. The patients that demonstrated the greatest reduction in plaque had not only the lowest LDL levels, but the lowest CRP levels as well.

Finally, in the recently published Jupiter study, patients who were considered at cardiovascular risk with relatively normal LDL-C values (not on statins), had a 44% risk reduction when a statin was added- not only lowering LDL-C further, but resulting in a marked reduction in CRP as well.

Our mission: To be an advocate for accessible, highest quality, cost effective cardiovascular care for the people of California and to actively support the cardiac care team in their efforts to achieve these goals.

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Summary:

The current medical literature lends strong support to atherosclerosis as an inflammatory disease. At the same time, the measurement of CRP has been shown to be a reliable marker of inflammation, and carries predictive value in determining which patients may be at higher risk for future cardiovascular events. These same patients may have heretofore gone unrecognized and untreated until much later in life. In addition, for those patients already on a statin, the CRP determination can act as a guide in adjusting therapy. As the cost of health care rises and the available funds diminish, it is time to realize that the prevention of the disease, rather than its treatment once fulminant, must be the standard of care.

Rob Greenfield, MD FACC FAHA FNLA

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