humans using the methods of genetics have not supported the notion that CRP contributes to the mechanism of atherosclerosis (126,127).

Inflammation is part of the mechanism of atherosclerosis, as follows. Low density lipoproteins (LDS) are taken up by monocytes. The monocytes bind to endothelial cells of the coronary artery. The monocytes differentiate into macrophages, which accumulate in the artery, resulting in the "fatty streak," which later becomes an atherosclerotic lesion (128). By definition, the pathological accumulation of macrophages in the walls of blood vessels is inflammation.

C-reactive protein is likely to be part of the mechanism for acute damage occurring during a heart attack (129). In this mechanism, CRP contributes to complement activation. During this acute timeframe, CRP levels in the bloodstream can reach 150 to 200 mg/L (130,131). The highest levels of plasma CRP occur at 2 to 4 days after myocardial infarction (132). These high values are much greater than CRP levels occurring in chronic timeframes. Ørn et al. (133) provide time-course data on plasma CRP in heart attack patients, and demonstrate that a peak (mean of 35 mg/L) occurs at 2 days. CRP is not the only biomarker used in the context of heart attacks. In a review, Chan and Ng (134) disclose details on a number of other useful biomarkers for diagnosing acute myocardial infarctions.

The above material is summarized by the following bullet points:

- Inflammation is an established, proven part of the mechanism of atherosclerosis.
- Long-term elevation of CRP is associated with increased risk for atherosclerosis.
- It is not certain that CRP has a causal role in the etiology, over the long term, of atherosclerosis.
- CRP has a causal role in promoting damage during the acute timeframe of a heart attack.

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