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C-Reactive Protein and Its Role In Periodontitis

[1] S. C. Gupta
[2] Vikas Jindal
[3] Rambhika Thakur

[1] Professor, Department Of Periodontology, Himachal Dental College, Sunder Nagar, H.P.

[2] Professor, Department Of Periodontology, Himachal Dental College, Sunder Nagar, H.P.

[3] Postgraduate Student
Department Of Periodontology
Himachal Dental College, Sunder Nagar, H.P.

Address For Correspondence:

Dr. S. C. Gupta
Professor, Department Of Periodontology,
Himachal Dental College, Sunder Nagar, H.P.

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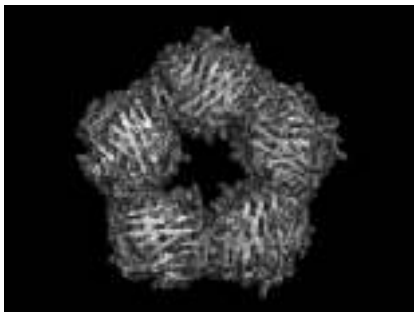
Abstract
C-Reactive Protein (CRP) was originally discovered by Tillett and Francis in 1930 as a substance in the serum of patients with acute inflammation that reacted with the C- polysaccharide of pneumococcus. Initially, it was thought that CRP might be a pathogenic secretion as it was elevated in people with a variety of illnesses including cancer. However, discovery of hepatic synthesis demonstrated that it is a native protein. C-reactive protein is a protein found in the blood and its levels rise in response to inflammation. Its physiological role is to bind to phosohocholine expressed on the surface of dead or dying cells (and some types of bacteria) in order to activate the complement system via the CIQ complex.

Key Words
CRP, Chronic Periodontitis, Adipocytes

CRP is synthesized by the liver in response to factors released by fat cells (adipocytes). It is a member of the pentraxin family of proteins.



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Periodontitis is a destructive inflammatory disease of the supporting tissues of the teeth. The host responds to the periodontal infections with an array of events involving both innate and acquired immunity. Although periodontitis is chronic in nature, acute-phase elements are also part of the innate immunity. The acute phase reactants have also pro-inflammatory properties. They activate compliment factors, neutralize invasive pathogens and stimulate repair and regeneration of a variety of tissues.

There is evidence that periodontitis as well as coronary artery diseases (CAD) are linked by inflammatory factors including CRP. It is an acute-phase reactant that is produced in response to diverse inflammatory stimuli including heat, trauma, infection, and hypoxia. CRP levels provide useful information for the diagnosis, monitoring and therapy of the inflammatory process and associated disease. CRP levels rise in the serum or plasma within 24-48 hours following acute tissue damage, reaching a peak during the acute stage and then decrease with the resolution of inflammation or trauma.

The acute phase reactants receiving most attention are C-reactive protein (CRP), plasminogen activator 1(PAS-1), and fibrinogen. CRP plays a key role in innate immune response and is easily measured due to its long plasma half life of 12-18 hours. In healthy individuals, CRP levels are found in trace amounts with levels <0.3 mg/l. Serum levels of CRP could exceed 100mg/l in the presence of systemic infection which provides a useful marker for tracking the course of infection.

It has been postulated that increased CRP level as a result of periodontal inflammation could provide an explanation of the reported relationship between periodontitis and coronary heart disease. In recent years it has become evident that CRP is a sensitive circulatory marker of inflammation. Atherosclerosis, though it is a multifactorial disease, inflammation also plays an important role in its pathogenesis. Thus increased CRP level as a result of periodontal inflammation could provide an explanation of the reported relationship between periodontitis and coronary heart disease.

There are now several reports indicating that bacteraemia may also occur frequently in periodontitis patients. The host responds to short lived bacteraemia and systemic cytokine dumping from untreated chronic periodontitis lesions in a similar manner as would be the case with other chronic infections or inflammatory processes. For example, elevated levels of interleukin-6 (IL-6), known to induce hepatocytes to produce CRP and other acute phase proteins and pro-coagulant mediators have also been reported in periodontitis patients. Thus, it is not surprising to note that changes in cellular and molecular components of peripheral blood have been observed in periodontitis.

CRP during its role in inflammatory process binds to the surface of pathogens and opsonizes them for uptake of phagocytes. CRP can also activate the classic complement cascade by binding to 'q' factor of complement factor 1 (C1q). Another pro-inflammatory function of CRP includes the induction of cytokines and tissue factor in monocytes. Therefore, it acts as anti-inflammatory by decreasing neutrophils migration to the site of inflammation, preventing adhesion of neutrophils to endothelial cells, and affecting clearance of nuclear antigens released from apoptic or necrotic cells. Apart from infections, inflammation and trauma, factors associated with increased levels of CRP include obesity, cigarette smoking, hormone use, metabolic syndrome, and cardiovascular disease. Moderate alcohol consumption, increased physical activity and medication use are associated with reduced CRP levels.

It needs to be stressed that CRP is a non specific

marker of the acute phase response. That is, many potential stimuli including unknown chronic infections and or inflammatory conditions, smoking, obesity and trauma may also account for mild increase in CRP.

Recent literature identifies adiposity as a key factor in low grade chronic inflammation. Higher body mass index is associated with elevated CRP concentration in adult men and women. Overweight women may thus be more likely to have chronic inflammation and elevated CRP levels.

Various studies of acute phase reactants in periodontitis have focused on patients with chronic periodontitis. It has been demonstrated that CRP levels are higher in periodontitis patients than in periodontally healthy subjects and that serum CRP levels are still higher in patients with more severe periodontitis. In addition recent trials have indicated that treatment of periodontal infections, whether by intensive mechanical therapy, drug therapy, or extraction can significantly lower serum levels of CRP.

REFERENCES

1. Thompson D, Pepys MB, Word SP (1999). The physiological structure of human C- reactive proteins and its complex with phosphocholine. Structure 7(2): 169-77.
2. Pepys MB, Hirschfield GM (2003). C-reactive protein: a critical update. J clin Invest 111 (12): 1805-12.
3. Lau DC, Dhillon B, Yan H, Szmilko PE, Verma S (2005). Am J Physiol Heart Circ Physiol: 288(5): H2031-41.
4. Tillot WS. Francis Jr T (1930). Serological reaction in pneumonics with non-protein somatic fraction of pneumococcus. J Exp Med 52: 561-85.
5. Paraskevas S, Huizinga JD, Loos BG. (2008). A systematic review and meta-analyses on C-reactive protein in relation to periodontitis. J Clin Periodontol 35:277-290.
6. Trang N Salzberg, Benjamin T. Overstreet, Jeffrey D. Rogers, Joseph V. Califano, Al M. Best, and Harvey A. Schenkein (2006). C - reactive protein levels in patient with aggressive periodontitis. J Periodontol 77(6): 933-39.
7. Linden GJ, Mc Clean K, Young I, Evans A, Kee F. (2008). Persistently raised C- reactive protein levels are associated with advanced periodontal disease. J Clin Periodontol 35:741-747.
8. Pittphat W, Savetsilp W, Wara- Aawapati N. (2008). C- reactive protein associated with periodontitis in a Thai population. J Clin Periodontol 35:120-125.