Conclusions: The results of the GENERATION study suggest that high plasma levels of CRP are not related to the rate of ISR after successful CS. More studies are needed to elucidate this issue.

<table>
<thead>
<tr>
<th>CRP quartiles</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median mg/dl</td>
<td>0.55</td>
<td>0.64</td>
<td>0.84</td>
<td>1.01</td>
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</tr>
</tbody>
</table>

**HIV Immunodeficiency Virus Infection and High C-Reactive Protein Correlates With Increased In-Stent Restenosis Rate**

**Methods:** 30 patients with HIV who had PCI with stents from 1997 to 2002 were studied.

**Objective:** We evaluated the outcome of patients with Human Immunodeficiency Virus (HIV) infection who had percutaneous coronary interventions (PCI) with stent implantation. Background: HIV and anti-retroviral therapy may propagate intimal proliferation and inflammation. These cascades have been postulated to accelerate in-stent restenosis. The atherogenic effects of various inflammatory cytokines that are up-regulated by HIV may contribute to accelerate coronary artery disease (CAD). C-reactive protein (0.0 - 5.0 mg/L) appears to correlate with increased atherogenesis. It is hypothesized that increased levels of C-reactive protein (CRP) correlate with increased in-stent restenosis.

**Results:** -174G/C genotypes presented as: 31% GG, 46% GC and 23% CC. After 6 months, control coronarography was performed to quantify restenosis.

**Conclusion:** Carriers of the apo E4 allele have higher restenosis rates after coronary stenting as compared to apo E2 carriers.

**IL-6 174G/C Polymorphism Influences Restenosis After Coronary Stent Implantation by Increased Inflammation and Platelet Activation**

**Background:** The G allele of interleukin-6 (IL-6) -174G/C polymorphism is associated with an enhanced plasma level of IL-6 after bypass surgery. IL-6's influence on the inflammation and on the mean platelet volume (MPV), a parameter for activated platelets, after coronary stent implantation (ST) is unknown. Furthermore, there is no data about the influence of IL-6 -174G/C on restenosis after ST.

**Methods:** Between 9/01 and 3/02 we enrolled 65 patients (58 men, mean age 63±9 years) before ST. -174G/C genotype and levels of IL-6. C-reactive protein (CRP) and MPV were determined before ST, 3h, 6h, 12h, 24h, 48h, and 7 days thereafter. After 6 months, coronary restenosis (>50%) was angiographically documented in 45% of GG. In 30% of GC and in 13% of CC genotype (comparison GG vs. CC: p<0.05).

**Conclusions:** 1. The G allele of -174G/C is associated with significantly higher levels of IL-6 resulting in significantly higher increase of CRP and MPV after stenting. 2. The risk for restenosis in homozygous carriers of the G allele is about three times higher than in patients without G allele, which might be explained by the enhanced platelet activation and inflammatory response after stenting.