does not predict the ability of an assay to detect *Chlamydia pneumoniae* in tissues.

We agree with Valassina et al. [1] that the presence of *Chlamydia pneumoniae* DNA alone in arterial specimens is insufficient proof of the presence of viable organisms in vascular tissue; however, cultural isolation of the bacterium from atherosclerotic plaques has been reported repeatedly [5, 6]. Furthermore, culture-derived data are in accordance with the results of our study on *Chlamydia pneumoniae* RNA in carotid plaques. Regarding the suggestion that inflammatory mechanisms such as molecular mimicry or induction of metabolic mechanisms are responsible for atheroma formation, rather than the presence of viable bacteria, we believe this to be partially true. However, the results of studies in animal models indicate that the presence of viable bacteria in vascular tissues is an essential element in the association between *Chlamydia pneumoniae* and development of atherosclerotic lesions [7].

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**References**


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**Table 1. Prevalence of NSI/SI primary human immunodeficiency virus type 1 isolates versus CCR2 allelic variants.**

<table>
<thead>
<tr>
<th>CCR2&lt;sup&gt;64I&lt;/sup&gt;</th>
<th>NSI</th>
<th>SI</th>
<th>NSI&lt;sup&gt;64I&lt;/sup&gt; + SI&lt;sup&gt;64I&lt;/sup&gt;</th>
<th>P&lt;sup&gt;*&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>SFMHS</td>
<td>98</td>
<td>42</td>
<td>24</td>
<td>0.033</td>
</tr>
<tr>
<td>Milan</td>
<td>23</td>
<td>6</td>
<td>6</td>
<td>0.137</td>
</tr>
<tr>
<td>Both</td>
<td>121</td>
<td>48</td>
<td>30</td>
<td>0.014</td>
</tr>
</tbody>
</table>

*NOTE.* Numerical values are numbers of individuals. NSI, non-syncytium inducing; SFMHS, San Francisco Men’s Health Study; SI, syncytium inducing. *P* Fisher’s exact P value.
significant differences in viremia levels were found among persons carrying different CCR2 alleles (not shown). In a previous study, infected persons carrying the CCR2-64I allele had a higher frequency of SI viruses, and the protective effect of this allele on disease progression was restricted to persons infected by NSI viruses [8]. In partial disagreement with these observations, the lack of protection from disease progression conferred by CCR2-64I was confirmed by separate Cox analysis of subjects infected with either NSI or SI viruses (RH, 1.105; 95% CI, 0.612–1.995; \(P = .74\), and RH, 0.889; 95% CI, 0.508–1.555; \(P = .679\), respectively).

We speculate that factors linked to CCR2-64I might counterbalance the negative effect of SI viruses on disease progression. Heterologous desensitization of both CCR5 and CXCR4 by MCP-1 ligation and slightly lower CXCR4 expression on primary T cells from persons carrying CCR2-64I might play a role in HIV pathogenesis [6]. Finally, the observation that CCR2-64I is related to an increased frequency of SI HIV strains suggests a potential role of CCR2 ligands (i.e., MCPs) in the dynamics of HIV quasi species, in terms of chemokine coreceptor utilization. This hypothesis is supported by the observation that MCP-1 could up-regulate HIV replication [9] and favor the emergence of SI HIV strains in peripheral blood mononuclear cell cultures established from some HIV-infected persons [10].

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References


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