**Letter to the Editor**

**Association between Brucella melitensis DNA and Brucella sp. Antibodies**

*Brucella* sp. antibodies, despite falling to low levels, can remain measurable after recovery from acute brucellosis (1). Recently, several studies have shown the persistence of *Brucella* sp. DNA in both chronic brucellosis patients and asymptomatic subjects with a history of brucellosis (2–4, 6). However, to our knowledge, the association between serum antibodies and *Brucella* sp. DNA has not been investigated.

We screened a cohort of 38 subjects with a well-documented history of brucellosis for the presence of *Brucella melitensis* DNA and *Brucella* antibodies. For that purpose, we tested both a quantitative real-time PCR (qPCR) assay (2) and an immunocapture-agglutination test (*Brucellacapt*; Vircell SL, Granada, Spain) that was performed as specified by the manufacturer. The *Brucellacapt* test has been described as offering results comparable to those of the Coombs anti-*Brucella* test, the most often used technique for the diagnosis of chronic brucellosis (5).

Twenty-seven (71%) subjects were men, and 11 (29%) were women. The mean age was 49 ± 14 years (range, 26 to 83 years). The diagnosis of acute brucellosis was made between 3 and 33 years previously, according to one or both of the following criteria: isolation of *Brucella* spp. from blood or any sample of body fluid or tissue and the presence of a compatible clinical picture together with the demonstration of specific antibodies at significant titers (Wright test titer of ≥1:160 or Coombs anti-*Brucella* test titer of ≥1:320) or seroconversion.

According to their clinical course after the initial episode, subjects were divided into three groups. Group A consisted of 10 (26%) focal disease subjects. Group B comprised 8 (21%) nonfocal disease subjects complaining of nonspecific symptoms, such as fatigue, malaise, arthralgia, and/or myalgia. The remaining 20 (53%) subjects were asymptomatic (group C). Chronic brucellosis patients included all patients diagnosed with focal disease and those whose symptoms had persisted for more than 1 year after the initial episode. Results are expressed as means ± standard deviations. *P* values less than 0.05 were considered statistically significant.

We found an association between being *B. melitensis* DNA positive and being antibody positive. Among the 22 subjects with detectable *B. melitensis* DNA, 19 (86%) subjects had *Brucella* antibodies, while among the 16 subjects without *B. melitensis* DNA, *Brucella* antibodies were detected in 7 (44%) (*P* = 0.005; chi-square test). In the case of the asymptomatic subject group, the DNA-antibody concordance was not statistically significant (*P* = 0.264; two-tailed Fisher’s exact test). The distribution of DNA-antibody results by group is shown in Table 1.

The chronic brucellosis patients harboring *B. melitensis* DNA are more likely to show a seropositive sample than the remaining subjects. These findings suggest that after the initial infection, either the viable *Brucella* or its antigenic and structural components persist in the host and may have diagnostic and pathogenic implications.

Financial support was provided by the “Fondo de Investigación Sanitaria” grant PI051885 from Spanish Ministry of Health of Spain and by the Consejería de Sanidad grants 06028-00 and PI-2006/43 from Fundación para la Investigación de Castilla-La Mancha (FISCAM) of Spain, Balagüe Center S.A grant, and Consejería de Sanidad grant MOV2007.1105 from FISCAM to María Jesús Castaño.

We have no conflicts of interest.

**REFERENCES**


### TABLE 1. Distribution of the qPCR and *Brucellacapt* results from 18 patients with chronic brucellosis and 20 asymptomatic subjects

<table>
<thead>
<tr>
<th>No. of patients/subjects with qPCR blood/serum result*</th>
<th>Focal disease patients (n = 10)</th>
<th>Nonfocal disease patients (n = 8)</th>
<th>Asymptomatic subjects (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+/+</td>
<td>+/−</td>
<td>+/−</td>
</tr>
<tr>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>40</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>80</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>160</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>320</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>≥640</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

* Reciprocal titers are shown.

**P** positive; **−** negative.

M. Jesús Cañasto Aroca*
Elena Navarro García
Unidad de Investigación Laboratorio de Biología Molecular

Javier Solera Santos
Servicio de Medicina Interna Hospital General Universitario C/Hermanos Falcó s/n 02006 Albacete, Spain

*Phone: 34 967 59 70 83
Fax: 34 967 24 39 52
E-mail: castanoaoroca@yahoo.es

Published ahead of print on 16 March 2011.