for the life of the host. In the latent infection, parasite-specific T-lymphocytes release high levels of gamma interferon, which is required to activate and synergize macrophages for toxoplasmacidal activity. Therefore, delayed immune reconstitution after transplantation puts patients at risk of reactivation of latent infection. The precise kinetics of immune reconstitution and rendered the patient susceptible to disseminated toxoplasmosis. Although more studies need to be done to improve the understanding of immunologic impairment responsible for toxoplasmosis reactivation, prophylactic therapy for toxoplasmosis could be beneficial for seropositive patients especially in cases of CD34+ positive selected transplantation with TBI regimen.

Minoru Nakane,* Kazuteru Ohashi,* Junya Tominaga,* Hideki Akiyama,* Kiyoshi Hiruma,* Hisashi Sakamaki*

*Bone Marrow Transplantation Team; Pathology Division, Tokyo Metropolitan Komagome Hospital, Tokyo, Japan

Key words
CD34 selection; toxoplasmosis; adult T-cell leukemia/lymphoma.

Acknowledgments
We thank Dr. A. Makioka (Jikei University, Tokyo, Japan) for performing the dye test.

Correspondence
Dr. Kazuteru Ohashi, M. D., Hematology Division, Tokyo Metropolitan Komagome Hospital, 3-18-22 Honkomagome, Bunkyo-ku, Tokyo, 113-8677, Japan.
Phone: international +81-3-38232101 – Fax: international +81-3-38241552 – E-mail: k.ohashi-k@komagome-hospital.bunkyo.tokyo.jp

Table 1. Evaluation of antitoxoplasma antibodies.

<table>
<thead>
<tr>
<th>Antibodies</th>
<th>ELISA</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>pre-transplantation</td>
<td>IgG</td>
<td>189</td>
<td>negative</td>
</tr>
<tr>
<td></td>
<td>IgM</td>
<td>64 X</td>
<td></td>
</tr>
<tr>
<td>day 143</td>
<td>IgG</td>
<td>44</td>
<td>negative</td>
</tr>
<tr>
<td></td>
<td>IgM</td>
<td>16 X</td>
<td></td>
</tr>
</tbody>
</table>

IgG and IgM antibodies were quantified with use of an ELISA. titers are expressed in units per milliliter (IU/mL). A patient is considered seropositive if titers are over 10 IU/mL. Pre-transplant serologic status positive for IgG but negative for IgM indicated past toxoplasma infection. On day 143, IgG titers decreased and IgM were not still detectable, in spite of definite proof of infection of toxoplasma in multiple tissues. These findings may be a reflection of impaired cellular and humoral immunity after transplantation, and may also indicate serologic status is not an appropriate tool for early diagnosis.

The Feldman dye test is a more sensitive and specific neutralization test in which the organisms are lysed in the presence of IgG antibody and complement. The patient is considered to have a chronic infection if titers are over 16 X. In case of acute infection, titers are normally over 1,024 X.

Table 2. Evaluation of antitoxoplasma antibodies.

<table>
<thead>
<tr>
<th>Day 143</th>
<th>IgG titers</th>
<th>IgM titers</th>
</tr>
</thead>
<tbody>
<tr>
<td>pre-transplantation</td>
<td>44</td>
<td>16 X</td>
</tr>
<tr>
<td>day 143</td>
<td>44</td>
<td>16 X</td>
</tr>
</tbody>
</table>

References


Frequency of Gilbert’s syndrome associated with UGTA1 (TA) polymorphism in Southern Italy

We screened 685 subjects from Southern Italy for a promoter polymorphism of the UDP-glucuronosyltransferase (UGTA1) gene tightly linked to Gilbert’s syndrome (GS), consisting in the insertion of a TA repeat in the TATA box. The frequency of the polymorphism was 0.387 which is similar to the frequencies reported for other investigated populations of different ethnic backgrounds and is consistent with the antiquity of this polymorphism.

Sir,
Gilbert’s syndrome (GS) is an inherited form of mild unconjugated hyperbilirubinemias, characterized by decreased bilirubin UDP-glucuronosyltransferase activity (UGTA1). Serum bilirubin levels vary according to time, intercurrent illness or fasting.

The recent identification of the UGTA1 locus, which encodes for a family of UGTA1 isoforms, has provided tools for molecular studies and for the correct definition of inheritance pattern. Although heterozygous missense mutations have been identified in patients with GS, some studies focused on the relationship between GS and inherited red cell defects (spherocytosis, G6PD deficiency, thalassemia) and neonatal jaundice.

The aim of our study was to establish the gene frequency of the TA repeat promoter polymorphism in a
Southern Italy population.

After being given informed consent we examined 685 subjects (300 males and 385 females) from Southern Italy. By means of polymerase chain reaction and gel electrophoresis the subjects were genotyped for the TA promoter polymorphism. The prevalence of homozygosity for the (TA)7 allele was 15%.

Table 1 reports the allele frequencies found in other studies and the present study.

In Caucasian people, the estimated gene frequency of (TA)7 is 0.387. The frequency of the (TA)7 promoter is lowest in Asian (0.16) and highest in African populations (0.426), where two other variants have been identified, (TA)5 and (TA)8, with relative frequencies of 0.035 and 0.069, respectively. Our data are in agreement with data reported by Beutler et al. concerning a Caucasian population. The allele frequency in an African population appears the same as that of non-Africans as well as the Sardinian population. These data are consistent with the oldness of the polymorphism, which appears to be homogeneously distributed in the world.

The result from the Asian population appears discordant; this could be due to the relative low number of subjects and to the relative heterogeneity of the examined population. It is noteworthy that a missense Gly71Arg mutation, associated with GS, is present in 19% of the population of Asian origin. Preliminary analysis on Caucasian chromosomes have demonstrated that this polymorphism is very rare (Iolascon et al., unpublished data).

(TA)7 polymorphism is strongly associated with higher bilirubin levels.

The very high prevalence of this polymorphism could be relevant in Mediterranean countries where there is a high incidence of inherited hemolytic diseases. The co-inheritance of GS with a hemolytic red cell defect could account for the heterogeneity of clinical findings (hyperbilirubinemia, gallstones) in affected kindred.

Achille Iolascon,* Silverio Perrotta,* Brigida Coppola,* Ruggiero Carbone,* Emanuele Miraglia del Giudice*

*Dipartimento di Biomedicina dell’Età Evolutiva, Università di Bari; *Dipartimento di Pediatría, Seconda Università di Napoli, Italy

Key words
Gilbert’s syndrome, hyperbilirubinemia, UGTA1, TA repeat.

Correspondence
A. Iolascon, M. D., Dipartimento di Biomedicina dell’Età Evolutiva, p.zza G. Cesare, 11, 70124 Bari, Italy. Phone: international +39-080-5478923 - Fax: international +39-080-5592290 - E-mail: a.iolascon@bioetaev.uniba.it

Table 1. UGTA1 allele frequencies.

<table>
<thead>
<tr>
<th>Ref.</th>
<th>(TA)6</th>
<th>(TA)7</th>
<th>n° of subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iolascon</td>
<td>43</td>
<td>57</td>
<td>685 (Italy)</td>
</tr>
<tr>
<td>Bancroft(9)</td>
<td>37</td>
<td>63</td>
<td>151 (USA)</td>
</tr>
<tr>
<td>Kaplan (5)</td>
<td>37</td>
<td>63</td>
<td>240 (Sephardic J.)</td>
</tr>
<tr>
<td>Beutler (7)</td>
<td>38</td>
<td>62</td>
<td>71 (Europe)</td>
</tr>
<tr>
<td>Beutler (7)</td>
<td>42</td>
<td>47</td>
<td>101 (Africa)</td>
</tr>
<tr>
<td>Beutler (7)</td>
<td>16</td>
<td>84</td>
<td>47 (Asia)</td>
</tr>
<tr>
<td>Akaba (8)</td>
<td>7</td>
<td>93</td>
<td>159 (Japan)</td>
</tr>
<tr>
<td>Galanello (4)</td>
<td>40</td>
<td>60</td>
<td>70 (Sardinia)</td>
</tr>
</tbody>
</table>

References