Significant Decrease of Titres of Circulating IgG after Oral Intake of a Preparation of Enterococcus faecalis in a Group of Ten Healthy Volunteers

The positive influence of orally ingested, live microorganisms, or probiotics [1] on the health status of the host organism has been recognized since 1907 [2] and is still a controversial subject. Beneficial properties, such as inhibition of potentially pathogenic microorganisms [3] and mutagen inactivation [4] are ascribed especially to Lactobacillus spp. Besides these gut microflora-associated effects, probiotics can exert a modulation of the immune system of the host [5]. The oral ingestion of an antigen may produce synthesis of IgA locally and/or at different secretory sites [6] or it may induce oral tolerance [7].

Despite the fact that in vertebrates other than humans – e.g. pigs and ruminants – the use of Lactobacillus spp. and Enterococcus spp. as probiotics has become generally adopted [8], little knowledge concerning the use of Enterococcus spp. in humans is available. The influence of Enterococcus species on the immune system of the host is of particular interest since Rusch et al. [9] observed – in humans – a significant decrease in infection risk associated with the oral intake of viable enterococci.

In this letter the effect of daily, oral intake of 10^7 viable cells of Enterococcus faecalis (Symbioflor 1®, SymbioPharm, Herborn-Dill, Germany) during 3 weeks on the humoral IgG status of the host is presented.

Ten healthy volunteers each donated two serum samples (A and B) with a 5-week interval. Immediately after this period each volunteer ingested 10^7 viable cells of E. faecalis daily, during 3 weeks. After this period a third serum sample (C) was taken from each volunteer. Finally, after a follow-up period of 3 weeks each volunteer delivered the fourth serum sample (D). In each individual serum sample the titre of circulating IgG against E. faecalis was assessed by means of a quantitative immunofluorescence method [10] using FITC-labeled goat-antihuman (Fab2) IgG as a conjugate.

The results of this study are summarized in Table 1. After the normality of distribution of the titres of circulating IgG against E. faecalis in pools A, B, C and D had been confirmed by means of the test for normality of Lilliefors [11], it was observed that the mean titre of pool B (1.54) falls within the 95% confidence interval of the mean titre of pool A (1.82) and vice versa. Because the titres from pool A and pool B did not differ significantly, they were averaged. The 95% confidence interval which results from this operation (i.e. 1.26 to 2.10) gives an indication of the height and the variability of the titre of circulating IgG against E. faecalis in a healthy group of volunteers during a 5-week period.

Table 1: Titres of circulating IgG directed against Enterococcus faecalis in a group of ten healthy volunteers.

<table>
<thead>
<tr>
<th>Volunteer</th>
<th>Serum A</th>
<th>Serum B</th>
<th>Serum C</th>
<th>Serum D</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>2.21</td>
<td>2.36</td>
<td>1.37</td>
<td>0.88</td>
</tr>
<tr>
<td>B</td>
<td>2.31</td>
<td>1.80</td>
<td>1.34</td>
<td>0.84</td>
</tr>
<tr>
<td>C</td>
<td>2.02</td>
<td>1.70</td>
<td>1.38</td>
<td>1.57</td>
</tr>
<tr>
<td>D</td>
<td>3.01</td>
<td>2.73</td>
<td>2.41</td>
<td>2.05</td>
</tr>
<tr>
<td>E</td>
<td>0.01</td>
<td>-0.87</td>
<td>0.01</td>
<td>-0.66</td>
</tr>
<tr>
<td>F</td>
<td>2.08</td>
<td>1.95</td>
<td>1.50</td>
<td>1.44</td>
</tr>
<tr>
<td>G</td>
<td>2.25</td>
<td>1.79</td>
<td>2.07</td>
<td>1.72</td>
</tr>
<tr>
<td>H</td>
<td>2.05</td>
<td>1.88</td>
<td>0.85</td>
<td>1.57</td>
</tr>
<tr>
<td>I</td>
<td>0.51</td>
<td>0.51</td>
<td>0.01</td>
<td>-0.67</td>
</tr>
<tr>
<td>J</td>
<td>1.72</td>
<td>1.56</td>
<td>0.78</td>
<td>0.86</td>
</tr>
</tbody>
</table>

* Serum samples A, B, C and D were collected after 0, 5, 8 and 11 weeks, respectively.

* Titres expressed as the natural logarithm of the median fluorescence level of 1,000 to 3,000 FITC-labeled bacteria minus the median fluorescence level of 1,000 to 3,000 unlabelled bacteria. Logarithmical transformation was performed to improve the normality of the distribution of the fluorescence-levels. Each serum was assayed in duplicate and a 0.9% NaCl-solution instead of serum served as a negative control.

The decrease was even more distinct after a follow-up period of 3 weeks (pool D). After a period of 6 months a long-term follow-up serum sample was obtained from all volunteers. Titres of circulating IgG in these samples were all indistinguishable from the titres of circulating IgG in pools A and B. The decrease of IgG-titres may reflect a mechanism which is responsible for the anti-inflammatory effect which is usually ascribed to probiotics. Further research concerning the influence of E. faecalis on the production of the other Ig-isotypes and on the activation of macrophages and natural killer cells should be performed in order to further elucidate the immune modulating capacity of E. faecalis as a probiotic.

G. Jansen, B. Deddens, M. Wilkinson, D. van der Waaij

References


Received: 26 February 1993/Accepted: 17 March 1993


Book Review

G. J. Galasso, R. J. Whitley, T. C. Merigan (eds.)
Practical Diagnosis of Viral Infections
380 pages
Raven Press New York 1992
Price: $ 49.50

The editors of this book anticipate the expansion of antiviral therapy in general clinical practice in the near future. With their manual they aim to help the practitioner in making both clinical and laboratory diagnoses in time to ascertain the best treatment of viral infections.

Chapters such as “Pathogenesis of Viral Infections” might rather discourage target readers from looking through further chapters dealing with general topics. This is due to the authors’ tendency to compile without comment information and speculation in every subheading (as in “Persistent Infections”). Without information about what is relevant to clinical virology the reader is especially lost when – in an attempt to explain principles – multiple viral infections are described which clearly have different clinical implications (LCM, HBV, CMV as chronic infections).

Hopefully the reader will not be deterred from reading Robert Yolken’s well-balanced, clearly structured, informative overview on what can or cannot be done in “Laboratory Diagnosis of Viral Infections.” Most of the following chapters provide basic information on the topic, but epidemiological data, which are the only help in cases where no rapid diagnostic procedures are at hand, are sometimes too dispersed (gastroenteritis, hepatitis) or abundant (respiratory infections). Astonishingly, a chapter on viral infections with rashes and similar skin manifestations is lacking. For example, nowadays extensive experience with measles and rubella is rare, but abortive manifestations in immunized, partially immune patients attracting wild virus infections at atypical ages have to be detected as early as possible to prevent small epidemics.

All in all, the manual provides an overview of clinical virology with a firmly based view on immunosuppression. As patients with this condition most frequently require antiviral chemotherapy, the editors will achieve their purpose. So the book may help those doctors still acquainted with classical virus disease, but in need of extending their knowledge of viral manifestations under the present circumstances.

J. Forster
Freiburg