immunological situation. The immunological condition of Mitsuda-negative contacts may be potentially different, and this may explain how some turn out lepromatous and some tuberculoid; while others become Mitsuda positive without presenting any clinical signs of the disease, and others still remain Mitsuda negative without showing any signs of the disease either.

The negative Mitsuda reaction in contacts thus indicates an immunological condition the potentiality of which can differ completely from one person to another. This should be carefully considered in connection with the evaluation of the data in Table 3, which show 23 vs 33 lepromin negatives for the vaccinated and unvaccinated groups respectively; and also in connection with Table 4, which shows no lepromatous cases as appearing among the 23 vaccinated persons and only 3 tuberculoid cases, while 6 lepromatous cases appear in the unvaccinated group.

8. If more could be known of the immunological conditions of the vaccinated group with unknown Mitsuda reactions, it would be easier to explain how no cases of contagion were observed in that group.

In conclusion, I must accept the criticism that the groups studied were not strictly comparable, due to the fact that there was a greater number of children—considered more susceptible—in the vaccinated group, and due also to our having added to that group the previously mentioned 110 lepromin-negative persons. However, these circumstances give added strength to the conclusion we reached, namely, that the reduction in the morbidity rate as regards leprosy can be attributed with all certainty to BCG vaccination.

It is our concept at present that BCG vaccination in leprogenic foci is an important prophylactic measure. This vaccine adds its immunologic, synergic effect to the specific defensive phenomena induced by the Hansen bacillus in the inhabitants of leprogenic foci. It would be interesting to get data on the question whether or not BCG vaccination, when used in nonleprogenic foci, would prevent them from becoming endemic.

In my opinion BCG vaccination is a weapon of far-reaching potentials in the fight against leprosy, but there is urgent need for coordinating results obtained by different workers using the same methods and the same manner of administering a standardized BCG preparation.

Ministerio de Sanidad
Caracas, Venezuela

JACINTO CONVIT, M.D.
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THE TUBERCULOSIS FACTOR IN REACTIVITY TO LEPROMIN

To The Editor:

This communication is in reply to your inquiry about certain of the data bearing on my thesis concerning the leprosy and tuberculosis factors in reactivity to lepromin, as summarized in an abstract in the Madrid Congress issue of The Journal [21 (1953) 584]. The paper from which
that abstract was prepared was written in 1952, and a part of it dealing with the influence of BCG vaccination was read at a meeting in Belo Horizonte that year (Anais do XI Congresso Brasileiro de Higiene, Impressora Oficial de Minas Gerais, Belo Horizonte, 1953, pp. 787-790). Although I sent that abstract to Madrid, I changed my mind about publishing the paper itself, awaiting the acquisition of further data.

I believe you are especially interested in the following observations involved in that study, on the bearing of the tuberculosis factor on lepromin reactivity. They pertain mainly to a lot of 292 orphanage children, aged 2-14 years, divided into two groups according to the results of the tuberculin test: (a) 139 negatives (to OT 1/10), and (b) 153 positives. The very dissimilar results of the tests with lepromin (Hayashi-Mitsuda type) are shown in the following tabulation. Also shown are the lepromin results in a group of 257 children of the same age range, without leprosy contact, who had been vaccinated intradermally with BCG after having been found tuberculin negative.

<table>
<thead>
<tr>
<th>Group</th>
<th>No. in group</th>
<th>Mitsuda positive</th>
<th>Mitsuda negative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. &amp; %</td>
<td>F+   F-</td>
<td>No. &amp; %</td>
</tr>
<tr>
<td>1. Orphanage children,</td>
<td>139</td>
<td>9 (6.5)    2    7</td>
<td>130 (93.5) 1 129</td>
</tr>
<tr>
<td>tuberculin negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Orphanage children,</td>
<td>153</td>
<td>142 (92.8) 68 74</td>
<td>11 (7.2)     2   9</td>
</tr>
<tr>
<td>tuberculin positive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. BCG-vaccinated</td>
<td>257</td>
<td>234 (91.1) 123 111</td>
<td>23 (8.9)      2 21</td>
</tr>
<tr>
<td>children (noncontacts)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a All groups aged 2-14 years; averages not available.
b Mantoux test, OT 1/10 dilution.

First to be noted is that only 9 (6.5%) of the tuberculin negatives were Mitsuda positive (but that many did react, despite the lack of evidence of either the tuberculosis or the leprosy factor), whereas no less than 92.8 per cent of the tuberculin positives gave that reaction. The difference could hardly have been more striking. The BCG-vaccinated children gave practically the same percentages as did the natural tuberculin positives of the orphanage group. It is to be understood that none of these children were tested with lepromin more than once; it cannot be said how many of Group 1 would have reacted if tested again.

There was the same correlation trend in the early (Fernandez = F) reaction, but to a much less degree. That reaction was seen in 70 (45.7%) of the tuberculin positives of the orphanage children also in 45.7 per cent of the BCG group, but in only 3 (2.2%) of the tuberculin negatives. Most of the few orphanage children who were Mitsuda positive despite insensitivity to tuberculin were also insensitive to lepromin with respect to the
26, 2 Correspondence

Fernandez reaction—7 out of 9. Of the total of 73 early positives, 70 were among the Mitsuda reactors, although there were 3 among the negatives. Yet it will be noted that in both Groups 2 and 3 (tuberculosis factor present), the late reactors were divided practically equally with respect to the early reaction.

On the other hand, a very few early reactors may be found among Mitsuda negatives, even—rarely—in the absence of tuberculin reactivity, which facts are difficult to explain.

To return to the Mitsuda results of Group 1, the finding that only 6.5 per cent were prepared to react positively to the first test differs notably from the findings in the Philippines of Guinto et al. [THE JOURNAL 23 (1955) 32-47] who, dealing with healthy, country schoolchildren 7-9 years of age, got 34.0 per cent Mitsuda positives among 153 tuberculin negatives—proportionately over five times as many as in my group.

On the other hand the 92.8 per cent of my Group 2, where only the tuberculosis factor is known to have been operative, is materially higher than the 77.5 per cent that Guinto et al. got in their 391 tuberculin-positive children. This difference would be hard to explain, unless it could be done on the ground of a difference of antigenicity of the lepromins used.

In considering these results in tuberculin negative children the Editor would doubtless give some importance to a third factor, i.e., environment. The orphanage children had spent all of their lives—or some material proportion thereof immediately preceding the testing—in the institution where the environment is exceptionally clean. Consequently, they had been protected from nonspecific, natural influences of the outside world, especially the soil, which might “condition” the individual to react to lepromin (or to large doses of tuberculin). These influences would have been at play with Guinto’s children.

Be that as it may, the comparison between the two groups of orphanage children is an exceptionally “pure culture” observation of the influence of natural contamination with the tubercle bacillus.

THE LEPROMIN REACTION IN TUBERCULOID REACTION CASES

(Continued)

To THE EDITOR:

Some time ago there was a lesser symposium in THE JOURNAL [24 (1956) 86] about the weakening or abolition of responsiveness to lepromin in tuberculoid cases during clinical reactions. The specific question was whether or not a patient with tuberculoid leprosy who was strongly reactive to lepromin before the clinical reaction would become completely nonreactive during that condition. However, certain of your correspond-