Lactase deficiency in a rural area of Mexico

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The prevalence of the adult type of intestinal lactase deficiency has marked racial variability and it is common in most non-Caucasian groups (1). Although to our knowledge there are no conclusive data regarding lactose tolerance in Mexico, a recent paper (2) suggests that the incidence of lactase deficiency may be frequent. In 11 Mexican-American males studied, 6 were shown to have the abnormality (2). The possibility that lactase deficiency might interfere with milk digestion, and the fact that at present there is a government drive in Mexico to increase milk consumption generally, encouraged us to study a rural community in order to try to answer two questions: a) Is lactase deficiency common in Mexico? b) If so, does it interfere with milk consumption?

Material and methods

The survey was done in Huanantla County, State of Tlaxcala, which is located approximately 100 miles southeast of Mexico City. Two groups were studied in the city of Huanantla, which has a population of 26,000, composed mostly of Mexican mestizos, and another group in the small locality of Ixtenco, with a population 1,500, some of which inhabitants still speak an Indian language, Otomi, and are considered to be less heterogeneous than those of Huanantla. Furthermore, the latter group belongs to a lower socioeconomic stratum than the urban one. In Huanantla, 193 high school students were studied; both sexes were equally represented, and their ages ranged from 13 to 17 years. The other group from that city was comprised of 100 factory workers, mostly males ranging in age from 18 to 72 years. In Ixtenco, 108 male and female high school students between 13 and 21 years of age served as the subjects of our study.

A lactose tolerance test was performed on all subjects. A standard dose of 50 g lactose was given to each individual (all weighed over 25 kg), and capillary blood samples were obtained before, 15, and 30 min after the lactose load. All subjects were in a fasting state. A field reflectometer (Ames Co., Elkhart, Indiana 46514) was used to measure blood glucose concentrations. To classify our results, we followed international standards (3); if an increase of blood glucose over 25 mg/100 ml of the basal value was obtained, the subject was classified as tolerant (normal intestinal lactase levels); if the increase was between 20 and 25, it was considered a doubtful result; and increases below 20 ml/100 ml indicated lactose intolerance (deficient intestinal lactase levels). Before the performance of the test, each individual submitted to a brief interrogation and related his or her milk drinking habits and general health status, particularly the existence of chronic or acute diarrhea. If the subject had diarrhea, he did not participate in the test. All subjects were seen 2 hr after testing so we could detect any untoward reactions to the lactose load.

In order to test the general validity of the methodology employed, two studies were done before the field trip: a) two glucose determinations were done at 3-min intervals on 20 individuals; the intrapair differences were found to be minimal and nonsignificant; b) in 20 subjects, the lactose tolerance test was performed by simultaneously measuring the glucose levels from capillary blood (with the reflectometer) and from venous samples (using the AutoAnalyzer).

As can be seen in Table 1, the concordance was good with only one major discrepancy, one individual was
classified as normal with the capillary method and as deficient with the venous or AutoAnalyzer technique. In addition to the lactose tolerance test, most individuals were typed for ABO blood groups, utilizing standard reagents.

**Results**

Table 2 shows the results obtained in the groups investigated. In addition to the data regarding the lactose tolerance tests, two other results are included: the presence or absence of gastrointestinal symptoms after the lactose loading test and an estimation of milk consumption. The latter was considered high when the individual's usual consumption was more than 1 glass of milk daily, and low when it was less than that amount. In the adolescents from Huamantla, 50% of those classified as having a high milk intake had at least 2 glasses of milk daily, whereas in Ixtenco, the majority ceased to drink milk from the time they were weaned from breast feeding.

Table 3 shows the results of the blood grouping data. The proportion of non-Indian genes was calculated by using Bernstein's formula:

\[
\text{Percent of gene admixture} = \frac{qX - Q}{q - Q} \times 100
\]

in which \(q\) and \(Q\) represent the frequencies of the theoretical base populations and \(qX\) is that of the hybrid one. Assuming that "pure" Indians lack gene A, and that in Spaniards, A has a frequency of 0.29 (4), the following admixture percentages are obtained: Huamantla adolescents, 41.3%; Huamantla adults, 42.7%; and Ixtenco high school students 28.4%.

**Discussion**

The blood group data (Table 3) indicate that the three groups investigated are mixed, as pure Indians are characterized by having an O blood group frequency close to 100%. As expected by their cultural characteristics, the Ixtenco group has less admixture than the Huamantla ones. In addition, the same table shows that all these groups, although hybrids, are in Hardy-Weinberg equilibrium.

The first question posed is readily answered by our data, and we have concluded that intestinal lactase deficiency is common in the groups investigated, as it is present in 296

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**TABLE 1**

Concordance of the lactose tolerance test measuring blood glucose levels from capillary (reflectometer) and venous blood (AutoAnalyzer)

<table>
<thead>
<tr>
<th>No. of individuals classified as:</th>
<th>Lactose tolerance test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ames reflectometer</td>
</tr>
<tr>
<td>Deficient</td>
<td>16</td>
</tr>
<tr>
<td>Doubtful</td>
<td>2</td>
</tr>
<tr>
<td>Tolerant</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
</tr>
</tbody>
</table>

**TABLE 2**

Correlation between the results obtained in the lactose tolerance test with the presence of symptoms and milk consumption

<table>
<thead>
<tr>
<th>Huamantla</th>
<th>Adolescents</th>
<th>Adults</th>
<th>Ixtenco</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of subjects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptomatology</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>105</td>
<td>7</td>
<td>16</td>
</tr>
<tr>
<td>Absent</td>
<td>43</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>Ingestion of milk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>79</td>
<td>12</td>
<td>22</td>
</tr>
<tr>
<td>Low</td>
<td>69</td>
<td>4</td>
<td>7</td>
</tr>
</tbody>
</table>

1 = intolerant; D = doubtful; T = tolerant.

A Abdominal cramps and/or flatulence and/or diarrhea within 6 hr following the test. B Daily milk ingestion of one glass or more. C Daily milk intake less than one glass, many have no consumption.
TABLE 3
Distribution of ABO blood groups in the three populations studied

| Group studied     | No. | O   | A   | B   | AB  | p(A) | q(B) | r(O) | $X^2$ | P >  
|-------------------|-----|-----|-----|-----|-----|------|------|------|-------|-------
| Huamantla, adolescents | 192 | 69.28 | 21.35 | 7.29 | 2.08 | 0.1199 | 0.0428 | 0.8373 | 2.01 | 0.50  
| Huamantla, adults   | 89  | 67.42 | 20.22 | 11.24 | 1.12 | 0.1243 | 0.0752 | 0.8003 | 0.61 | 0.80  
| Ixtenco            | 91  | 82.41 | 12.96 | 4.63 | 0.00 | 0.0824 | 0.0304 | 0.8872 | 0.40 | 0.90  

$^a$ Observations against expected phenotypes are compared, the latter being calculated with the gene frequencies obtained.

individuals (73.8%) of the 401 studied (Table 2). The proportions of normal, doubtful, and deficient subjects in the three groups are quite similar, the differences being statistically non-significant ($X^2 = 2.60, P > 0.50$). In view of the fact that the Ixtenco population is less homogeneous, it might be expected that the prevalence of deficient individuals would be higher. This was not seen, which may be explained by the fact that 1) the differences in admixture are not great, 2) some Caucasian groups (5) have a high prevalence of lactase deficiency, and 3) there are no published data regarding Spaniards who constitute the main Caucasian gene group in this instance. At any rate, the proportion of deficient subjects encountered in this study is similar to what has been described for Mexican-American males (2) and for Indians and Eskimos in Alaska (6).

Symptomatology does not occur at random in Huamantla, and if the three groups are analyzed together ($X^2 = 13.28, P < 0.01$), symptoms do appear more frequently among the deficient subjects. In fact, as can be seen in Table 2, 192 of the 296 deficient individuals (64.9%) had gastrointestinal symptoms following the lactose load, as did 41.3% of the doubtful and 46.3% of the normal subjects. In Ixtenco, our results were different, and we have no explanation for this fact. Symptoms vary from explosive diarrhea, accompanied by flatulence and abdominal cramps, to any of the three symptoms alone. The calculations above were made considering as positive the presence of any of these symptoms. If, however, only diarrhea is considered, the number of people scored as positive decreases, but the same relative differences between the deficient and the other persist. It is worth noting that some subjects with a normal lactose tolerance test have diarrhea after the lactose load, whereas some of the deficient individuals are free of any symptoms. The reason for this phenomenon is not well-known but it has also been observed by other investigators (5, 7). In any event, it points out that studies utilizing exclusively clinical data would correlate poorly with those employing the lactose load.

A striking fact shown by this study is that, independent of the milk consumption habits of the three groups investigated (which are quite different (Table 2)), the prevalence of lactose deficiency is similar. This suggests that in man, milk drinking is incapable of inducing intestinal lactase activity, a conclusion reached by other authors as well (8–10).

The answer to our second question is not completely clear. Table 2 shows that many deficient subjects were grouped among those who daily drank one or more glasses of milk. However, it is worth noting that among the adolescent group in Huamantla, the proportion of normal subjects with high milk consumption is significantly different ($X^2 = 8.61, P < 0.05$) from that of the other individuals. This phenomenon is not observed in the other two groups, or if all three are analyzed together. However, it is precisely in the first one that, for several reasons, milk availability is clearly greater than in the other and suggests the possibility that at least some deficient individuals limit milk ingestion because of clinical intolerance. To further test this idea, a comparison was made between the proportion of deficient individuals who drank no milk in each group against the general percentage of deficient subjects, and no statistical differences were found. In spite of the above result, we
believe that the question is not settled, especially because the authors of a recent study (11), performed in the United States among black and white children, obtained data suggesting that milk consumption was definitely higher in individuals with normal lactose levels.

From the pragmatic point of view, it seems clear that intestinal lactase deficiency cannot be equated with milk intolerance, at least in the quantities ingested by the subjects studied. Any program designed to increase milk consumption in the Mexican population, at least to a level similar to that seen in the adolescents of Huamantla, would probably be well tolerated by most individuals, regardless of their intestinal lactase activity. Further studies are in process to test this idea.

Summary

The prevalence of the adult type of intestinal lactase deficiency in three groups of residents of a rural area of Mexico was studied. Of the 401 individuals tested, 73.8% were intolerant, and the relative frequency of intolerance in each group was similar. However, traditional milk consumption habits of the lactose-intolerant individuals were quite different. In one group, few people had consumed any milk since they were breast fed; in another, over 50% ingested more than one glass of milk daily, and the other was intermediate. The symptomatology following the lactose load was more frequent in those individuals classified as intolerant, but some tolerant ones also had symptoms, whereas some of the intolerant ones did not. Whether low intestinal lactose levels interfere with milk consumption is not definitively answered by this study, but it seems clear that many individuals classified as intolerant are able to ingest milk without difficulty, at least in the relatively small quantities that are typical of this and many other regions of Mexico.

References