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COMMENT ON ALKANANI ET AL.

Alterations in Intestinal Microbiota Correlate With Susceptibility to Type 1 Diabetes. Diabetes 2015;64:3510–3520

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The search for potential microorganisms associated with autoimmunity and their metabolic role is crucial to understand the origin and evolution of type 1 diabetes (T1D) and other autoimmune diseases. An interesting article by Alkanani et al. (1) published in *Diabetes* described the microbiota structure of American patients with new-onset T1D, comparing them with subjects with islet autoimmunity, seronegative first-degree relatives, and a healthy control group without family history of autoimmunity.

One of their main findings was an increase in the relative abundance of *Bacteroides* and a decrease of *Prevotella* in seropositive subjects with multiple antibodies versus one autoantibody. These results are consistent with ours in Mexican children with T1D compared with healthy control subjects (2). Thus, both studies support the existence of a diabetogenic microbiome that contributes to T1D development and could help explain the rapid progression to T1D in children with multiple autoantibodies.

In addition, Alkanani et al. compared the microbial diversity among seropositive and seronegative subjects and no significant differences were found. These findings differ from a study by a European group (3), which presented a reduced diversity after seroconversion, but are similar to our findings in Mexicans (2) who were born and reside in the Arizona-Sonora border. This suggests that islet autoimmunity and T1D progression are probably not associated with altered bacterial diversity but with the bacterial abundance in the gut microbiota.

In the article by Alkanani et al., there was no effect of HbA_{1c} or T1D duration time when relative abundances in new-onset T1D and seronegative groups were compared. These data are not shown in the supplementary material, and according to the inclusion criteria, only patients with less than 6 months' duration were considered. These results differ from those found in T1D Caucasian (4) and Mexican (2) children, where significant associations among those factors were present 2 years after diagnosis.

Finally, it is known that age is a strong driver of gut microbiota composition (3) and that microbiota are more unstable in infants than in adults. Regarding this, Alkanani et al. considered a very wide age range (2–45 years), but later adjusted it to include only subjects younger than 18 years. As a result, the microbiota variability of the youngest age-group may have subtracted significance from their results by avoiding the detection of further differences between their groups.

Duality of Interest. No potential conflicts of interest relevant to this article were reported.

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