celiac sprue occurs at the same time as critical tissue development and the resulting tissue may reflect the nutrient deficiencies and/or abnormal immune response.

For part of the study, 86 children ranging in age from 3 to 22 years, who had biopsy-proven celiac disease (41 boys and 45 girls) were studied. Ten of these children had only deciduous teeth (baby), 45 had various combinations and the remaining had fully developed permanent teeth. Control subjects were used for both the dental enamel defects portion and the dental maturity segment. Dental enamel defects (hypoplasias) were studied using systematic observation, photography and dental casts. For children with only “baby teeth”, x-rays of the first permanent molars were studied to determine the degree of enamel defects. Dental maturity was estimated from both clinical eruption of teeth and from a score obtained from full-mouth x-rays of seven left upper teeth.

The results showed that of the 76 celiac children with permanent teeth, 73 (96%) had dental enamel defects. This compares with 47 (31%) of the 150 clinical controls. Of the celiac children with enamel defects, 75% of their permanent teeth were affected, compared to only 8% in the control subjects. In the celiac subjects, at least two groups of teeth (ex. Incisors and molars) were affected.

The two upper front teeth (maxillary permanent central incisors) were always affected. This is understandable, since 90% of the enamel of these teeth develops between 10 months and 4 years – when the malabsorption and immune response in undiagnosed celiac sprue is unchecked.

It is not known whether it is the malabsorption or immune response that is primarily responsible for the enamel defects. While the enamel defects in the teeth of those with celiac sprue were symmetrical and time-related, the enamel defects in the control children were not symmetrical or systematic.

Also to be noted is the severity of the enamel defects was greater in celiac children than in the control group. Twenty-nine percent of the celiac children had evident or severe defects as compared to less than 2% of the control subjects.

Enamel defects proceed from the tip of the tooth towards the area that will be situated near the gum line. Therefore, a fairly accurate estimate can be given of when the enamel defects occur. Using the upper two front teeth as a time line indicator, the effects of gluten ingestion was studied as well. For the 14 of 16 children who were diagnosed before the age of 2 years and had strictly followed their diet, the enamel area formed last (adjacent to the gum) was not affected, although the preceding enamel areas were. In the 10 of 11 who had been diagnosed and then took the gluten challenge before the age of 3, enamel defects occurred in the gingival portions of the tooth enamel as well as other areas. Twenty-eight of the 39 diagnosed after the age of 4 showed severe enamel defects.

An interesting issue was discovered during this study – there appeared to be a direct correlation between the severity of the clinical celiac symptoms and the dental damage in the 76 children with permanent teeth before the age of 4 years. The more severe the symptoms, the more severe the enamel defects.

When the dental maturity was measured, those with celiac sprue were delayed in comparison with the control group. Children with celiac sprue had later eruption of their permanent teeth. The bone age of the celiac children was retarded compared to the control group.

Because there still seems to be a great deal to learn in this area, I am continuing my research and will have an update for you in future issues. Cleo Anderson, SACS president elect