Editorial

Many steps define the evolution of mankind from its primitive existence to modern life. These span the spectrum of the ability to produce fire, the invention of the wheel, up to modern inventions such as electricity and those associated with the industrial revolution. Among the steps, the ability to cultivate grains and use wheat as part of our diet is considered as an integral part of human development, enabling an enrichment of the diet and the creation of technological solutions for food products and baking.

These innovations, dating back about 10,000 years (with recent archeological findings suggesting over 20,000 years), improved the taste, texture, and variety of our diet; however, alongside they brought about a new disease, celiac disease (CD), which would not exist without the consumption of gluten, the wheat protein, and its related prolamins present in barley and rye.

In recent years, it has become apparent that gluten contains immunological properties that may contribute to the development of CD, for which a genetic predisposition is required. These properties may also play a role in a wide range of diseases that can be attributed directly to wheat (e.g. wheat allergy and non-celiac gluten sensitivity) or could be responsive to gluten elimination from the diet due to its potential toxic effects. The current issue of *Annals of Nutrition and Metabolism* is dedicated to a fascinating protein named gluten. Frits Koning elegantly describes this complex protein and provides us with the

known immunological mechanisms by which gluten may exercise its toxic effects.

Carlo Catassi opens a window to a wide range of diseases that may or may not be related to gluten, of which non-celiac gluten sensitivity stands out as the most intriguing entity due to the difficulties in its objective diagnosis and its potential prevalence which could be much higher than that of CD. Time will tell whether all or only a few of the conditions detailed in Catassi's review will be cured or improved with a gluten-free diet and what would be the exact role of other food ingredients (e.g. α -amylase/trypsin inhibitors) in the pathogenesis of various wheat-related symptoms and diseases.

Yolanda Sanz reminds us that our microbiota may play an important role in CD pathogenesis, as immune dysfunction seen in subjects with chronic inflammation and autoimmune disorders causes unfavorable changes in our gut microbiota named dysbiosis. This is also true for CD patients where such changes could explain part of the environmental triggers that are needed to initiate the disease on top of the genetic predisposition. These changes may be part of the pathogenesis of CD and may aggravate overt disease. This elaborate review provides observations on the associations reported between gut microbiota alterations and CD. Furthermore, the gluten-free diet itself has an effect on gut microbiota, and these complicated interactions are discussed as well.

Finally, as CD is so common and carries the risk of complications, various infant nutrition strategies were for a long time viewed as a means of preventing CD or delaying the presentation of the disease. Following the Swedish epidemic, it was thought that breastfeeding prevents or delays the presentation of CD and early introduction of gluten (between 4 and 6 months of age) while the infant is still being breastfed as a protective or delaying effect as well. Anna Chmielewska and colleagues summarize the available evidence on these strategies and show, to our disappointment, that breastfeeding does not protect chil-

dren at risk of developing CD and that the early introduction of gluten is not protective as well.

We are sure that the curious reader will find updated answers to many questions on gluten consumption, gluten sensitivity, CD pathogenesis, and dietary treatments; hopefully, these answered questions will initiate prospective studies on the many new and open questions in the field.

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