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Vitamin C as a supplementary therapy for celiac disease?

Lindfors, K.; Kaukinen, K.

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Introduction

Celiac disease is one of the most common food-related enteropathies in Western countries, affecting up to 1–2% of the European population.[1, 2, 3] The disease occurs in a subgroup of genetically predisposed individuals who are positive for either human leukocyte antigen DQ2 or DQ8. The disease is triggered by consumption of dietary wheat-, rye- and barley-derived gluten and it often manifests with intestinal symptoms such as diarrhoea and malabsorbtion. The intestinal symptoms correlate with findings of small-bowel mucosal damage characterised by villous atrophy with crypt hyperplasia. Celiac disease is also hallmarked by the presence of antibodies against gluten-derived gliadin peptides as well as autoantibodies against transglutaminase 2.[4] The small-bowel mucosal injury in celiac disease develops gradually from initial infiltration of intraepithelial lymphocytes to shortening of the villus structure together with enlargement of crypts, finally into overt villous atrophy and crypt hyperplasia in response to activation of the small-intestinal innate and adaptive immune responses. Gluten-derived gliadin contains high amounts of repetitive glutamine- and proline-rich sequences, this rendering them highly resistant to proteolytic degradation by human gastric, pancreatic and brush-border enzymes, even in healthy individuals.[5, 6] Thus even fairly long peptides persist, some of which have been identified as toxic, inducing early effects on the small-bowel mucosal epithelium.[7, 8] Of the innate immune components these peptides induce, interleukin (IL)-15 has been shown to play a central role in the pathogenesis of celiac disease, as it contributes to intestinal epithelial cell destruction.[9] On the other hand, another subset of the gliadin peptides are immunogenic...

Lindfors, K.[a]; Kaukinen, K.[a,b]

[a]Pediatric Research Center, University of Tampere and Tampere University Hospital, Tampere, Finland

[b]School of Medicine, University of Tampere and Department of Gastroenterology and Alimentary Tract Surgery, Tampere University Hospital, Tampere, Finland

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