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### Title: Gluten degradation by the gut microbiota of ulcerative colitis patients

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**Background:** Several studies have reported improved disease symptomology in ulcerative colitis (UC) patients consuming a gluten free diet. This observation coupled with diversity depletion in the gut microbiota of UC patients led us to hypothesize that UC-associated enteric microbes differentially metabolize dietary gluten to produce immunogenic products that promote inflammation.

**Methods:** Gluten concentration in stool was determined using gluten-specific ELISA, and gluten intake was assessed by food frequency survey in UC (n=12) and healthy controls (HC; n=13). Gluten-metabolizing bacteria were isolated on minimal media supplemented with 1% gluten from UC and HC and identified by 16S rRNA profiling. Cell-free culture media from gluten metabolizing gut bacterial isolates was assessed for immunogenicity in vitro using HT29 colonocytes.

**Results:** Compared to HC, UC patients didn't consume gluten differently (Mann-Whitney; p > 0.10) and exhibited equivalent levels of gluten in their feces (Mann-Whitney; p = 0.163). The profile of gluten-degrading bacteria isolated from UC stool was distinct (Chi-square; p = <0.0001). Compared with Enterococcus isolates, products of gluten degradation by Bacillus strains induced higher IL8 and lower occludin (Mann-Whitney; p = 0.002 and p = 0.059 respectively) gene expression in colonocytes irrespective of whether they originated from UC or healthy gut

**Conclusion:** Members of HC and UC microbiota exhibit gluten-degrading ability, metabolites of which influence genes involved in inflammation and barrier function in enteric colonocyte cultures. Preliminary findings of this study warrant further investigations into the mechanisms by which gut microbiota contribute to UC pathogenesis through gluten degradation.

#### Biography

Emma Harringer has completed her Medical Degree at the age of 27 years from University of Southern Denmark and her predoctoral fellowship from University of California San Francisco, supported by the Lundbeck Foundations DARE program. She is starting her medical career at Hvidovre Hospital as a gastrointestinal surgeon this November and are hoping to join your International Conference on Gastroenterology and Liver next summer.

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## Title: Serum trace element alteration in patients with non-alcoholic fatty liver disease: an evidence from cross- sectional study

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**Objectives:** Trace elements play an essential role in metabolic functions of the liver; however, their levels get altered during pathophysiological states. In this study, we aimed to evaluate and compare serum levels of selective trace elements, namely, Copper, Iron, Magnesium, Manganese, and Zinc, between the non-alcoholic fatty liver disease (NAFLD) patients and healthy volunteers.

**Methods:** A cross sectional study was conducted by enrolling NAFLD patients (n=170) and apparently healthy volunteers (n=62). Inductively coupled plasma- atomic emission spectrometry (ICP-AES) was used to quantify the serum levels of selected trace elements. We also measured serum levels of Tumor necrosis factor (TNF $-\alpha$ ) and malondialdehyde (MDA) to investigate the effect of NAFLD progression on trace elements.

**Results:** In NAFLD patients, serum Copper levels [88.5 (38.1, 286.7)  $\mu$ g/dL] were higher than healthy volunteers [74.9 (25.5, 148.8)  $\mu$ g/dL] with p value <0.001. In contrast, Zinc levels were lower in NAFLD group [107.3 (12, 270.6)  $\mu$ g/dL] as compared to health volunteers [125 (88.7, 261.1)  $\mu$ g/dL] with p value <0.001. Likewise, In the NAFLD patients, Iron, Magnesium, and Manganese levels were also lower than healthy volunteers with p value <0.001. Furtherment— $\alpha$  and MDA levels were significantly higher in the NAFLD group as compared to healthy volunteers.

**Conclusions:** The NAFLD patients had significantly altered serum trace element profile as compared to healthy volunteers, suggesting the role of trace elements in the pathogenesis of NAFLD. Hence, a trace element-based intervention can have a potential therapeutic role in the management of the NAFLD.

#### Biography

I completed my post-graduation (master's in medical pharmacology) at the age of 25 years from All India Institute of Medical Sciences, New Delhi, India which is a premier medical institute in the country. I have published 6 papers in reputed journals. 3 other manuscripts are communicated to reputed international journals. Currently, I am pursuing Doctor of Philosophy in Pharmacology from the same institute. I am working on animal models of Non-Alcoholic Fatty Liver Disease. I have an experience of animal handling and surgery, cryosectioning and histopathological staining, performing ELISA, working with ICP- AES, performing western blotting and real time PCR.