

# Tight junctions protein genes expression in gluten sensitive patients

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# Background:

Reaction to gluten can involve an allergic (wheat allergy), non-allergic [gluten sensitivity (GS)], or an autoimmune [celiac disease (CD)] mechanism.

Recent evidences suggest that early changes in intestinal permeability (IP) may play a pivotal role in the pathogenesis of CD.

Conversely, no data are currently available on the role of intestinal barrier dysfunction in the pathogenesis of GS .



# Aims:

To investigate the changes in IP and TJ protein genes expression of Claudin 1, 2, 3, 4 and ZO-1 in intestinal mucosa of celiac disease and gluten sensitive patients.

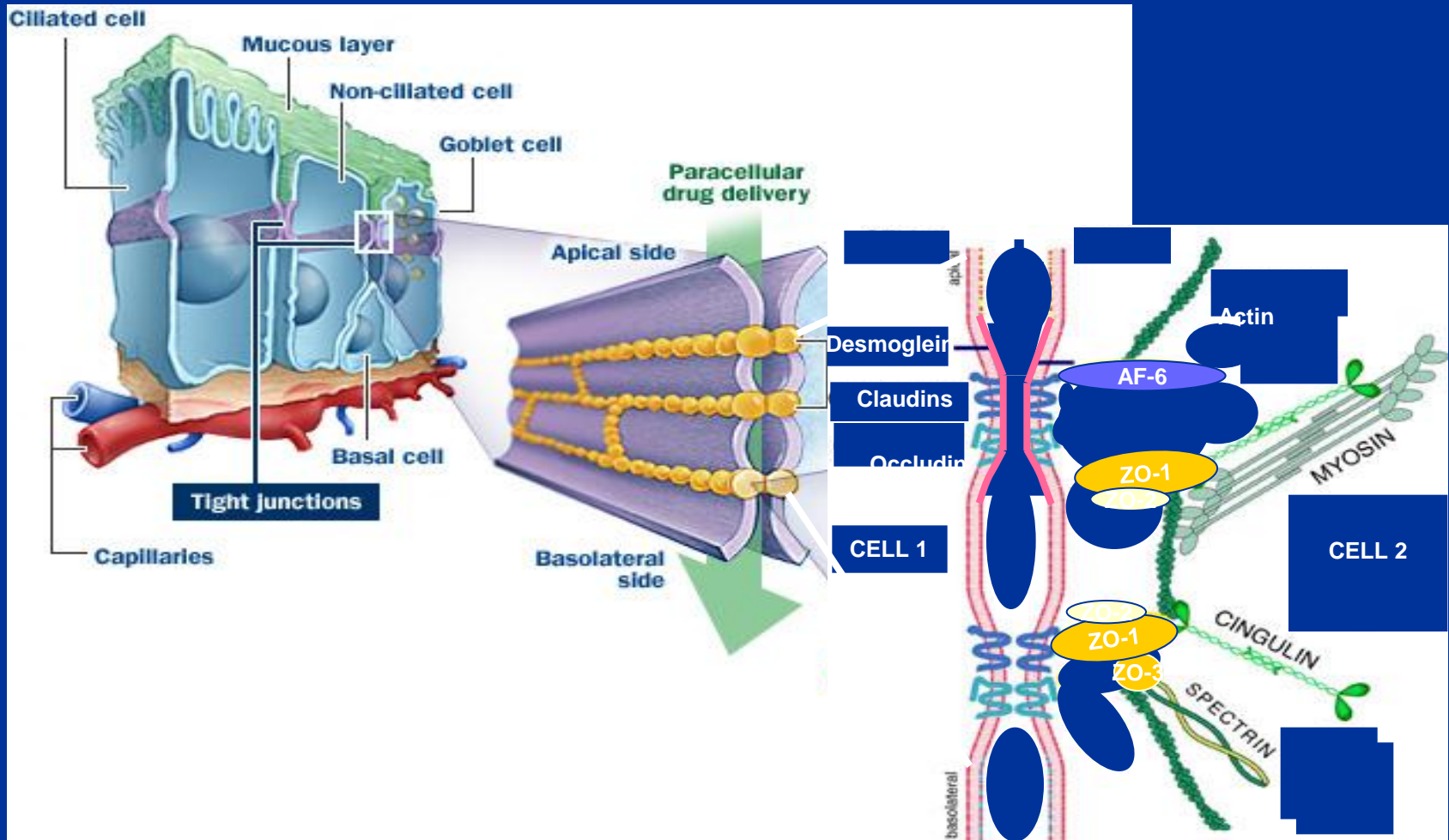


# Methods:

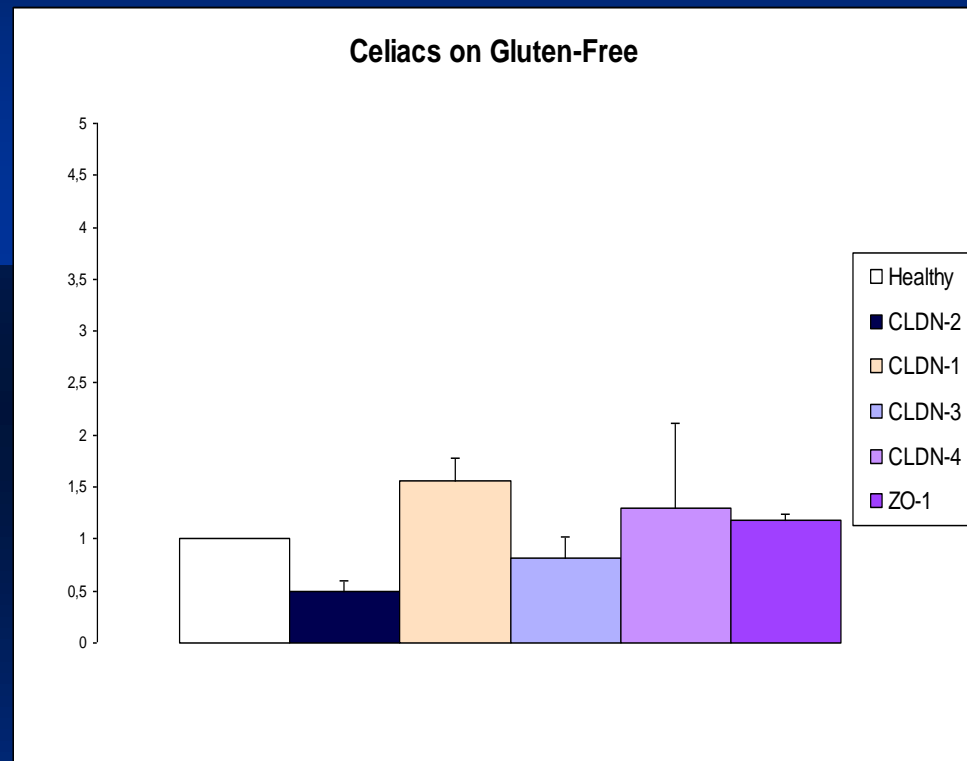
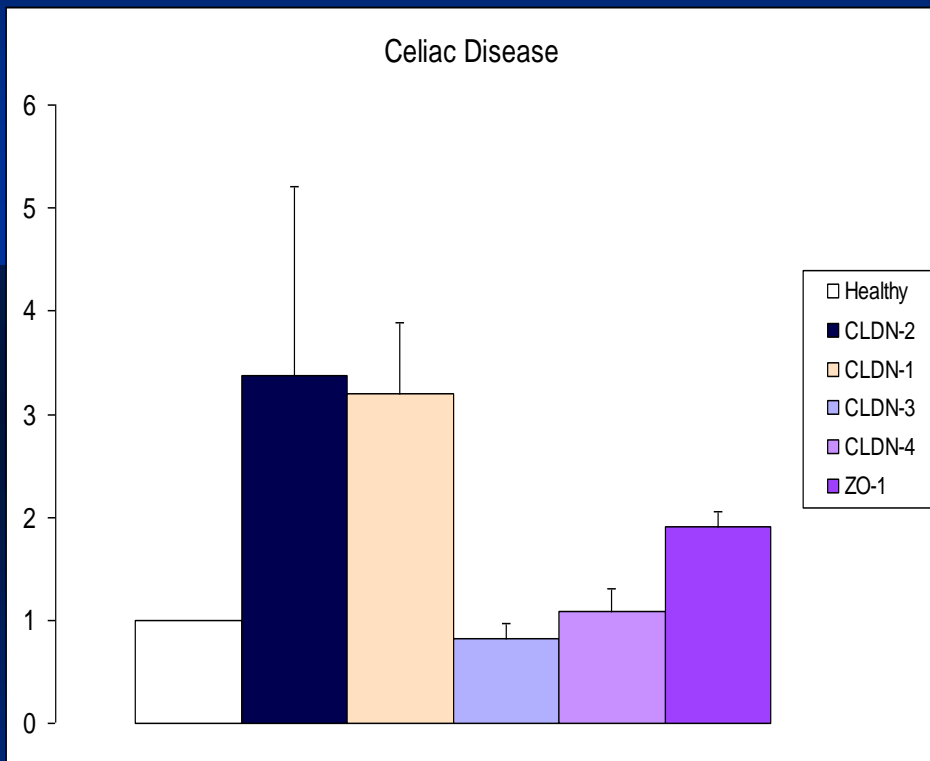
- Biopsy samples were obtained from 8 GS patients, 13 patients with active CD, 3 patients with CD in remission, and 10 healthy controls.
- Quantitative gene expression of tight junctions proteins Claudin (CL) 1, CL2, CL3, CL4, and ZO-1 was performed by Real-time PCR.
- IP was evaluated by means of the lactulose/mannitol test (LA/MA).



# Intercellular Tight Junctions



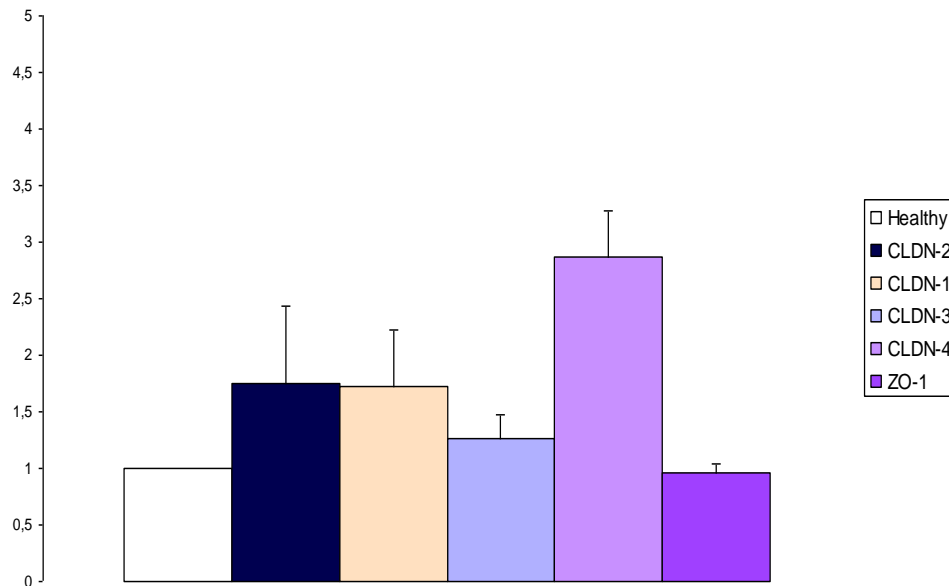
# Results: Intercellular TJ expression CD



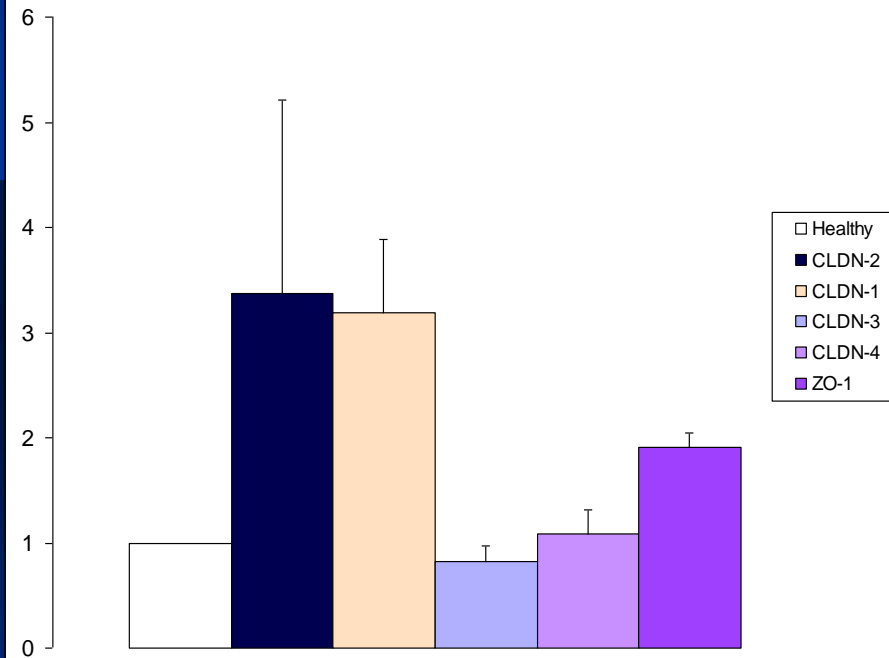
A significant over-expression of both CL1 and CL2 was observed in CD patients, while no significant changes in CL3, CL4, and ZO-1 were detected, compared to CD patients on gluten free diet.

# Results: Intercellular TJ expression GS

## Gluten Intollerants



## Celiac Disease



Expression of CL4 was increased three folds in GS subjects compared to both CD patients and healthy controls, while no changes in CL1, CL2, CL3, and ZO-1 expression were detected.

# Conclusions:

- Compared to CD patients, GS subjects showed normal IP and CL1 and CL2 expression.
- Up-regulation of CL4 in GS patients did not influence IP.
- These results suggest that the pathogenesis of GS is different from that of CD and does not involve the loss of intestinal barrier function.