

GUT-DRIVEN VASCULAR INFLAMMATION AND OXIDATIVE STRESS: EXPLORING THE MECHANISMS OF ENDOTHELIAL DYSFUNCTION IN AN EXPERIMENTAL COELIAC DISEASE MODEL

Sebastian Steven

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A humanized mouse model of coeliac disease demonstrates that gluten challenge triggers intestinal inflammation, elevated blood pressure, vascular dysfunction, and oxidative stress, with IL-17A as a potential mediator linking gut and cardiovascular pathology.

KEY POINTS

- Gluten-challenged NOD-DQ8 mice developed duodenal and jejunal T-lymphocyte infiltration and reduced villus/crypt ratio, mirroring human coeliac disease.
- Gluten challenge increased blood pressure and impaired endothelium-dependent and -independent vascular relaxation compared to controls.
- Pro-inflammatory gene transcripts were upregulated in both duodenum and aorta, with increased CD11b+ myeloid cell infiltration into the aortic wall and elevated oxidative stress markers in heart tissue.
- Plasma IL-17A concentrations were elevated in gluten-challenged mice, and all cardiovascular complications were reversible upon return to a gluten-free diet.
- The findings support a mechanistic gut-to-cardiovascular inflammatory axis in coeliac disease, relevant to researchers investigating autoimmune disease and cardiovascular risk.

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