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of being an important mediator of the inflammatory response to tissue damage and infection, and it is released from activated monocytes and macrophages, and also from necrotic cells.³⁹ The chromatin-rich granules of phagocytic cells (e.g. the neutrophil and the eosinophil) and other mononuclear leukocytes accumulate calcium and magnesium during phagocytosis.³⁹ In this way calcium becomes "trapped" within the phagocyte's granules and is later released during cell death.⁵⁰ Therefore, there is considerable interest in the possibility that the onset of tissue damage may play a role in the onset of ischemia-associated DIND because,

during an ischemic insult, the ATP-dependent flux of Ca²⁺, known as Ca²⁺ homeostasis, depolarizes cell membrane potential, which may allow entry of Ca²⁺ through non-ATP-dependent channels.³⁹ Thus, IP₃ releases the Ca²⁺ from the stores which in turn opens the store-gated Ca²⁺ channel.³⁹ This influx of Ca²⁺ initially causes some cell swelling (osmolality).⁴⁷ Indeed, the prevalence of IBS in these two populations was similar to the prevalence in the general population.² (see Text S1 for additional references). Andrey et al.⁴ Andrey et al.⁴ Hirsch et al. However, the TH2 immune response in the gut mucosa is critical in the pathology of celiac disease because TH2 immune responses promote the loss of tolerance to gluten in the intestinal mucosa and induce inflammation-related tissue damage.³¹ Consecutive studies investigated the role of CD over the years.⁹ CD is found in 4-5% of patients with primary open-angle glaucoma (POAG) and in approximately the same number of patients with a family history of open-angle glaucoma.¹⁰ Taken together, these findings indicate that TH2-derived mediators may actually participate in the pathogenesis of CD.^{20.22.23.24.30.31.51} The prevalence of CD in these patients was 3.1% of the total 1,760 patients with IOP > 21 mm Hg (average follow-up duration, 3.4 ± 0.8 years). It was 3.4% of the 161 patients with POAG (average follow-up duration, 3.3 ± 1.0 1cb139a0ed

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