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CŒLIAC DISEASE AND AGEING

The overall prevalence of **cœliac disease** appears much higher than previously thought. Approximately one percent of the population, in most countries of Western Europe, suffers from cœliac disease. But lots of those patients remain undiagnosed, both because the disease stays silent (which doesn't mean that it is harmless) and because the diagnosis is often missed for a long time. Reasons for the lack of proper diagnosis are the belief that cœliac disease only appears among infants and small children, and also the belief that patients have to complain from diarrhea and others digestive troubles to be affected.

Indeed, two first years of life provide many diagnoses from diarrhea, loss of weight or stunted growth. Even if some cases are uncovered during the next decades, a lot of them are detected, for unknown reasons, during the fifth decade of life. Then, more patients are diagnosed much later, as shown from different publications: **19 %** are **65** years old or older at the time of diagnosis in one study; **29 %** are diagnosed at or after age **60** in another one. The prevalence of remaining undiagnosed cœliac disease is significant among the elderly.

The diagnostic of "**leaky gut syndrome**" is proposed when the intestinal mucosa looses its barrier function (normally provided by tight junctions between the enterocytes) and its absorption function (normally provided by the microvilli from the brush border). This condition is characterized by the abnormal entrance of large amounts of antigens and toxins, due to leaky tight junctions between the enterocytes, thus triggering the immune system and the liver detoxification pathways. Multiple micronutritional deficiencies develop due to the damaged brush border, devoid of proper absorption. ***Zonulin*** is a novel human protein (identified in 2000) which induces tight junction disassembly and a subsequent increase of intestinal permeability, for reasons that are not yet clear.

Still, it has been discovered in 2003 that ***gliadin*** - the most allergenic sub protein from ***gluten*** - induces zonulin release in intestinal epithelial cells. Indeed, we know since a quarter of a century that, among patients suffering from cœliac disease, both gastric permeability and intestinal permeability are characteristically elevated. This was not surprising since those patients display a severe villous atrophy (the cornerstone for diagnosis confirmation), but we have now uncovered the pathologic role of gliadin, inducing abnormal zonulin release.

Consequently, we understand multiple pathologies resulting from cœliac disease such as **fatigue**, **anemia**, **osteoporosis**, **neuropathy** and **depression** (being the result of multiple essential micronutrients deficiencies); such as elevated liver **transaminases** (being the result of liver toxic overload); such as autoimmune diseases like type I **diabetes** and **thyroiditis** or **cancer** (being the result of increased intestinal permeability and its negative impact on the immune system). All have been repeatedly published in mainstream medical journals and they obviously correspond to logical consequences of the "**leaky gut syndrome**". Several interesting case studies concerning elderly patients have been covered by medical journals. They will be given as illustrations of several pathologies linked to cœliac disease.

The treatment of cœliac disease consists in excluding all gluten containing cereals from the patient's diet, i.e. ***wheat***, ***rye*** and ***barley***. ***Oat*** seems to be tolerated, even though it contains gluten, because this type of gluten doesn't provide something similar to gliadin.

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