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PREVALENCE OF OSTEOPENIA AND OSTEOPOROSIS AT THE TIME OF DIAGNOSIS IN A SERIES OF ADULT CELIAC PATIENTS

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Background and aim: A low bone mass is a well-recognized manifestation of celiac disease (CD). It can determine osteopenia or osteoporosis, and rarely pathological fractures. A bone damage can be present even in the absence of classical abdominal symptoms and also as only sign of disease. In literature, the prevalence of osteoporosis in CD is about 35%, but these data generally do not refer to the time of diagnosis, but to the follow-up.
We have evaluated the prevalence of osteopenia and osteoporosis at diagnosis, in a series of adult celiac patients divided in different age groups.
Material and methods: As a first step we have excluded celiac patients diagnosed at paediatric age and those with presumed secondary causes of low bone mass, such as renal failure, cirrhosis, adrenal diseases, Down's syndrome, multiple sclerosis, neoplasms etc. Then we have evaluated 219 consecutive adult celiac patients (18–72 age ranged; F 186, 85%). Bone mineral density (BMD) was assessed by dual-energy x-ray absorptiometry in lumbar spine and femoral neck. A T-score of <-1 was considered as osteopenic and T-score <-2.5 at any of these sites was considered osteoporosis. Patients have been divided in three age class (15–30, 31–50 and >50 yrs) to compare T-score results.
Results: At diagnosis of CD, 32% of celiacs were osteopenic and 19% osteoporotic. A low bone mass was present in 38% of 15–30 group, 50% of 31–50 yrs and 73% of >50 yrs (>50 yrs vs other groups, $p<0.02$). No patient was osteoporotic in 18–30 age class. Osteoporosis was more frequent in women in post-menopause than in pre-menopause (58% vs 13%, $p<0.0001$), regardless to Marsh grade. No significant differences were found among age groups with respect to osteopenia. Finally 50% of males (all >50 years) were osteoporic and 19% osteopenic at diagnosis of CD.
Conclusions: Our study confirms that women in post-menopause have a higher bone damage, at diagnosis of CD. Our data suggest to perform a bone densitometry at diagnosis of CD in males >30 years old and in females of any age, especially in post-menopause condition where also other mechanisms can be involved. The screening of osteopenia/osteoporosis is pivotal in CD where a low bone mass may be the only sign of undiagnosed disease. The usefulness of screening osteoporotic patients systematically for CD is instead still an open question.

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MUCOSAL DUODENAL TISSUE FROM GLUTEN-SENSITIVE PATIENTS DO NOT HAVE INCREASED EXPRESSION OF IgA B-CELL SWITCH MARKERS

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Background and aim: Gluten-sensitive individuals (GS) cannot tolerate gluten and may develop gastrointestinal symptoms similar to those in celiac disease (CD), but the overall clinical picture is generally less severe and is not accompanied by the presence of tissue transglutaminase autoantibodies (tTG) or autoimmune co-morbidities. We have previously shown that higher numbers of recently activated B cells and IgA-isotype switched plasma cells could be detected in the active CD intestinal mucosa, suggesting in loco gliadin-induced B-cell activation and switch into IgA-producing plasma cells. Aim was to investigate the involvement of mucosal B cell activation in the Gluten Sensitive patients by measuring of Activation Induced Cytidine Deaminase (AICD) gene expression, and I-alpha germline & C-alpha mature transcripts that are signal of B cell differentiation.
Material and methods: B-cell activation and differentiation into IgA-secreting plasma cells were assessed in the intestinal mucosa from 10 HC, 16 active CD- and 13 GS-patients by measuring of (AICD) gene expression and of Iα germline and Cα mature transcripts by real-time RT-PCR of bioptic cDNA.
Results: Compared to HC, CD showed significantly higher expression of mucosal B-cell activation marker AICD ($p=0.004$), and mucosal IgA isotype switch markers Iα ($p=0.021$) and Cα ($p=0.003$). In contrast, GS patients had mucosal B-cell activation and IgA isotype switch markers similar to values detected in HC.
Conclusions: These findings support and extend the idea that, the two gluten-associated disorders, CD and GS, are different clinical entities where GS is a condition prevalently associated with gluten-induced activation of the innate, rather than adaptive, immune response.

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INTRA-DUODENAL RELEASE OF A BITTER COMPOUND DECREASES CALORIC INTAKE IN HEALTHY VOLUNTEERS

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Background and aim: α-gustducin and bitter taste receptors (T2R) are expressed both in the oral cavity and in the gastrointestinal (GI) tract. Experimental data showed that bitter tastants induce the release of gut hormones from enteroendocrine cells in the gut, suggesting a possible role of bitter taste receptors in the control of food intake and GI functions. We aimed to test the effects of a bitter taste receptor agonist on food intake and GI feelings.
Material and methods: We enrolled 19 healthy subjects (9 males, age 27±7, BMI 24±6) in a double-blind placebo controlled study. Each subject randomly received an acid-resistant capsule containing placebo or 18 mg of quinine HCl. 60 minutes after capsule administration, the subjects underwent to an ad libitum test, until the maximum satiation. Meal test was composed by white bread, cheese and meat cream (89 kcal/portion: 50% carbohydrate, 31% fat, 19% protein). Caloric intake, meal duration and satiation levels, scored on a Visual Analogue Scale (VAS) were calculated at the end of the meal test. A questionnaire assessing GI sensations (bloating, fullness, nausea, epigastric discomfort and hunger) was administered before and at the end of the test. Data (mean ± SD) were compared by using paired t test.
Results: No oral bitter sensation or side effects was observed both with quinine HCland placebo. No significant differences in terms of GI sensations and hunger feelings were observed between the two sessions of the study. The amount of calories ingested was significantly lower when subjects received quinine HCl than placebo (564±262 vs 667±278 kcal; $p=0.02$). Conversely, quinine HCl did not affect the meal duration (14.4±4.2 vs 16.6±4.6 min; p=NS) and the satiationintensity (82 vs 82 mm; p=NS).
Conclusions: The intra-duodenal release of a bitter compound significantly decreases caloric intake in an ad libitum test meal without affecting GI sensations and hunger feeling. As the bitter compound does not influence meal duration, we hypothesize that quinine HCl decreases the caloric intake by affecting the rate of meal portions consumption. Evaluation of gut hormones kinetics and studies with other bitter taste receptor agonist are needed to establish the role of gastrointestinal bitter taste receptor in the control of food intake.

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EFFECT OF BARIATRIC SURGERY ON SERUM LEVELS OF GASTROINTESTINAL HORMONES IN OBESE NAFLD PATIENTS

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Background and aim: The gut releases a number of humoural peptides that signal to the brain to control food intake and to control energy homeostasis, such as peptide YY (PYY), glucagon-like peptide (GLP)-1, GLP-2, ghrelin (GHR), orexin (ORE) and cholecystokinin (CCK). Over the last years,