High fat consumption in children with celiac disease

P. Ferrara[1], M. Cicala[2], E. Tiberi[1], C. Spadaccio[2], L. Marcella[2], A. Gatto[1], P. Calzolari[3], G. Castellucci[4]

(1) Department of Pediatric Sciences, Università Cattolica Sacro Cuore, Rome, Italy ; (2) Campus Bio-Medico University, Rome, Italy ; (3) Pediatric of Public Health, Assisi, Italy ; (4) Department of Pediatric, Foligno-Perugia, Italy.

Abstract

Aim : **The purpose of this study was to estimate the caloric intake and fat consumption in children with celiac disease (CD) following a gluten-free diet (GFD).**

Patients and methods : **This study enrolled 100 subjects, including 50 children with CD on a gluten-free diet and a control group of 50 healthy children. Statistical analysis to compare groups was performed using one-way ANOVA.**

Results : **A significant increase in fat consumption was observed in children with CD as compared to healthy children. The daily fat intake was 72.5 ± 37.2 g per 100 g of food in the CD group and 52.9 ± 35.4 g per 100 g of food in the control group ($p < 0.008$). A significant difference in fat intake was found between celiac and healthy females (10.21 ± 3.15 g/100 g in the celiac group vs 7.46 ± 2.91 g/100 g in the control group), $p = 0.004$.**

Conclusions : **This study describes a significantly higher fat consumption in patients with CD on GFD as compared to controls. This increase was more pronounced in females and during the puberal age. Based on these interesting preliminary results we estimate that further investigations are necessary, such as a randomized multicentre study on the long-term effects of GFD with particular attention to the imbalance in daily fat intake.** (Acta gastroenterol. belg., **2009**, 72, **296-300**).

Key words : Celiac disease, fat, metabolism.

Introduction

Celiac disease (CD) is one of the most frequent chronic diseases in childhood with a prevalence of approximately 1% in Caucasian populations (1). CD is caused by an inappropriate T-cell-mediated immune response to the ingestion of cereal proteins in genetically susceptible individuals. It is a multifactorial disease that may depend on genetic, immunological, and environmental factors.

In CD patients, gliadin (the principal wheat protein) is presented to sensitised T cells in conjunction with the HLA-DQ2 or HLA-DQ8 antigen, leading to the production of cytokines. These cytokines cause tissue damage within the mucosa and activate plasma cells to produce antibodies to gliadin, tissue transglutaminase and endomysium (2).

Once the diagnosis of CD is confirmed, patients need to be placed on a strict life-long gluten-free diet (GFD), which represents the only treatment currently available to prevent the recurrence of symptoms and other potential consequences.

Nowadays gluten-free foods are widely diffused thanks to the increasing number of industries producing a wide range of nutrients free of gluten and derivatives. Analysis of ingredients and contents of available GFD products revealed that they are often enriched with fats in order to make them more palatable. As a result, while this kind of diet allows celiac patients to avoid consumption of immunoreactive antigens, on the other hand it determines a significant increase in daily fat intake that, in turn, could expose patients to further metabolic and psychological complications. For this reason we decided to retrospectively evaluate the actual caloric and fat content of a GFD in CD children, and to compare it to a similar *at libitum* diet in healthy children.

Materials and methods

In this study we retrospectively evaluated a total of 100 children, 50 with CD (18 males and 32 females ; age range 6 to 16 yrs ; mean age 10.68 years) in the study group, and 50 healthy children (17 males and 33 females ; age range 6 to 17 yrs ; mean age 10.74 years) in the control group.

In the study group inclusion criteria were : diagnosis of CD formulated according to the European Society of Paediatric Gastroenterology and Nutrition (ESPGAN) criteria ; regular consumption of a GFD for at least one year ; normal findings in mucosal biopsy and normal serological and biochemical markers (3). The control group consisted of healthy subjects under a free diet.

Two interviewers, blind to the actual clinical conditions of the subjects, administered an anonymous preformed and validated questionnaire to both parents. The questionnaire was structured in order to obtain precise and unequivocal data concerning age, sex, habits of food consumption during the day and characteristics of foods assumed including type, name, manufacturer, quantity, weekly rate of consumption and nutritional aspects including caloric intake per 100 grams of products and fats percentage (Fig. 1). In order to avoid bias, interviews were conducted separately for parents and children, in respect of privacy laws.

As the data analysis has been performed by a retrospective method there was no need for Institutional Review Board (IRB) approval.

Correspondence to : Pietro Ferrara, Department of Pediatric Sciences, Università Cattolica Sacro Cuore, Largo A. Gemelli 8, 00168 Rome, Italy.
E-mail : pferrara@rm.unicatt.it

Submission date : 13/08/2008
Acceptance date : 02/06/2009

QUESTIONNAIRE ON HABITS OF FOOD CONSUMPTION

SEX _____ AGE_____

TIME _____ (i.e. breakfast, snack1, lunch…)

Type of food	Name/Manufacturer	Quantity (in g)	Week rate consumption	Fat per 100 g	Kcal per 100 g

Fig. 1. — Questionnaire form administered to children for the food intake at breakfast, snack 1, lunch, snack 2, dinner and after 8

Statistical analysis

Data were processed using SPSS (Statistical Package for Social Sciences) release 13.0 for Windows (SPSS, Chicago, IL). Data are presented as mean ± SD. One-way ANOVA was performed to compare groups with different treatments, followed by multiple pair wise comparison procedure (Tukey test). Consumptions of normality were checked and met. Holm-Sidak method was used to increase the power of the analysis. Pearson's product-moment *r* coefficient was calculated to evaluate correlations. Significance was considered at the 0.05 level.

Results

Total amount of food, type of food and temporal distribution of meals during the day were compared between the two groups.

We found a statistically significant increase in the total daily fat intake in the CD group as compared to the control group, with 72.5 ± 37.2 g per 100 g of food vs 52.9 ± 35.4 g per 100 g of food, respectively ($p < 0.008$). No statistical difference was observed in the mean caloric intake of CD and control group, 2303.4 ± 464.5 Kcal/die vs 2181.5 ± 338.6 Kcal/die, respectively (Table 1).

Figure 2a shows data concerning caloric intake from each single meal and comparison of values between CD and control group. Caloric intakes of lunch and dinner were not statistically different in the 2 groups, whereas they were significantly higher in the morning and afternoon snacks of CD children as compared to controls (456.36 ± 78.8 Kcal and 434.33 ± 98.32 Kcal in CD vs 341.38 ± 71.08 and 373.12 ± 72.53 in the control group ; $p < 0.001$). On the other hand, significantly higher caloric intakes were observed in healthy subjects as compared to celiacs at breakfast and after eight snacks ($p < 0.001$ and $p < 0.05$, respectively). Data regarding fat consumption per each single meal are shown in Figure 2b.

Distribution of fat and caloric intake between males and females was also considered, with the evaluation of mean caloric and fat intake per single meal. No differences were found within the two genders of each groups, confirming homogeneity of data and property of sampling technique. On the other hand, when the CD group was compared to the control group a significant difference was observed between celiac females and healthy females in both caloric (371.49 ± 97.51 Kcal/die vs 355.77 ± 68.82 Kcal/die, respectively ; $p = 0.003$) and fat (10.21 ± 3.15 g per 100 g of food vs 7.46 ± 2.91 g per 100 g of food, respectively ; $p = 0.004$) intake (Table 1).

Moreover, although the caloric and fat intake in CD children was significantly increased, the analysis of the total amount of food per day showed lower values in the CD group as compared to the control group.

Discussion

CD can be considered as a gastrointestinal disorder characterized by chronic inflammation, leading to injury of the mucosal lining of the small intestine. The mucosal damage and subsequent malabsorption of nutrients results in a variety of sequelae and complications.

Table 1. — **Caloric and fat intake**

	Children with CD	*Healthy children*	*p value*
Mean caloric intake/die (Kcal/die)	2303.4	2181.5	> 0.05
Fat intake/die (g per 100 g of food)	72.5	52.9	*< 0.008*
Mean daily caloric consumption per single meal in females (Kcal/die)	371.49	355.77	*< 0.003*
Mean daily fat consumption per single meal in females (g per 100 g of food)	10.21	7.46	*< 0.004*
Mean daily caloric consumption per single meal in males (Kcal/die)	363.33	348.18	> 0.05
Mean daily fat consumption per single meal in males (g per 100 g of food)	8.85	7.26	> 0.05

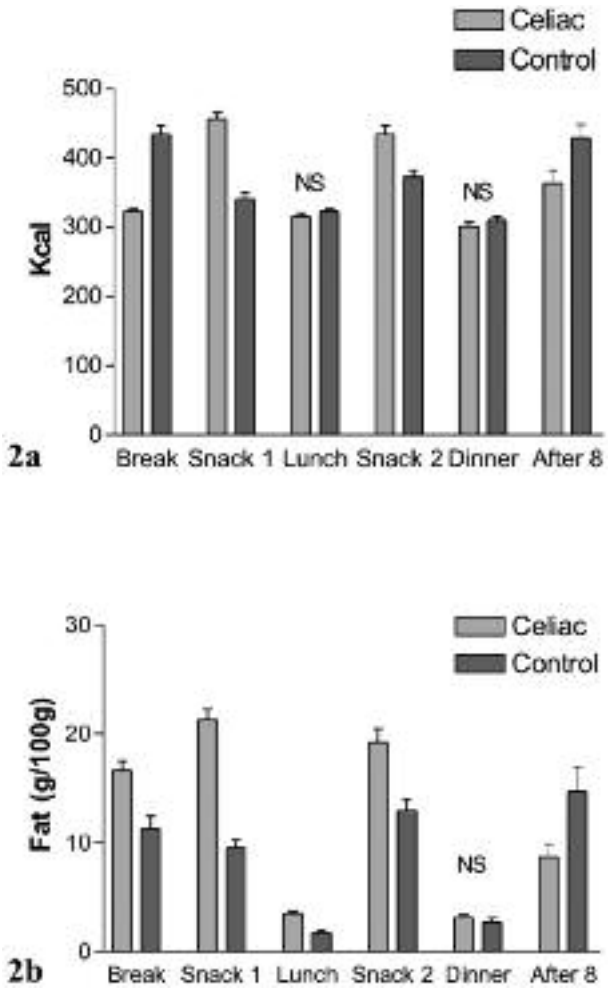

Fig. 2a, b. — Kcalories (2a) and fat, expressed as g of fat in 100 g of food (2b), consumption per single meal in celiac and control group.

The cornerstone of treatment in CD is the strict observance of a GFD (4).

The elimination of gluten from the childrens' diet usually leads to a dramatic and rapid response, but it may sometimes be delayed. Weight gain and relief of emotional symptoms in the parents and children usually occur before cessation of diarrhoea and other signs of improvement.

As a secondary deficiency in the disaccharidases of the small intestine occurs in approximately 65% of celiac patients with villous atrophy, together with a specific lactase deficiency, it is recommended that dairy products be avoided only initially, since many patients with CD find intolerance to these agents disappear when the condition comes under control with a GFD (5,6).

This GFD represents so far the only efficient therapy allowing both reduction in bowel inflammation and prevention of celiac clinical sequelae, such as abnormal intestinal permeability and mucosal damage (7), hepatic disease -which is strictly connected to malabsorption and intestinal permeability (8), poor bone mineralisation (9), growth retardation (10), and cutaneous manifestations (11).

Thanks to the increasing number of industries producing gluten-free food, nowadays the availability of almost all kinds of food can significantly reduce the limitations in lifestyle, which are uneasily accepted by children.

Given both the pivotal role of GFD in the therapeutical management of CD and the importance of an adequate variety of foods in childrens' diet together (mainly due to the social aspects related to meals), we proposed an accurate evaluation of GFD characteristics and their potential impact on both clinical status and lifestyle. Analysis of ingredients and contents of GFD revealed that gluten-free foods are enriched with fats in order to make them more palatable. As a result, while this kind of diet allows celiac patients to avoid consumption of immunoreactive antigens, it also determines a significantly greater fat intake. As a result, in the CD group retrospectively evaluated in this study, we observed a statistically significant increase in the total daily fat intake as compared to controls, despite a comparable caloric intake in the two groups.

Therefore, we also decided to evaluate separately the caloric intake provided by each single meal. This allowed us to overtake potential biases concerning

differences in the actual intake and eating habits within patients, and to obtain a clear understanding of the fat/caloric intake associated to the different daily meals. As a result, potential speculations on the possibility to modify nutrient components of some of the gluten free food commercially available could be proposed. In fact the greatest fat and caloric consumption for celiac children lies behind snack time (Fig. 2). Therefore, we suggest that this findings could represent a valid clue to improve nutrient composition of gluten free snacks. We believe that the increase of fat consumption is related to both more fat in daily foods and in manufactory products. The first observation is been reported in previous studies, where was described in CD patients a higher amount of fats intake related to the wrong choice of natural foods. CD patients in fact ate less foods containing carbohydrates such as bread, pizza or pasta without an increase of alimentary items of the same group (rice, corn and potatoes) but with an augment of intake of foods such as meat, eggs and legumes (12,13). Mariani *et al.* highlighted also the major intake vs the control subjects of the snacks with a high content of lipids like in our study (12).

The data presented in this study trigger concerns regarding the safety of a long term gluten free diet and its potential impact on clinical conditions, especially in children. A prolonged increase in dietary fats provides an additional source of cholesterol and saturated fatty acids that could deeply affect lipid metabolism and endocrine system functioning. In CD most clinical manifestations caused by chronic bowel inflammation can be reversed by a GFD and cease in adult age. However, changes in the lipid profile caused by an higher fat intake could result in a wide spectrum of clinical manifestations that could result in other long-term consequences in the adult age.

CD has been associated with the development of insulin-resistance, diabetes mellitus, hypogonadism and growth hormone deficiency (14). The latter has a relevant importance in determining growth retardation and alterations in body composition (15-21). GFD has been shown to improve both growth and distribution of body composition. Interestingly, two studies evaluating the effects of 4 years GFD on body composition, revealed a significant increase in body weight, bone mass, body mass index, and fat mass, not accompanied by a parallel increase in lean-tissue mass or muscle mass (22,23). In our study we show a significant increase in fat consumption in celiac patients treated with GFD, which was more pronounced in females than in males. The mean fat intake was significantly higher in the celiac females as compared to healthy females, while a non statistically significant difference was observed between celiac and healthy males. Given the profound changes occurring in females at the moment of puberal growth in terms of fat distribution and lipid profile, our results suggest that an accurate evaluation of the possible long term risks of hyperlipidemia, abnormalities of insulin-dependent metabolisms, obesity and the related psychological problems should be carefully considered.

The study is been conducted on Italian children with their food habits so our results might not be applied to other children in Europe, in particular where the food habits are different, but considering our results the advice is to put more attention to the fat intake in all children on GFD.

In conclusion in this retrospective study we demonstrate a significantly higher fat consumption in 50 celiac patient under GFD as compared to 50 age-matched healthy subjects. This increase was more pronounced in females than in males. We estimate that this difference in the results between males and females can be explained by the variety of manufactory products and the dietary habits of the children in fact with this study we want to highlight the possibility to improve nutrient composition of gluten free food in particular in the gluten free snacks and the importance to evaluate not only the compliance with the GFD but also the dietary habits to identify an eventual nutritional imbalance of the diet itself. We also observed an higher caloric intake at the morning and afternoon snacks for celiacs. This study has revealed the importance of snacks in determining the actual fat intake for celiacs, which hopefully result in introducing better dietary guidelines for celiac patients. Despite the universally recognised importance of GFD as unique therapeutic tool in CD, the potential long term effects of this therapy after both the local and systemic inflammatory damage has been reversed, should be considered carefully especially in terms of potential metabolism abnormalities. Based on these interesting preliminary results we estimate that further investigations, such as a randomized multicentre study on the long-term effects of GFD with particular attention to the imbalance in daily fat intake, are to be conducted.

References

1. HILL P.G., MC MILLAN S.A. Anti-tissue transglutaminase antibodies and their role in the investigation of coeliac disease. Ann Clin Biochem., 2006, **43** : 105-117.
2. DUGGAN J.M. Coeliac disease : the great imitator. *Med. J. Aust.*, 2004, **180** : 524-526.
3. WALKER-SMITH J.A., GUANDALINI S., SCHMITZ J. *et al.* Revised criteria for diagnosis of celiac disease. (Report of The Working Group of the European Society of Paediatric Gastroenterology and Nutrition). *Arch. Dis. Child*, 1990, **65** : 909-911.
4. FARRELL R.J., KELLY C.P. Celiac sprue. *N. Engl. J. Med.*, 2002, **346** : 180-187.
5. DUNCAN A., PARK R.P., LEE F.D., RUSSELL R.I. A retrospective assessment of the clinical value of jejunal disaccharidase analysis. *Scand. J. Gastroenterol.*, 1994, **29** : 1111-1116.
6. BODE S., GUDMAND-HOYER E. Incidence and clinical significance of lactose malabsorption in adult CD. *Scand. J. Gastroenterol.*, 1988, **23** : 484-488.
7. DUERKSEN D.R., WILHELM-BOYLES C., PARRY D.M. Intestinal permeability in long-term follow-up of patients with CD on a gluten-free diet. *Dig. Dis. Sci.*, 2005, **50** : 785-790.
8. KAUKINEN K., HALME L., COLLIN P., FÄRKKILÄ M., MÄKI M., VEHMANEN P., PARTANEN J., HÖCKERSTEDT K. CD in patients with severe liver disease : gluten-free diet may reverse hepatic failure. *Gastroenterology*, 2002, **122** : 881-888.

9. TAU C., MAUTALEN C., DE ROSA S., ROCA A., VALENZUELA X. Bone mineral density in children with CD. Effect of a Gluten-free diet. *Eur. J. Clin. Nutr.*, 2006, **60** : 358-363.
10. BOERSMA B., HOUWEN R.H., BLUM W.F., VAN DOORN J., WIT J.M. Catch-up growth and endocrine changes in childhood CD. Endocrine changes during catch-up growth. *Horm. Res.*, 2002, **58** : 57-65.
11. FRODIN T., GOTTHARD R., HED J., MOLIN L., NORRBY K., WALAN A. Gluten-free diet for dermatitis herpetiformis : the long-term effect on cutaneous, immunological and jejunal manifestations. *Acta Derm. Venereol.*, 1981, **61** : 405-411.
12. MARIANI P., VITI M.G., MONTUORI M., LA VECCHIA A., CIPOLLETTA E., CALVANI L., BONAMICO M. The gluten-free diet : a nutritional risk factor for adolescents with celiac disease ? *J. Ped. Gastroenterol. Nutr.*, 1998, **27** : 519-523.
13. BARDELLA M.T., FREDELLA C., PRAMPOLINI L., MOLTENI N., GIUNTA A.M., BIANCHI P.A. Body composition and dietary intakes in adult celiac disease patients consuming a strict gluten-free diet. *Am. J. Clin. Nutr.*, 2000, **72** : 937-939.
14. CICLITIRA P.J., MOODIE S.J. Coeliac Disease. *Best Practice & Research Clinical Gastroenterology*, 2003, **17** : 181-195.
15. GREENFIELD J.R., SAMARAS K. Evaluation of pituitary function in the fatigued patient : a review of 59 cases. *Eur. J. Endocrinol.*, 2006, **154** : 147-157.
16. SALARDI S., CACCIARI E., VOLTA U., SANTONI R., RAGNI L., ELLERI D., CICOGNANI A., VAIRA D. Growth and adult height in atypical coeliac patients, with or without growth hormone deficiency. *J. Pediatr. Endocrinol. Metab.*, 2005, **18** : 769-775.
17. POZO J., MARTOS-MORENO G.A., BARRIOS V., ARGENTE J. The IGF system in childhood : physiology and clinical implications. *J. Endocrinol. Invest.*, 2005, **28** : 38-42.
18. BOZZOLA M., GIOVENALE D., BOZZOLA E., MEAZZA C., MARTINETTI M., TINELLI C., CORAZZA G.R. Growth hormone deficiency and coeliac disease : an unusual association ? *Clin. Endocrinol. (Oxf.)*, 2005, **62** : 372-375.
19. CAPPA M., LOCHE S. Evaluation of growth disorders in the paediatric clinic. *J. Endocrinol. Invest.*, 2003, **26** : 54-63.
20. LECORNU M., DAVID L., FRANCOIS R. Low serum somatomedin activity in celiac disease. A misleading aspect in growth failure from asymptomatic celiac disease. *Helv. Paediatr. Acta*, 1978, **33** : 509-516.
21. VERKASALO M., KUITUNEN P., LEISTI S., PERHEENTUPA J. Growth failure from symptomless celiac disease. A study of 14 patients. *Helv. Paediatr. Acta*, 1978, **33** : 489-495.
22. CARBONE M.C., PITZALIS G., FERRI M., NENNA R., THANASI E., ANDREOLI A., DE LORENZO A., BONAMICO M. Body composition in coeliac disease adolescents on a gluten-free diet : a longitudinal study. *Acta Diabetol.*, 2003, **40** : S171-173.
23. SMECUOL E., GONZALEZ D., MAUTALEN C., SICCARDI A., CATALDI M., NIVELONI S., MAZURE R., VAZQUEZ H., PEDREIRA S., SOIFER G., BOERR L.A., MAURIÑO E., BAI J.C. Longitudinal study on the effect of treatment on body composition and anthropometry of celiac disease patients. *Am. J. Gastroenterol.*, 1997, **92** : 639-643.