

COELIAC UK'S RESEARCH CONFERENCE 2017

The gluten free diet; classic coeliac disease and more

Friday, 24 March 2017



THE GLUTEN FREE DIET; CLASSIC COELIAC DISEASE AND MORE

ROYAL COLLEGE OF PHYSICIANS

11 St Andrew's Place, Regent's Park, London, NW1 4LE

Chaired by Prof David Sanders, Royal Hallamshire Hospital and University of Sheffield

10:00 **Arrival and networking – refreshments**

10:30 **Coeliac UK welcome – Chief Executive Sarah Sleet**

Management of coeliac disease:

10:35 **Current challenges in the management of coeliac disease**

Dr Jeremy Woodward

11:00 **Gluten immunogenic peptides in faeces and urine; experience from a paediatric clinic**

Dr Alfonso Rodriguez-Herrera

11:25 **Therapeutic advances in coeliac disease**

Dr Daniel Leffler

11:50 **The gut microbiome composition and dynamics in coeliac disease**

Dr Yolanda Sanz

12:15 **Lunch and poster exhibition**



The gluten free diet beyond the gut:

- 13:30 **Decoding the genetic background of coeliac disease**
Dr Iris Jonkers
- 13:55 **Coeliac disease and Type 1 diabetes**
Dr Matthew Kurien
- 14:20 **Gluten and the brain**
Prof Marios Hadjivassiliou
- 14:45 **Break – refreshments**

The gluten free diet and FODMAPs:

- 15:15 **Gluten, wheat and FODMAPs: dietary triggers of irritable bowel syndrome and non coeliac gluten sensitivity**
Dr Umberto Volta
- 15:40 **Dietetic interventions in non responsive coeliac disease**
Nick Trott
- 16:05 **The bidirectional relationship between IBD and self reported non coeliac gluten sensitivity**
Dr Imran Aziz
- 16:30 **Chair's closing remarks and presentation of poster prize**
- 16:35 **CLOSE**

This event has been kindly sponsored by BFree, Schär, Thermo Fisher and Warburtons



Sarah Sleet

Chief Executive of Coeliac UK

Welcome

Welcome to the Coeliac UK Research Conference, 2017. We hope this conference will contribute to your ongoing professional development and provide an opportunity to share experiences.

Today's programme reflects upon developments in the management of coeliac disease, research that supports or directly underpins the charity's strategic aims in this area. We also extend our focus to the use of the gluten free diet in other gluten related diseases and beyond the gut.

As part of our commitment to supporting research we aim to build a critical mass for future research and also encourage and inspire a new generation of researchers focusing on coeliac disease. We have joined forces with the Medical Research Council to offer jointly funded

Fellowships. We want to encourage more high quality applications and so please do visit the MRC website (www.mrc.ac.uk) to see if this is of interest to you. The next round for applications opens in July.

This year we are reviewing our Research Strategy and one key aim is to create a priority setting partnership to identify the top ten research priorities in coeliac disease, in time for our 50th birthday celebrations in early 2018. We need the support of patients and those who care for them, to make this a success - we hope we can count on your support!

Thank you,

A handwritten signature in black ink that reads "Sarah Sleet". The signature is written in a cursive, flowing style.

More information about the priority setting partnership is available on p64 of this brochure.



Prof David Sanders

Chair of Coeliac UK's
Research Conference

Biography

Professor David S Sanders is a consultant gastroenterologist and a professor of gastroenterology at the Royal Hallamshire Hospital & the University of Sheffield. He has published >300 peer reviewed papers (H-score 50). He is internationally recognised for his work in coeliac disease, gastrostomy feeding and small bowel endoscopy. His other research interests include pancreatic exocrine insufficiency, irritable bowel syndrome and gastrointestinal bleeding.

In 2010 he received a European Rising Star Award and a Cuthbertson Medal in 2011 (UK Nutrition Society). He has been selected to receive the most prestigious award of the Swedish Gastroenterology Society in 2017 (Bengt Ihre Award). His clinical work with patients who have coeliac disease has resulted in him being awarded the Coeliac UK Healthcare Professional of the Year Award (2010) & the inaugural Complete Nutrition Coeliac Health Care Professional Award (2013). He is fortunate to work as part of the

Sheffield Gastroenterology Team which has been recognised for their standards of care. The Small Bowel Endoscopy Service won one of the inaugural British Society of Gastroenterology National GI Care awards (2011) & the Medipex award (2013). In 2012 the Percutaneous Endoscopic Gastrostomy team won both Health Service Journal primary care and integrated clinical care awards. In 2014 the Hallamshire Gastroenterology team, won one of the SAGE (Shire Awards for Gastrointestinal Excellence) awards for their primary care and GI bleed unit services. Professor Sanders has chaired both the British Society of Gastroenterology (BSG) Small Bowel and Nutrition Section (2006-2012) & the BSG Audit Committee (2010-2013).

He is the current Chair of the Coeliac UK Health Advisory Council, BSG Council Member and President of the International Society for the Study of Coeliac Disease (ISSCD).

<http://www.profdavidsanders.co.uk/>



Dr Jeremy Woodward

Consultant Gastroenterologist,
Addenbrooke's Hospital,
Cambridge, UK

Biography

Dr Jeremy Woodward trained in Cambridge and London and completed a PhD in immunology in Birmingham prior to his appointment as a consultant gastroenterologist at Addenbrooke's Hospital, Cambridge in 2002. He is the lead clinician for nutrition and chairs the Trust nutrition steering group. He provides specialist clinics in coeliac disease, immunodeficiency related gastrointestinal disease, nutrition, ERCP and pancreatic disease, runs a regional

video capsule enteroscopy service and is a lead clinician in intestinal failure and intestinal transplantation. He currently chairs the Anglia Nutrition Network, and is an enthusiastic teacher and lecturer in nutritional matters. His research interests include intestinal failure, transplantation and coeliac disease.



CURRENT CHALLENGES IN THE MANAGEMENT OF COELIAC DISEASE

Abstract

Guidelines on the diagnosis and management of coeliac disease have been issued recently by the British Society of Gastroenterology and the National Institute of Health and Care Excellence (NICE). Despite valiant attempts to produce evidence based guidance, the existing evidence base is weak and there is little on which to support firm recommendations.

Key deficiencies remain in a number of fundamental areas. Our current tests for the diagnosis of coeliac disease do not provide the degree of certainty that was once believed and misdiagnosis is common. The significance of sub-threshold diagnostic tests is unclear, and with wider acceptance of non-coeliac gluten sensitivity as an entity it

is becoming apparent that the current paradigm for the definition of coeliac disease is itself problematic.

The management of coeliac disease after diagnosis is flawed by the lack of appropriate outcomes or endpoints which in turn leads to lack of uniformity in process and quality of follow up as well as the current recommendations for indefinite annual review which are challenging to implement.

Whilst new developments may lead us to more robust criteria for diagnosis and follow up, there is a clear need for generating better outcome measures that allow us to define and refine pathways for diagnosis and care.



Dr Alfonso Rodriguez-Herrera

Consultant Paediatric
Gastroenterologist, Instituto
Hispalense de Pediatría,
Seville, Spain

Biography

Alfonso Rodriguez-Herrera is a medical specialist in paediatrics with special interest in paediatric gastroenterology and nutrition. He gained his PhD from the University of Seville with a thesis on coeliac disease.

Currently he is head of gastroenterology of IHP, a large medical company based in southern Spain. He is engaged in teaching as Associate Professor at Pablo Olavide University. He is a member of the specialist register of the Irish Medical Council (Paediatrics) and full member of ESPGHAN (European Society for Paediatric Gastroenterology, Hepatology and Nutrition). His main research lines are biomarkers of dietary compliance (allergy, coeliac disease and obesity), technical aids for gastroenterology and nutrition, and development of new ingredients for child nutrition.

As principal investigator he has led research and trials on obesity prevention, virtual reality applied to medical training and models of intestinal microbiota in obesity. He is author or co-author of different book chapters and papers connected to his area of expertise. In close cooperation with the Faculty of Pharmacy, Seville he recently participated in papers; "Monitoring of gluten free diet compliance in celiac patients by assessment of gliadin 33-mer equivalent epitopes in feces" and "Detection of gluten immunogenic peptides in the urine of patients with coeliac disease reveals transgressions in the gluten free diet and incomplete mucosal healing".

He has been granted with several awards in the area of paediatric gastroenterology. He is one of the authors of the patent P201400569 for gluten free diet monitoring.

MONITORING ADHERENCE TO THE GLUTEN FREE DIET WITH GLUTEN IMMUNOGENIC PEPTIDES IN FAECES AND URINE - EXPERIENCE FROM A PAEDIATRIC CLINIC

Abstract

There is a general consensus that strict gluten free diet (GFD) adherence in coeliac patients leads to full clinical and histological remission. It is linked to an improvement in quality of life and reduced long term complications. Persistent gluten exposure is usually unintentional. Exposure may occur no matter how careful a patient is, due to cross contamination or simple lack of knowledge regarding the diet. Serum markers for coeliac disease (CD) play an important role in CD management (mostly tTGA); however, the evidence suggests that it is not sensitive enough to detect occasional significant dietary transgressions that impede mucosa healing.

The resistance of gluten peptides to gastrointestinal digestion, in particular gluten immunogenic peptides (GIP) related to the immunotoxic 33-mer peptide, ensures that an important part of the ingested gluten is eliminated in faeces. A proportional fraction of the GIP absorbed in the gastrointestinal tract makes it to the circulation and is excreted in urine. Detection of GIP in faeces and urine has been proposed as a new non invasive biomarker to detect

gluten intake and verify GFD compliance in patients with CD.

In a clinical setting the response rate to a GFD was evaluated by dietary questionnaire, coeliac serology, and correlations between faecal GIP and traditional methods of monitoring the GFD have been investigated. The majority of children with CD, between zero and three years of age showed faeces negative for GIP, revealing good compliance. The proportion of patients with faeces positive for GIP increased in children between four and 12 years of age. No association was detected between faecal GIP and dietary questionnaire or anti-tTG antibodies.

GIP analysis in samples of faeces and urine could resolve some scientific and clinical problems in CD management such as detection of inadvertent lapses after appearance of acute symptoms; non compliance assessment of the GFD before onset of any anatomic damage; to prove gluten intake during CD diagnosis; examining the adherence to the GFD in the initial period after diagnosis when patients are less familiar with this diet.



Dr Daniel Leffler

Director of Research,
Celiac Center, Beth Israel
Deaconess Medical Center,
Boston, Massachusetts, USA

Biography

Daniel Leffler, MD, MS currently serves as the Director of Research at the Celiac Center at Beth Israel Deaconess Medical Center and is an Associate Professor of Medicine at Harvard Medical School. Dr Leffler currently serves as a Medical Director in the Gastrointestinal Therapeutic Area at Takeda Pharmaceuticals as well as continuing to care for patients with coeliac disease and other gastrointestinal disorders and he participates in clinical and translational research in coeliac

disease. Dr Leffler has been the recipient of a five year career development grant from the National Institute of Health as well as multiple foundation and industry sponsored grants for the advancement of digestive health. He lectures nationally and internationally on these topics and has co-authored a book with Melinda Dennis, RD: 'Real life with celiac disease. Troubleshooting and thriving gluten free' published by the American Gastroenterological Association.

THERAPEUTIC ADVANCES IN COELIAC DISEASE

Abstract

Over the past 20 years, celiac disease has evolved from a condition felt to be rare with little if any unmet medical need, to a disease known to be common and for which current therapy is suboptimal for many patients. In this talk I will review the reasons why novel therapeutic options are now felt to be valuable, a variety of agents currently under investigation and the methodology and rationale for evaluating safety and efficacy of new treatments for coeliac disease.





Prof Yolanda Sanz

Professor of Research,
Institute of Agrochemistry and
Food Technology. National
Research Council (IATA-CSIC),
Valencia, Spain

Biography

Dr Sanz has a PhD in Pharmacy; was Assistant Professor of Human Dietetics and Nutrition (University of Valencia; 2005-2011) and is currently Assistant Professor of different MSc degrees related to nutrition, functional foods and safety and coeliac disease at national and international universities.

She has been a member of the Panel of Nutrition, Dietetic Products and Allergies (NDA) of the European Food Safety Authority (EFSA) since 2009 and shall be until 2018. Within this period, she was Vice-Chair from 2012-2015.

Her scientific fields of interest are the influence of the gut microbiota and

its interactions with the diet and the genotype on metabolic and immune mediated disorders (coeliac disease, obesity and the associated disorders), and the efficacy and mechanisms of the action of probiotics. She has been leader of 28 research grants and, currently, coordinates a large European collaborative project focused on the role of the human microbiome in diet related disorders (MyNewGut; www.mynewgut.eu). She has had more than 140 publications in international peer reviewed journals, contributed to many conferences and made eight patent applications.

THE GUT MICROBIOME COMPOSITION AND DYNAMICS IN COELIAC DISEASE

Abstract

The intestinal microbiota establishes mutualistic relationships with the human host influencing multiple physiological functions, like those mediated by the immune system. In fact, many complex immune mediated diseases have been linked to changes in the composition and function of the gut microbiota and its genome (microbiome), including coeliac disease (CD). Epidemiological studies indicate that common perinatal and early postnatal factors influence both CD risk and intestinal microbiota structure. Prospective studies in healthy infants at risk of developing CD reveal that the HLA-DQ genotype, in conjunction with other environmental factors, influences the microbiota composition and could contribute to the risk of developing the disease. Furthermore, CD patients have

imbalances in the intestinal microbiota (dysbiosis), which are not fully normalized despite their adherence to a gluten-free diet. Therefore, it is hypothesized that disease can promote dysbiosis that aggravate CD pathogenesis and dysbiosis, in turn, can initiate and sustain inflammation through the expansion of pro-inflammatory pathobionts and decline of anti-inflammatory mutualistic bacteria. Studies in experimental models are also contributing to understand the role of intestinal bacteria and its interactions with a predisposed genotype in promoting CD. Advances in this area could aid in the development of microbiome informed intervention strategies that optimize the partnership between the gut microbiota and host immunity for improving CD management.



Dr Iris Jonkers

Rosalind Franklin Fellow,
Department of Genetics,
University of Groningen,
University Medical Center
Groningen, The Netherlands

Biography

Dr Jonkers' training as a molecular biologist started as a PhD student in the group of Joost Gribnau, at the Erasmus Medical Center in Rotterdam, Netherlands, followed by her postdoctoral training in the USA at Cornell University in the group of John T Lis, focusing on gene expression regulation. Now, she is applying this background to understand how genetics influences gene expression and can lead to coeliac and autoimmune disease. Her research within the genetics department of the University Medical Center, Groningen is funded by a Rosalind Franklin Fellowship.



DECODING THE GENETIC BACKGROUND OF COELIAC DISEASE

Abstract

Coeliac disease is an autoimmune disease that is partly caused by the genetic background of the patient. The main genetic risk factors are the HLA-DQ2.5 and HLA-DQ8 locus, which are necessary but not sufficient to cause coeliac disease. These genes encode a protein that facilitates the recognition of gluten peptides by immune cells, which consequently triggers the first steps in inflammation. However, large genetic studies have identified 45 additional

genetic loci that each increase the risk for coeliac disease. Still, how these loci contribute to coeliac disease pathology is not well understood. Therefore, our research is focused on elucidating the role of genetics in coeliac disease to increase understanding of the molecular underpinnings of the disease and find targets for alternative treatments for patients with the condition.



Dr Matthew Kurien

Academic Clinical Lecturer,
University of Sheffield,
Sheffield, UK

Biography

Dr Kurien has an interest in small bowel disease and clinical nutrition. He qualified in 2003 and subsequently completed a postgraduate diploma in medical education at the University of Nottingham. In 2010 he was appointed as a Bardhan Research and Education Trust (BRET) clinical research fellow under the supervision of Professor David Sanders. This culminated in an MD from the University of Sheffield in 2014 for his work in gastrostomy feeding. His current

research interests are in the area of coeliac disease, with the aim of improving detection and subsequent management. In 2016 he was the recipient of the Julie Wallace Award from the Nutrition Society, and one of four UK trainees shortlisted for 'The Royal College of Physicians Excellence in Patient Care Awards'.

COELIAC DISEASE AND TYPE I DIABETES

Abstract

The clinical overlap between coeliac disease (CD) and Type 1 diabetes (T1D) is recognised, reflecting a shared genetic background. Classic intestinal symptoms of CD may not always present in those with T1D, leading to recommendations that active case finding should be adopted in this high risk group. The mainstay of treatment for CD is a gluten free diet, however this can pose significant challenges to those with T1D who are already on restricted diets. This

presentation reviews the prevalence of CD in T1D, the genetics of the two conditions, and evaluates the challenges associated with dietary adherence. It is proposed to also look at the current screening practices that occur for CD in T1D, alongside highlighting novel insights into the pathogenic overlap between T1D and CD.





Prof Marios Hadjivassiliou

Consultant Neurologist,
Royal Hallamshire Hospital,
Sheffield, UK

Biography

Professor Marios Hadjivassiliou is a Consultant Neurologist with an interest in the neurological manifestations of systemic disease, the ataxias and peripheral neuropathies. His primary research is in the neurological manifestations of gluten related diseases, an area that he studied initially for his MD thesis and which has become the focus of his research career for the last 20 years. His pioneering research has led to the definition of previously unrecognised disease entities including gluten ataxia and gluten neuropathy. He has published extensively on the subject in high impact journals including three first author papers in the Lancet. He runs weekly

clinics for patients with neurological problems related to gluten sensitivity and a weekly ataxia clinic, receiving referrals from all over the UK and internationally. He is the director of the Sheffield Ataxia Centre, accredited by Ataxia UK as an Ataxia Centre of Excellence, one of only three in the UK, and is a founding member of the Sheffield Institute of Gluten Related Diseases (SIGReD). He is a member of the Medical and Health Advisory Boards for Ataxia UK and Coeliac UK. He is the Deputy Academic Director and Theme Lead for Neuroinflammation in the Academic Department for Neurosciences, Sheffield, UK.

GLUTEN AND THE BRAIN

Abstract

The presentation will concentrate on the latest research on the neurological manifestations of gluten related diseases. The aim will be to familiarise health professionals with the commonest neurological manifestations in an attempt to increase awareness and aid diagnosis.

Data on the frequency of neurological dysfunction (including cognitive deficits) in patients with classic coeliac disease presentation (assessed with brain imaging, neurological and neuropsychological evaluation) will be compared with patients presenting purely with neurological dysfunction.

In addition the concept of Non Coeliac Gluten Sensitivity, which in fact was first

reported in the context of neurological presentations, will be discussed, again with emphasis on the neurological manifestations and the role of serological biomarkers in the diagnosis.

Relevant aspects of the pathophysiology will be covered that may explain why gluten sensitivity is a spectrum and why we observe such diverse manifestations.





Prof Umberto Volta

Professor of Diagnostic Immunopathology, University of Bologna, Italy

Biography

Umberto Volta gained his degree in Medicine with honours, July 1976 from the University of Bologna. He became a specialist in cardiology and vascular disease in 1979 and in internal medicine in 1986. From 1976 to 1986 he was the Clinical Assistant at the Institute of 1st Medical Pathology and from 1986 to date, Professor of Diagnostic Immunopathology, University of Bologna. From 1999 to 2012 he was the Director of the Celiac Disease Centre of St. Orsola-Malpighi Hospital, Bologna.

Dr Volta's research interests include immunology and clinical aspects of coeliac disease; acute and chronic liver diseases; immunological abnormalities

and cardiovascular involvement in connective tissue diseases; organ specific and non organ specific autoimmunity in type 1 diabetes mellitus. He has 450 publications of which 162 are in peer reviewed journals. Dr Volta was President of the Scientific Board of the Italian Association for Celiac Disease (AIC) (2006-2014) and Member of AIC Scientific Board (2014 to date). He is also member of the Board of the European Society for the Study of Celiac Disease (ESSCD) from 2015 to date and vice president of the Ethical Committee of St. Orsola-Malpighi Hospital from 2013 to date.

GLUTEN, WHEAT AND FODMAPS: DIETARY TRIGGERS OF IRRITABLE BOWEL SYNDROME AND NON-COELIAC GLUTEN SENSITIVITY

Abstract

Irritable bowel syndrome (IBS) can be regarded as the prototype of all functional bowel disorders for its high prevalence worldwide and impact on patients' quality of life. Patients with IBS suffer from abdominal pain associated with bowel habit changes. In recent years growing attention has been paid to the causative role of food in IBS. Wheat is regarded as one of the most relevant IBS triggers, although which component(s) of this cereal is/are involved remain(s) unknown. Gluten, other wheat proteins, e.g. amylase-trypsin inhibitors (ATIs), and fructans (which belong to fermentable oligo-di-mono-saccharides and polyols [FODMAPs]) have been identified as possible factors for symptom generation. These dietary triggers precipitate or exacerbate symptoms in patients with IBS by evoking luminal distension / changing intestinal permeability, microbiota composition / inducing mast cell degranulation and finally by altering the gut brain axis likely via their opioid like activity. The awareness that gluten can be one of the IBS triggers has expanded the spectrum of gluten related disorders by adding a new entity referred to as non-coeliac gluten sensitivity (NCGS). NCGS has been defined as a syndrome characterised by symptoms occurring a few hours or days after the ingestion of gluten and wheat in patients testing negative for coeliac disease and wheat allergy. The pathogenesis of NCGS is still unclear, but innate immunity seems to exert a pivotal role, together with

factors similar to those mentioned for IBS (ie low grade intestinal inflammation, increased intestinal permeability and changes in intestinal microbiota). The epidemiology of NCGS is far from being established with a highly variable prevalence in the general population ranging from 0.6% to 6%. From a clinical stand point NCGS is characterised by a wide array of gastrointestinal (IBS like) and extra intestinal manifestations (mainly involving peripheral/central nervous systems, as well as skin and joint/muscle). Although no routine test has been identified so far, the combined use of serum markers of intestinal cell damage and systemic immune activation (FABP-2, AGA, LBP, sCD14 and antimicrobial antibodies) might become useful for NCGS diagnosis. The double blind, placebo controlled, crossover (DBPCC) trial using gluten and rice starch confirmed NCGS diagnosis only in a small percentage of cases, supporting the hypothesis that also ATIs and FODMAPs, contained in wheat, can have a role in generating NCGS / IBS overlapping symptoms. FODMAPs, which are also present in many foods, including vegetables, dairy products and fruits, display a relevant role in eliciting intestinal symptoms observed in both IBS and NCGS patients. A better knowledge of the relationship between IBS and NCGS is expected to promote standardisation in dietary strategies (gluten wheat free and low FODMAP) as effective measures for the management of IBS and NCGS patients.



Nick Trott

Gastroenterology Dietitian,
Royal Hallamshire Hospital,
Sheffield, UK

Biography

Nick Trott studied at the University of Wales Institute, Cardiff where he obtained an Honours degree in Nutrition and Dietetics. Post graduate training, Nick has worked in a variety of clinical areas within the NHS including Critical Care, Oncology and Haematology. Currently Nick works as a Gastroenterology Dietitian, covering the specialist coeliac clinic at the Royal Hallamshire Hospital, Sheffield. His areas of particular clinical interest include coeliac disease, gluten ataxia, non coeliac

wheat sensitivity and the low FODMAPs approach to functional bowel disorders. Nick was presented with the Complete Nutrition Coeliac Professional of the year award in 2016 and is currently completing a post graduate master's degree in clinical research.



DIETETIC INTERVENTIONS IN NON RESPONSIVE COELIAC DISEASE

Abstract

A small, but significant, proportion of patients with coeliac disease (CD) continue to have ongoing symptoms and a lack of histological improvement despite apparent adherence to a gluten free diet. Such patients are deemed to have Non Responsive Coeliac Disease (NRCD).

Medically these patients require the confirmation of a coeliac diagnosis, exclusion of other causes of symptoms and if necessary the assessment of a possible refractory disease.

The most common cited reason for NRCD is of either inadvertent or purposeful exposure to gluten. From this perspective dietetic assessment and guidance plays

a crucial role not only in diagnosis but in the treatment of NRCD, through the identification of possible sources of gluten contaminants. Additionally some patients are susceptible to even minute amounts of gluten and require a super sensitive dietary approach.

In the last few years new novel dietary treatments have been developed that may also facilitate a reduction in symptoms and improve histological recovery. These approaches are discussed with reference to the low FODMAP and gluten contamination elimination diets.



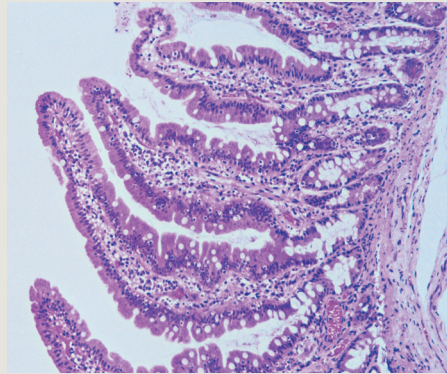


Dr Imran Aziz

Post-Doctoral Fellow in
Functional Gastrointestinal
Disorders, Sahlgrenska Academy,
Gothenburg, Sweden

Biography

Dr Imran Aziz qualified from Sheffield Medical School in 2004 and became a Consultant Gastroenterologist in 2016. During this time period he has published on irritable bowel syndrome, coeliac disease and successfully defended his postgraduate thesis on non coeliac gluten sensitivity. Imran is currently enjoying life as a post-doctoral fellow in Functional Gastrointestinal Disorders at the Sahlgrenska Academy in Gothenburg, Sweden.



THE BIDIRECTIONAL RELATIONSHIP BETWEEN IBD AND SELF REPORTED NON COELIAC GLUTEN SENSITIVITY

Abstract

Non-celiac gluten sensitivity, and the associated use of a gluten free diet (GFD), are perceived to belong to the spectrum of irritable bowel syndrome (IBS). However, recent reports suggest substantial use of a GFD in inflammatory bowel disease (IBD). We assessed the bidirectional relationship between IBD and self-reported non-celiac gluten sensitivity (SR-NCGS).

A cross-sectional questionnaire screened for SR-NCGS, and the use of a GFD, in four groups; Ulcerative colitis (n=75), Crohn's disease (n=70), IBS (n=59) and dyspeptic controls (n=109). We also assessed diagnostic outcomes for IBD in 200 patients presenting with SR-NCGS.

The prevalence of SR-NCGS was 42.4% (n=25/59) for IBS, followed by 27.6% (n=40/145) for IBD, and least amongst dyspeptic controls at 17.4% (n=19/109); p=0.015. The current use of a GFD was 11.9% (n=7/59) for IBS, 6.2% (n=9/145) for IBD, and 0.9% (1/109) for dyspeptic

controls; p=0.02. No differences were established between ulcerative colitis and Crohn's disease. However, Crohn's disease patients with SR-NCGS were significantly more likely to have stricturing disease (40.9% vs. 18.9%, p=0.046), and higher mean CDAI score (228.1 vs. 133.3, p=0.002), than those without SR-NCGS. Analysis of 200 cases presenting with SR-NCGS suggested that 98.5% (n=197) could be diet related IBS. However, 1.5% (n=3) were found to have IBD; such patients had associated alarm symptoms, and/or abnormal blood parameters, prompting colonic investigations.

SR-NCGS is not exclusive to IBS but is also associated with IBD, where its presence may be reflecting severe or stricturing disease.

THANK YOU TO OUR SPONSORS:



POSTER COMPETITION ABSTRACTS

Entries to the Coeliac UK Research Conference 2017 poster competition.

There is a £500 prize for the best abstract. The lead author may use the prize money to help pay towards

attendance at an international event to increase dissemination of their research.

The winner of our poster competition will be announced at the end of the conference.

£500

**PRIZE
FOR THE BEST
ABSTRACT**

GENETIC DETERMINANTS OF COELIAC AND GASTRIC AUTOIMMUNITY IN CHILDREN AND YOUNG ADULTS WITH TYPE I DIABETES

Authors

Kozhakhmetova A¹, Wyatt R, Caygill C, Williams C, Aitken R, Wenzlau J, Williams A, Gillespie K

Institution

University of Bristol, UK¹, Ak13473@bristol.ac.uk

Abstract

Introduction:

Individuals with coeliac disease (CD) and autoimmune body gastritis are at increased risk of type 1 diabetes (T1D), but absolute risks are unclear. These disorders are characterised by genetic predisposition, and environmental influences. This study determined the frequency of autoantibodies to tissue transglutaminase (TGA) and H+/K+-ATPase (ATPA) in well characterised patients with T1D, to identify genetic characteristics of multiple autoimmunity.

Method:

TGA and ATPA were measured by radioimmunoassay in serum from children and young adults with T1D participating in the Bart's Oxford family study (n=1061; male/female 464/597; median age 11.8 yrs, range 0.7- 28 yrs; median duration of T1D 3 months, range 0-17 yrs). CD diagnosis was available from questionnaires. DNA was typed for DR3-DQ2 (DR3), DR4-DQ8 (DR4) and other (DRX) HLA class II alleles using PCR-SSP. The CTLA4 SNP rs3087243 was analysed using Taqman genotyping. Data analysis, including gene interaction tests were performed using logistic regression.

Results:

TGA and/or CD prevalence was 9.1%; ATPA were positive in 8.4%; with both autoantibodies present in five patients. Coeliac and gastric autoimmunity were associated with female gender (OR 1.7, $p = 0.014$ and OR 2.1, $p = 0.001$, respectively). Older age and later T1D onset decreased risk of coeliac autoimmunity (OR 0.9, $p = 0.00007$), but increased risk of gastric autoimmunity (OR 3.6, $p = 0.02$).

Risk for coeliac autoimmunity was associated with DR3/DR3 (OR 6.1, $p = 0.008$), DR4/DR4, DR3/DRX and DR3/DR4 genotypes (OR 5.4, $p = 0.014$, OR 4.4, $p = 0.022$ and OR 4.1, $p = 0.021$, respectively). Interaction between DR3/DR3 and the CTLA4 A allele, resulted in decreased risk (OR for interaction 0.1, $p = 0.036$). Increased risk for gastric autoimmunity was associated with DR3 (OR 3.0, $p = 0.0002$) and DR3/DR4 genotypes (OR 4.6, $p = 0.016$), as well as DRB1*0404 alleles (in males, OR 2.4, $p = 0.046$), while the CTLA4 G allele conferred protection (OR 0.6, $p = 0.05$).

Discussion:

Prevalence of coeliac and gastric autoimmunity in individuals with T1D is increased, with females at greater risk. Coeliac autoimmunity is higher in younger patients, in contrast to gastric autoimmunity. Coeliac autoimmunity was linked to DR3 genotypes, but DR4/4 homozygosity also increased risk. The minor (A) SNP allele in CTLA4, decreased risk in DR3/DR3 patients. Gastric autoimmunity was linked to DR3 and DR3/DR4 genotypes. Unlike coeliac autoimmunity, the CTLA4 G allele decreased risk of gastric autoimmunity. A larger study is ongoing to predict individuals with T1D at increased risk of coeliac and gastric autoimmunity and understand common mechanisms of autoimmunity.

AUTOANTIBODIES TO TISSUE TRANSGLUTAMINASE TARGET EPITOPES DEPENDENT ON RESIDUES IN THE N-TERMINAL DOMAIN

Authors

Kozhakhmetova A¹, Wyatt R, Elvers K, Emery D, Williams C, Annis P, Lampasona V, Gillespie K, Williams A

Institution

University of Bristol, UK¹, Ak13473@bristol.ac.uk

Abstract

Introduction:

Tissue transglutaminase (tTG), is the main autoantigen of coeliac disease (CD). Specific tTG epitopes, including N- and C-terminal sites as well as the catalytic triad, were reported to be targeted by autoantibodies in CD, but the findings are controversial. We aimed to confirm which of these sites is critical for antibody binding as this could aid design of more specific assays and inform studies of disease pathogenesis.

Method:

Plasmid DNA encoding human tTG was mutated at sites in the catalytic core (Cys277, His335, Asp358 to alanine), N- (Arg19, Glu153 to serine) and C-terminal domains (Met659 to serine) using PAGE-purified mutagenic primers. Antibody binding to a panel of 35S-labelled tTG mutant antigens synthesized in vitro was assessed by radioimmunoassay in sera from 111 TGA positive individuals (mean age 40 years, range 1.1 - 80 years) out of 445 patients sent for measurement of CD associated autoantibodies to the Cork University Hospital, Ireland. Wilcoxon signed ranks test (SPSS) was used for statistical analysis.

Results:

Showed that single mutations of catalytic core amino acids Cys277, His335 or Asp358 had little effect on the relative binding (RB) of antibodies to antigen compared with native tTG (median RB; p-values: 100%; 0.723, 93%; 6.1E-5, 106%; 0.019, respectively for each mutant), while the triple Cys277/His335/Asp358 mutation slightly decreased RB (85%; 3.7E-16). Single mutation of the N-terminal amino acids, Arg19 or Glu153, elicited significantly decreased RB in $\geq 90\%$ of sera (66%; 4.2E-17, 76%; 1.0E-11, respectively), as well as for the double Arg19/Glu153 (48%; 3.1E-17) and triple Arg19/Glu153/Met659 (48%; 1.1E-15) mutants. Mutation of Met659 alone had a variable effect, decreasing RB in 36% of sera, but increasing binding in others (102%; 0.004). The combined Cys277/His335/Asp358 and Arg19/Glu153/Met659 tTG mutations reduced binding in 96% of sera (60%; 2.2E-19).

Discussion:

The N-terminal domain of tTG involving amino acids Arg19 and Glu153 is important to tTG autoantibody epitope integrity in CD. Simultaneous mutation of these amino acids has the highest impact on antibody-binding to tTG, while modifying the catalytic core, previously reported as being important, had little effect on binding. The effect of the C-terminal mutation was more variable. These findings suggest that a major epitope is located in the N-terminus, but additional regions of the antigen are likely to contribute to antibody binding. Characterisation of mechanisms and epitopes involved in anti tTG autoimmunity, should aid development of targeted therapeutic manipulations in CD patients or at risk individuals.

CLINICAL INERTIA AND COELIAC DISEASE

Authors

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Abstract

Introduction:

Clinicians' knowledge and practice may directly affect patients' diagnostic pathway. An Endomysial antibody (EMA) has a >90% positive predictive value for coeliac disease. Furthermore NICE have recommended that patients with suspected coeliac disease should have an endoscopy and biopsy within six weeks. This should serve to reduce the temptation by the patient to start a gluten free diet. We aimed to determine GI consultant practice by assessing their 'grading' for patients referred from primary care with a positive EMA. In addition, we sought to determine gastroenterologists' views about coeliac disease.

Method:

Data regarding time to diagnostic endoscopy was collected from adult patients who had a positive EMA test in primary care from two centres (n=151). As a comparator cohort, we collected data regarding the time from symptom presentation in primary care to index endoscopy in adults referred with suspected IBD (n=92). In addition, an unselected cohort of gastroenterology consultants and specialist registrars (n=50) completed a questionnaire regarding coeliac disease.

Results:

The median time from positive EMA identified in primary care to referral for diagnostic endoscopy was 23 (12-35) days; the time from referral to endoscopy was 55 (26-90) days. Overall, coeliac patients waited significantly longer from referral to endoscopy than patients who were diagnosed with IBD (34.5 [18-70] days; $p = 0.006$). Overall time from EMA positive blood test to endoscopy was 78 (58-120) days.

32% (16) of gastroenterologists failed to identify that coeliac disease was more prevalent in the adult population than IBD. 16% (8) of respondents felt that a diagnosis of coeliac disease does not significantly impact patient quality of life. 36% (18) felt that doctors were not required for the adequate management of coeliac disease.

Discussion:

There are delays in diagnosis for patients with coeliac disease. This may impact treatment intensification and thus patient related outcomes. Our data suggest that provider related beliefs may contribute to clinical inertia in this condition. We advocate enhancing both undergraduate and postgraduate training about coeliac disease to help reduce this effect.



TO REVIEW CHANGES IN PRESENTATION OF CHILDREN WITH COELIAC DISEASE IN OUR CENTRE

Authors

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Abstract

Introduction:

Coeliac disease (CD) is a lifelong autoimmune condition primarily affecting the small intestine. It is characterised by symptoms which include bloating, diarrhoea, nausea, constipation, tiredness, weight loss and anaemia. Some patients may also be asymptomatic but have a strong family history or other autoimmune conditions. Treatment is lifelong exclusion of gluten. New guidelines for the diagnosis of CD in children came out in 2012. The aim of this abstract is to assess the presenting symptoms of CD in patients within our centre and compare to previous data reported in 2009.

Method:

Using a retrospective database all children diagnosed with CD in our centre were identified since 2013 when our centre adopted the new ESPGHAN/ BSPGHAN guidelines. Information was gathered including age at diagnosis, presenting symptoms and associated co-morbidities.



Results/Discussion:

173 patients were identified. Median age at diagnosis was 7.83 years. 102/173 (59%) patients reported more than one symptom at diagnosis. 18/173 (10%) of patients had Type 1 Diabetes at time of diagnosis of CD, of these 5/18 (28%) were reported as asymptomatic. Of the whole cohort 14/173 (8%) patients described themselves as asymptomatic. Other reported co-morbidities included hypothyroidism (3/173) and Trisomy 21 (2/173). 87% of patients had no associated co-morbidity.

The most frequently reported symptoms were abdominal pain in 87/173 (50%) and abnormal appetite. Abnormal energy levels were reported in 58/173 (33%) and 81/173 (47%) respectively. Other common reported symptoms include diarrhoea (25.8%), fatigue (25.8%) and constipation (16%). Weight loss was only reported in 25/173 (14%) patients.

The median age at diagnosis is comparable to our review in 2009 (7.83 yrs c/w 7.25 yrs). Symptom presentation appears to be changing with more typical symptoms of CD such as poor weight gain and diarrhoea being less common in present day (14% c/w 17.4% and 46.5% c/w 25.8% respectively). Abdominal pain was the most frequently reported symptom in both data sets (50% in current data set c/w 51.2% in 2009 data set). The majority of patients in our current data set had no associated co-morbidity and when compared with the number of patients with co-morbidity in 2009 there was a reduction (13% patients in current data set c/w 28.5% in 2009 data set). The number of asymptomatic patients had reduced in our current data set (8% in current data c/w 14% in 2009 data set). This data shows that the presenting symptoms in patients with CD are changing from our previous audit in 2009.

LENGTH OF TIME FOR TTG ANTIBODY LEVELS TO NORMALISE FOLLOWING DIAGNOSIS OF COELIAC DISEASE.

Authors

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Abstract

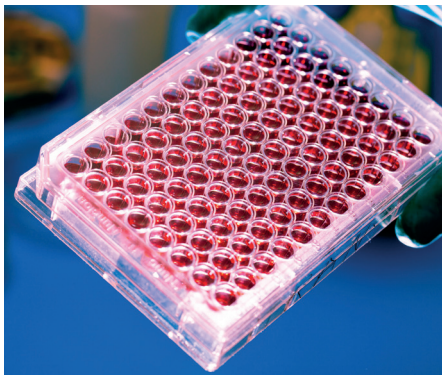
Introduction:

Coeliac disease is an autoimmune condition which is life long and treated with a gluten free diet. Diagnosis criteria for paediatric patients changed in 2012 following the introduction of BSPGHAN/ ESPGHAN guidelines and this was adopted by our centre in 2013. Patients can now be diagnosed on serology if tissue transglutaminase (tTG) antibodies are greater than 10 times the upper limit of normal (ULN) and are IgA Endomysial Antibody (EMA) positive on a second sample. Patients also need have positive

HLA tissue typing. Those with tTG's less than ten times the ULN and those who are asymptomatic or with a co-morbidity will require an upper GI endoscopy with small bowel biopsies. tTG antibodies remain the gold standard for assessing compliance with the diet. The aim of this abstract is to assess the length of time for tTG to normalise following the diagnosis of coeliac disease.

Method:

All patients diagnosed with coeliac disease in our service since the new guidelines were introduced in 2013 were identified via retrospective database. Data collected included tTG at the time of diagnosis, the method of confirmation of the diagnosis (serology/biopsy), tTG at six months and one year following diagnosis.



Results/Discussion:

111 patients were included in the audit. 57/111 (51%) underwent a biopsy to confirm the diagnosis. Reasons for this included tTG less than 10 times upper limit normal, child under 2 years, patients with associated condition or patients who reported themselves as asymptomatic. Ninety seven tTG results were available at six months post diagnosis of these 30/97 (31%) were within normal range (less than 7U/ml) ($p = 0.000$). At 12 months post diagnosis 93 results were available and 54/93 (58%) were within normal range ($p = 0.000$).

This data demonstrates that over half of patients' tTG antibody levels would be within normal range at a year following diagnosis. This is useful data as many parents are keen to know when levels have normalised.

THE ROLE OF A POINT OF CARE TEST, SIMTOMAX (IgA/IgG DEAMIDATED GLIADIN PEPTIDE), IN PREDICTING HISTOLOGICAL REMISSION IN COELIAC DISEASE ON A GLUTEN FREE DIET

Authors

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Abstract

Introduction:

Coeliac disease (CD) is a chronic inflammatory enteropathy treated with a life long gluten free diet (GFD). Non adherence can lead to symptoms and complications such as osteoporosis and malabsorption. Moreover, patients with persistent villous atrophy are twice as likely to develop lymphoproliferative malignancies compared to those who achieve mucosal healing. Therefore, the optimal assessment of treatment response is evaluation of duodenal histology. However, there is little

consensus in the UK on routine re-biopsy during follow up. Duodenal biopsy requires a gastroscopy which is invasive and can be poorly tolerated. There is currently no reliable surrogate marker for histological remission in daily clinical practice. We aimed to assess the role of an IgA/IgG-deamidated gliadin peptide (DGP) based finger prick point of care test (POCT), Simtomax[®], in predicting histological remission in CD.

Method:

We prospectively recruited patients with known CD attending for a gastroscopy with duodenal biopsy for the assessment of histological remission. IgA endomysial antibodies (EMA), IgA tissue transglutaminase antibodies (TTG), IgA levels and Simtomax® were performed in all patients at endoscopy. Patients also completed a validated GFD adherence questionnaire devised by Biagi et al. which gives a 5 point score (0-4), with the highest score indicating strict dietary adherence. A gastroscopy was performed with quadrantic biopsies taken from the second part of the duodenum and one from the duodenal bulb. We compared all surrogate markers to the gold standard of duodenal histology.

Results:

A total of 217 (70% female, age range 16-83, median age 53) patients with CD on a GFD (median duration 6 years) were recruited from 2013-2017. Eighty five (39.2%) patients had persistent villous atrophy. Simtomax® was the most sensitive surrogate marker in predicting villous atrophy ($p = 0.0005$). (Table 1).

Discussion:

Of all clinically available surrogate markers, Simtomax® had the highest sensitivity in predicting villous atrophy. Simtomax® has the additional advantage of convenience being a finger prick test, providing rapid results within 10 minutes. In combination with clinical and dietetic assessments, Simtomax® could aid clinical decision making on the necessity of follow up duodenal biopsy within the same consultation.

Table 1: The sensitivities of Simtomax®, TTG, EMA and adherence score in predicting histological remission measured against histology.

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Simtomax®	67.1 (56.0-76.9)	59.1 (50.2-67.6)	51.4 (45.0-57.6)	73.6 (66.6-79.6)
TTG	44.7 (33.9-55.9)	86.4 (79.3-91.7)	67.9 (56.4-77.5)	70.8 (66.5-74.8)
EMA	37.7 (27.4-48.8)	89.4 (82.9-94.1)	69.6 (56.5-80.1)	69.0 (65.1-72.6)
Adherence score	24.7 (16.0-35.3)	86.4 (79.3-91.7)	53.9 (39.8-67.3)	64.0 (60.8-67.2)

QUALITY STANDARDS IN COELIAC DISEASE: A RETROSPECTIVE EVALUATION IN A SINGLE SPECIALIST CLINIC

Authors

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Abstract

Introduction:

Quality standards in coeliac disease management were recently published by the National Institute for Health and Care Excellence. These specify a new six week target for the time from referral to endoscopy, which was previously covered by the 18 week referral to treatment (RTT) pathway. They also state that all newly diagnosed patients should discuss a gluten free diet with a specialist dietitian. We retrospectively evaluated practice in the Oxford University Hospitals NHS Foundation Trust coeliac clinic against these criteria, and against national guidelines (duodenal bulb sampling at endoscopy and screening for nutritional deficiency).

The medical records of 110 patients referred to our clinic between September 2015 and September 2016 were

examined. The date of referral and endoscopy were recorded, along with relevant demographic, clinical and laboratory information. Data were collected and analysed in Microsoft Excel.

Eighty five patients (68% female, median age 34) were seen with suspected or newly diagnosed coeliac disease, of whom 76 (89%) were referred with positive coeliac serology and would be subject to the six week target. Six patients declined or delayed endoscopy, and endoscopy or referral information were not available for 4 patients. For the remaining 66 patients, median time from referral to endoscopy was 12 weeks 2 days (SD 37 days), with 59 patients (89%) within 18 weeks, but only 11 patients (17%) within 6 weeks (Figure 1). Duodenal bulb biopsies were taken at endoscopy in 31 patients (44%).

A diagnosis of coeliac disease was made in 74 (87%) of all patients referred, of whom 67 (90%) were referred to a specialist dietitian. Haematinics (iron studies, vitamin B12 and folate) were measured in 67 patients (90%), bone densitometry was measured in 51 patients (69%) and all patients were offered a follow up appointment in the coeliac clinic. Iron deficiency was found in 31 patients (45%) of patients tested, folate deficiency in 12 patients (18%), vitamin B12 deficiency in 5 patients (7%) and vitamin D deficiency in 23 patients

(38%). Osteoporosis was diagnosed in 5 patients (10%) and osteopenia in 10 patients (20%).

Appropriate dietitian referral, specialist follow up and screening for nutritional deficiency and bone disease occur within the Oxford coeliac disease service. Compliance with recommended biopsy protocols was only 44%. Whilst most referrals met the previous 18 week RTT pathway, few would have met the new quality standards.

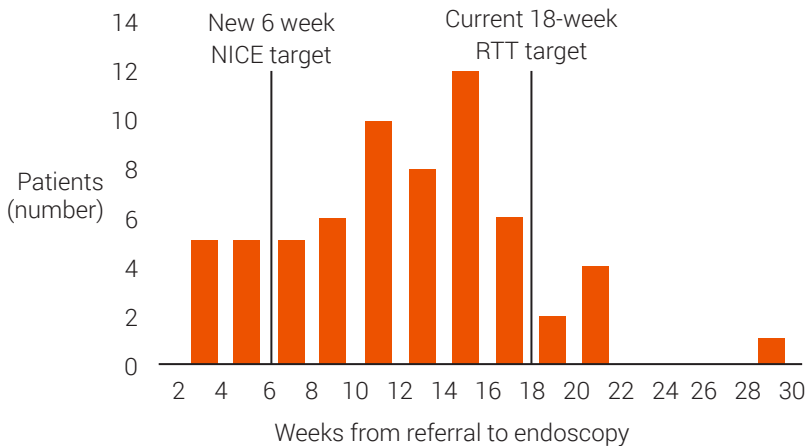


Figure 1: Time from referral to diagnostic endoscopy for patients with suspected coeliac disease.

USING MRI TO IMPROVE OUR UNDERSTANDING OF COELIAC DISEASE

Authors

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Abstract

Introduction:

Studies have shown disordered gut transit in untreated coeliac disease. This research uses MRI to investigate difference in gut transit in adults newly diagnosed with coeliac disease (CD) and healthy volunteers who do not have coeliac or other bowel disease and do not exclude gluten from their diet.



Method:

14 healthy volunteers 9 males 5 females aged 20 - 67, mean 47, and 14 newly +diagnosed coeliac patients, 10 females and four males aged 20 - 59, mean 36, participated. They completed two validated questionnaires to assess their quality of life, the Patient Health Questionnaire 15 Somatic Symptom scale (PHQ15-SS) and the hospital anxiety and depression scale (HAD). They underwent a short MRI scan 24 hours after ingesting 5 MRI transit marker capsules. The MRI study measured the whole gut transit time (WGTT) as previously validated.

Results:

The two groups were matched for age, gender and body mass index (BMI). Compared to the HVs, the CD patients had significantly high scores for HAD-D ($p = 0.0007$) and PHQ15-SS ($p = 0.0002$) symptom questionnaires. In the coeliac patients WGTT was double compared to the HVs, this difference being significant ($p = 0.01$).

Discussion:

This preliminary experience suggests that MRI can offer new insights into the pathophysiology of CD. The imaging exam is quick, non-invasive and acceptable to patients and could help monitoring and follow up, complementing existing more invasive techniques.

MONITORING OF VITAMIN D IN AN ADULT SECONDARY CARE COELIAC POPULATION

Authors

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Abstract

Introduction:

Vitamin D deficiency is a complication in patients with coeliac disease but there is no UK consensus on frequency of monitoring or supplementation. We sought to determine practice in monitoring and supplementing vitamin D over the last 14 years in a single hospital centre adult coeliac population in Birmingham.

Method:

Hospital informatics data was obtained for 1423 patients diagnosed with coeliac disease coding in UHB GI/dietetic clinics. It was determined whether vitamin D was checked and prescribed.



Results:

Overall, 19.2% (n=274) of all patients diagnosed with coeliac disease had their vitamin D levels checked. Of these, 24% were very deficient (<30nmol/L) and a further 30% were deficient (<50nmol/L). 229 people (16%) were prescribed supplementation in the hospital records. There were no overt trends by BMI.

Of the Asian population (n=156), 28.8% (n=45) had their vitamin D levels checked. 64.4% (n=29) of these were deficient (<50nmol/L). Of the Caucasian population (n=1020), 17.5% (n=179) had their levels checked. 49.7% were deficient (n=89).

Conclusion:

One fifth of coeliac patients have vitamin D levels checked at hospital. Of those, around half need supplementation. Given variation by ethnicity and limited evidence of prescriptions, seemingly the process of diagnosing and supplementing vitamin D deficiency in the adult coeliac population needs further study.



COMPARISON OF ADHERENCE TO A GLUTEN FREE DIET IN CAUCASIANS AND SOUTH ASIANS WITH COELIAC DISEASE, USING THE COELIAC DISEASE ADHERENCE TEST (CDAT) SCORE

Authors

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Abstract

Introduction:

Dietary adherence in coeliac disease is multifactorial and ranges from 36% to 96%. The aim of this research is to identify differences between the adherence to a gluten free diet in patients with coeliac disease from Caucasian and South Asian populations.

Method:

A combined cross sectional survey using validated questionnaire and coeliac disease adherence test (CDAT) score and review of clinical and laboratory data was utilised. From 1248 histologically confirmed patients with coeliac disease 972 (85% Caucasians, 86% Females) met the inclusion criteria and were approached through letters with available support in seven ethnic languages.



Results:

The completion rate for the Caucasian population was 40.6% (n=337) and 26.5% (n=38) for South Asian population. Gluten free dietary adherence, CDAT score ranged from 7 to 30 with mean value of 14.7; overall adherence to the gluten free diet was 61%. There were no significant differences between gluten free diet adherence based on ethnicity, age, or gender. Membership of Coeliac UK, affordability of gluten free foods and understanding food labelling were significant factors in gluten free dietary adherence.

Conclusion:

In contrast to an earlier study there were no ethnic differences in adherence to the gluten free diet; membership of Coeliac UK and affordability of GF products are still associated with adherence to the gluten free diet.



DISORDERED EATING PATTERNS IN COELIAC DISEASE: A FRAMEWORK ANALYSIS

Authors

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Abstract

Introduction:

The need for gluten free dietary management in coeliac disease may lead to the development of disordered eating patterns. A theoretical model of disordered eating has been proposed to explain disordered eating in coeliac disease. The model suggests that both extreme adherence to the gluten free diet and dietary transgressions contribute to disordered eating in coeliac disease. The aim of this study was to explore the experiences of typical and disordered eating in coeliac disease to gain a greater understanding of these processes and explore specific pathways within this model.

Method:

We interviewed 21 individuals with coeliac disease about their experiences with food and food environments (participants were recruited from a previous database). Information about disordered eating status was assessed via questionnaire. The interviews were analysed qualitatively using Framework analysis, which was underpinned by the theoretical model of disordered eating in coeliac disease.

Results:

Experiences differed between those participants who scored high on measures of disordered eating and those who scored low (typical eaters). Participants who scored high on measures of disordered eating were concerned about the consequences of their gluten free diet on body image and they described eating patterns similar to binge/restrict cycles. Typical eaters reported being able to integrate their dietary self management into their daily lives; however, general concerns around food and cross contamination were associated with a restriction in food intake.

Discussion:

Our findings suggest that the theoretical model of disordered eating provides an adequate explanation of disordered eating patterns in coeliac disease. Disordered eaters reported eating patterns suggestive of a binge/restrict cycle, which was associated with poor dietary management. However, some typical eaters reported extreme adherence by going for long periods of time without food. They believed that limiting food consumption was protecting them from gluten exposure, particularly when outside the home. Current measures of disordered eating did not capture the consequences of these beliefs on eating patterns. This form of dietary self management may result in eating behaviours that could be considered 'disordered'. The need to follow a gluten free diet and be vigilant around food has to be balanced with concerns around food.

FOOD INTAKE AND PROCESSING OF FOOD CUES IN COELIAC DISEASE: A PILOT STUDY

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Abstract

Introduction:

The need for vigilance around food in coeliac disease (CD) may contribute to disordered eating attitudes and a restrictive eating pathology in some individuals. Concerns around food and dietary restriction are reported in unfamiliar settings or when an unknown individual is preparing food. The CD Food Attitudes and Behaviours scale (CD-FAB) quantifies food concerns and the behavioural response to these concerns, with high scores indicating greater concerns and dietary restriction.

Hypervigilance to food cues may reinforce concerns about food content and preparation, which may contribute to disordered eating attitudes and behaviours. We anticipated that higher CD-FAB scores would be associated with:

- 1) limited food intake; and
- 2) a greater vigilance towards foods images.

Method:

Forty one individuals with CD completed the CD-FAB and a food task. During the food task, participants were given the opportunity to try four gluten free foods (two savoury, two sweet); participants were free to inspect the food packaging before the contents were emptied into four bowls. Individuals were invited to consume as much food as desired; we were interested in how much food (if any) was consumed. Variables that influence food consumption were assessed via questionnaire (mood, food liking, satiety, hunger, food familiarity, disordered eating). A computerised measure of vigilance towards gluten free and gluten containing food images was completed by those with CD and ten healthy controls (using the dot probe paradigm).

Results:

Self reported food attitudes and behaviours, as assessed by the CD-FAB did not explain additional variance in calories consumed, above and beyond covariates (disordered eating and food familiarity). Compared to healthy controls, individuals with CD showed greater vigilance towards gluten containing images ($t(1,48) = 2.03$, $p=0.048$) but no relationship between CD-FAB scores and vigilance towards food images was found.

Discussion:

Contrary to our hypothesis, CD-FAB scores were not associated with food intake and vigilance to food images. These findings may stem from methodological issues within the study, including the perceived safety of a lab environment and the ability to check the packaging of foods. However, compared to healthy controls, there is evidence that those with CD process gluten containing food images differently. This is in line with qualitative reports that indicate increased food preoccupation in CD. These results require replication and further exploration in a larger sample.

GLUTEN NEUROPATHY: CLINICAL CHARACTERISTICS AND IMPACT TO MENTAL HEALTH

Authors

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Abstract

Introduction:

After cerebellar ataxia, peripheral neuropathy (PN) is the commonest neurological manifestation of gluten sensitivity. We aimed to examine the clinical characteristics and estimate the prevalence of pain in patients with PN and gluten sensitivity. We also investigated its impact on patients' mental status.

Method:

Between October 2015 and January 2017 all consecutive patients attending a specialist gluten/neurology clinic, were invited to participate. All patients were examined clinically and neurophysiologically. Pain was assessed via the DN4 questionnaire and the visual analogue scale (VAS). Overall Neuropathy Limitations Scale (ONLS) was used to assess the severity of neuropathy. The Mental Health Index (MHI-5) was used to measure participants' general mental health status.

Results:

In total, 55 patients (74.5% males) with PN and gluten sensitivity were recruited. The age of the patients ranged from 45 to 88 years (mean 70.0 ± 9.8 years). Twenty-nine patients (52.7%) were on a strict gluten-free diet (Biagi score 3 or 4).

Symmetrical sensorimotor axonal neuropathy was the commonest form of neuropathy (69.1%), followed by sensory ganglionopathy (29.1%) and mononeuritis multiplex (1.8%). The ONLS score ranged from 1 to 7 (mean 3.2 ± 1.8).

Pain was present in 35 patients (63.6%). Based on the DN4 questionnaire in 34 (97.1%) patients with painful PN, the pain was neuropathic in nature. Based on the VAS, the pain intensity on examination ranged from 0 to 7 (mean 2.6 ± 2.3) and the maximum intensity of pain ranged from 2 to 10 (mean 7.0 ± 2.3). In 10 patients (18.2%) pain was the first symptom of their neuropathy.

Comparison between groups of painful and not painful PN did not show significant differences regarding age, gender, neuropathy severity, neuropathy type or type of diet (gluten free or not). Patients with pain, however, presented with significantly worse general mental health status based on the MHI-5 score (76.6 ± 13.6 versus 85.8 ± 9.6 , $p = 0.01$).

Discussion:

Pain is very prevalent in PN associated with gluten sensitivity and has a significant effect in patients' mental health status. Screening for PN and evaluation of pain in patients with gluten sensitivity is highly recommended.

THE COELIAC DISEASE ASSESSMENT QUESTIONNAIRE (CDAQ): RESPONSIVENESS TO CHANGE

Authors

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Abstract

Introduction:

The Coeliac Disease Assessment Questionnaire (CDAQ) is a 32 item patient reported outcome measure (PROM) developed to assess quality of life in adults with coeliac disease. It is suitable for use in research, including clinical trials, and clinical practice. The CDAQ is a reliable and valid measure, but its ability to detect change over time is yet to be assessed. Therefore, the aim of this study was to assess the CDAQ's responsiveness to change.

Method:

Recently diagnosed Coeliac UK members (n=1443) were invited to participate in a postal (n=500) or online (n=943) survey, completing the CDAQ at two points in time, four months apart. Baseline, follow up, and change scores for each of the CDAQ's five domains (Stigma, Dietary burden, Symptoms, Social isolation, and Worries and concerns) and Overall index scores were calculated. The following distribution based indicators of responsiveness to change were calculated: effect size (ES), standardized response mean (SRM), and minimal detectable change (MDC).

Results:

In total, 277 respondents completed both questionnaires and were included in the analysis. The mean time since diagnosis at baseline was 5.21 months (SD 2.99). Small to moderate ES for the CDAQ Overall index score (0.19), Symptoms (0.27), and Worries and concerns (0.19) domains were found. Small to moderate SRMs for the Overall index score (0.37) and dimension scores (0.22-0.39), except Stigma (0.03), were found. The MDC is estimated as 2.06 for the overall index score, and between 14.08 and 18.99 for the dimension scores.

Comparison between groups of painful and not painful PN did not show significant differences regarding age, gender, neuropathy severity, neuropathy type or type of diet (gluten free or not). Patients with pain, however, presented with significantly worse general mental health status based on the MHI-5 score (76.6 ± 13.6 versus 85.8 ± 9.6 , $p = 0.01$).

Discussion:

In a sample of people with recently diagnosed coeliac disease, the Overall index, Symptoms, and Worries and concerns scores have been found to be responsive to change. The remaining dimensions (Stigma, Dietary burden, and Social isolation) were less responsive. This study highlights the challenges faced assessing responsiveness in coeliac disease, where much of the impact on quality of life is as a result of following a gluten free diet. In this sample, some changes (e.g. improvement in symptoms) would have occurred prior to the study, whereas other changes (e.g. reduced stigma or dietary burden) may take longer to occur or may not occur in a sample that continues to follow a gluten free diet. To overcome this, the CDAQ's responsiveness should be assessed in clinical trials of therapeutic treatments which aim to supplement or replace the gluten free diet. An analysis using anchor based indicators of responsiveness is underway.

ESTIMATING THE QUALITY OF LIFE IMPACT OF COELIAC DISEASE DIAGNOSIS USING RETROSPECTIVE POPULATION SELF REPORTS

Authors

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Abstract

Introduction:

Despite a general improvement in pathways to diagnosis, coeliac disease (CD) may still be diagnosed only after a long period of sometimes severe symptoms. These symptoms may cause significant discomfort and anxiety to the sufferers and their families by negatively impacting on their health related quality of life and further affecting their relationships, social lives and work opportunities. This study aims to quantify the quality of life impact of CD, before and after diagnosis, for patients and their carers, and explore its variation over time, geographical areas of the UK and by socioeconomic groups of the population.

Method:

A postal survey similar to one we conducted in 2006 was carried out at the end of 2015 involving a representative, geographically stratified, sample of 4000 individuals drawn from the membership list of Coeliac UK. Information on socio demographic characteristics of respondents, time since diagnosis, type and duration of symptoms prior to diagnosis, and quality of life, measured using the EQ-5D, before and after diagnosis of both patients and their families are analysed through descriptive and regression based methods to tease out the key drivers of differences in quality of life before and after diagnosis among different socioeconomic strata of the population over time.

Results:

Preliminary analyses of the 1584 returned questionnaires show that the disease is diagnosed only after, on average, 12 years and 9 months of often severe symptoms. This represents only a 3 months improvement compared to the findings of the 2006 study.

On a scale 0 to 1, where 0 is equivalent to death and 1 means full health, participants rated their quality of life before diagnosis as being, on average, equal to 0.65. After diagnosis, their quality of life improved by about 30% to a score of 0.85, which is very similar to what is reported by the general population. In comparison, results from the 2006 study found that participants rated their quality of life before diagnosis lower (0.56) than in the 2015 study (0.65), but reported a similar score after diagnosis (0.84 in 2006 versus 0.85 in 2015).

Data also suggest variation in quality of life experienced by people with coeliac disease pre/post diagnosis by age, family income, geographical location, and access to GF food on prescription.

Discussion:

Preliminary results underscore the importance of further improving early detection of the disease to better the quality of life of people affected by it, especially among people living in deprivation.

THE ECONOMIC BURDEN OF COELIAC DISEASE IN THE UK: A RETROSPECTIVE POPULATION BASED STUDY

Authors

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Abstract

Introduction:

Although the prevalence and health impact of coeliac disease (CD) is considerable, research on the impact of detecting and treating CD on out-of-pocket patients' costs, healthcare resource use and overall economic burden of the disease is still limited.

This study aims to quantify the cost - and its variation over time - of CD in the UK to: (i) patients, in terms of out-of-pocket expenses, including cost of gluten free (GF) food, especially after restrictions on prescriptions of GF products have been introduced in some parts of the country; (ii) the National Healthcare System (NHS) in terms of use of healthcare services due to symptoms and management of the condition; (iii) family and friends of the person with coeliac disease.

Method:

A postal survey similar to one we conducted in 2006 was carried out at the end of 2015 involving a representative, geographically stratified, sample of 4000 individuals drawn from the membership list of Coeliac UK. Information on resource use and costs incurred pre- and post-diagnosis by the patient (e.g. private consultations, food supplements) and the NHS (e.g. GP visits, prescriptions, hospitalisations) are analysed through descriptive and regression based methods to disentangle the key drivers of differences in the economic burden of CD before and after diagnosis among different socioeconomic strata of the population over time.

Results:

Preliminary analyses of the 1584 returned questionnaires indicate that after diagnosis, people with CD incurred a number of out-of-pocket expenses related to the management of their health condition (e.g. costs of private medical consultations), with an overall average amount of £139 per person per year. The corresponding amount found in the 2006 study was £110 per person per year. The largest out of pocket expenditure item after diagnosis was a reported mean increase in food shopping costs of £861 per CD person per year. The corresponding amount in the 2006 study was £679 per person per year. In terms of NHS use, in the early years after diagnosis, respondents (excluding those diagnosed for less than one year) reported to have on average one GP consultation and one routine medical examination per year, with half of

respondents also reporting one dietitian consultation and/or one referral to the gastroenterologist per year, in relation to their coeliac disease. However, the use of these healthcare services dropped dramatically as time since diagnosis increased (Table 1). Family and friends frequently reported feeling limited in daily life situations (eg restaurants, travel) and feeling worried about the person diagnosed with CD's health and wellbeing.

Discussion:

Preliminary results show significant additional out of pocket and NHS costs associated with CD after diagnosis. Unless ameliorated, the high costs of adhering to a gluten free diet may challenge adherence in low income groups.

Table 1: NHS use after diagnosis

NHS service	TIME SINCE DIAGNOSIS			
	1 to < 5 years Mean (Std Dev)	5 to <10 years Mean (Std Dev)	10 to <20 years Mean (Std Dev)	>20 years Mean (Std Dev)
Routine medical examination	2.03 (3.83)	0.96 (1.46)	0.68 (0.98)	0.35 (0.62)
GP	1.51 (3.54)	0.73 (1.55)	0.56 (1.27)	0.21 (0.42)
Dietitian	1.13 (1.81)	0.43 (0.55)	0.24 (0.37)	0.07 (0.12)
Gastroenterologist	0.84 (2.77)	0.34 (0.71)	0.26 (0.45)	0.09 (0.17)

SIGNIFICANCE OF DIFFERENT ACCESS ROUTES FOR GLUTEN FREE BREAD AND IMPLICATIONS FOR PEOPLE WITH COELIAC DISEASE

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Abstract

Introduction:

In the UK, 1 in 100 people has coeliac disease, a lifelong autoimmune disease treated by a strict gluten free diet. Non-adherence to the diet can result in long term complications including anaemia, osteoporosis and small bowel cancer. This research examines access to gluten free bread, a key staple in the diet, in three areas of deprivation in Aylesbury, Buckinghamshire.

Method:

A street by street survey identifying locations where gluten free food could be accessed was carried out to collect data on the availability of gluten-containing and gluten free bread in food shops and pharmacies in three areas in Aylesbury, classified as being in the second most deprived decile of deprivation, Index of Multiple Deprivation (IMD level 2). These data were mapped using Geographical Information System software to model access to bread.

Results/Conclusion:

Access to gluten free bread was limited in the areas studied. Whilst 100% of stores stocked a loaf of gluten-containing bread only 19% of stores stocked gluten free bread (Figures 1 and 2). Gluten free bread was not available in any of the small chain/franchise convenience stores, independent convenience stores, newsagents or petrol station shops. Access to gluten free bread via community pharmacies provided an important access route in the areas investigated where limited availability existed in shops (Figure 3).

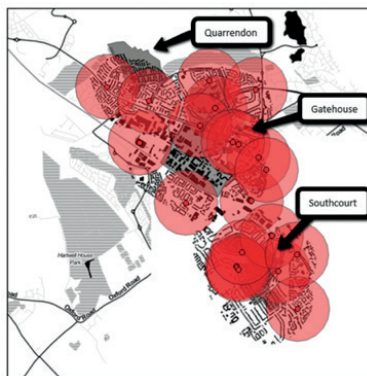


Figure 1. Distribution of shops selling standard GC bread. Map shows location with 400 metre walking distance shown as red circular isochrones.



Figure 2. Distribution of shops selling GF bread. Map shows locations with 400 metre walking distance shown as yellow circular isochrones.



Figure 3. Distribution of outlets (shops and pharmacies) providing access to gluten-free bread. Map shows locations with 400 metre walking distance isochrones.

A COMPARISON OF THE NUTRITIONAL CONTENT OF A RANGE OF COMMERCIAL AND PRESCRIPTION GLUTEN FREE BREAD PRODUCTS

Authors

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Abstract

Introduction:

Prescription gluten free (GF) food is restricted or no longer available in many areas across England. Therefore people with coeliac disease are increasingly reliant on commercially available products. In a gluten-containing diet 30% of calcium and 44% of iron intake is derived from cereals and cereal products, but in the past GF substitute products have often been shown to be not naturally rich or fortified with iron.

Method:

Nutritional content data for GF bread was collected directly from manufacturers, food labels or supermarket websites (Sep 2016 - Jan 2017). Information on kcal, fat, iron and calcium was obtained from six commercial and five prescription only manufacturers. This was compared with the nutritional composition of gluten containing bread (Public Health England, 2015).

Results:

Data was collected for 43 GF breads; nutrient content varied greatly between manufacturers. Compared with gluten containing bread, GF substitute breads were typically higher in kcals and fat (up to 11% increase in kcals and 135% increase in fat); the majority (95%) of GF breads had higher fat content. Where GF breads were fortified they contained equivalent or higher content of calcium and iron (Table 1). However many companies do not fortify or analyse the iron and calcium content; only 8/26 (31%) commercial GF breads and 13/17 (76%) prescription GF breads surveyed were fortified with iron and/or calcium. Some GF breads not fortified do contain calcium based preservatives or mineral rich seeds but as the levels are not analysed nutrient contribution is uncertain.

Discussion:

Nutritional content of GF substitute breads varies greatly between manufacturers and products; the majority of GF breads are higher in fat than the gluten containing breads. GF products that are fortified with iron and calcium typically have equivalent or higher iron and calcium content, however in the GF

breads not fortified the content of these nutrients was often uncertain. From the survey, a higher proportion of the prescription GF breads were fortified than commercial GF breads. Products should be considered on an individual basis but as it is not compulsory to report micronutrient content on packaging it can be difficult to compare products.

Table 1: Comparison of calorie, fat iron and calcium content of GF and gluten containing breads.

	Average portion size (g)	Kcal per 100g	Fat (g) per 100g	Iron (mg) per 100g in fortified products	Calcium (mg) per 100g in fortified products
GF bread (n=26)					
White	32	254	6.1	2.2 (n=3)	280 (n=3)
Brown/seeded	34	265	8.2	3.0 (n=5)	248 (n=5)
Average	33	260	7.3	2.7	260
Range	25-50	190-338	1.1-12.7	0.84-3.6	39-639
Prescription GF bread (n=17)					
White	36	244	5.7	1.9 (n=3)	222 (n=5)
Brown/seeded	34	254	6.9	1.5 (n=4)	220 (n=8)
Average	35	251	6.5	1.6	221
Range	29-50	203-316	2.5-14.1	2.6-4.5	120-630
Gluten containing bread					
White (n=5)	40	236	2.1	1.5	155
Brown/seeded/wholemeal (n=26)	40	231	4	2.3	147
Average	40	234	3.1	1.9	151

PRIORITY SETTING PARTNERSHIP IN COELIAC DISEASE – WE NEED YOUR SUPPORT

This year we have teamed up with the James Lind Alliance, experts in priority setting partnerships (PSP), bringing patients, their carers and clinicians together to agree a top ten list of research priorities.

Several other charities have had very successful partnerships. By raising the profile of the research needs for their particular cause, they have been able to direct research to where it's needed most and attract funding to answer some of the top ten unanswered questions.

In 2014, we carried out a pilot project which has helped us with the focus for this next phase. There are a number of stages within a PSP and we are currently preparing for the launch meeting to be chaired by the JLA Adviser, Maryrose Tarpey and attended by select Coeliac UK staff and the Steering Group which comprises; patients, carers, dietitians, general practitioners, gastroenterologists, paediatric gastroenterologists and a neurologist.

Key stakeholders, the British Dietetic Association, British Society of Gastroenterology, British Society of

Paediatric Gastroenterology, Hepatology and Nutrition, Primary Care Society of Gastroenterology and Ataxia UK have been invited to help disseminate information about the PSP to ensure we are all inclusive and everyone will have the opportunity to have their voice heard. No one understands coeliac disease better than those who live with the condition and those who care for them.

Following the launch meeting the first survey will go live this May, to start the collection of unanswered questions in coeliac disease. We very much hope you will support the project and participate in this survey.

We hope to have a top ten list of research priorities in coeliac disease in time for our 50th birthday celebrations in early 2018.

For further information and regular updates on the progression of the PSP, please visit www.coeliac.org.uk/RP2017

For Further information about the JLA please visit: <http://www.jla.nihr.ac.uk/>

CAMPAIGNING FOR LIVES UNLIMITED BY GLUTEN

For nearly 50 years we've been campaigning for better diagnosis of coeliac disease and more access to gluten free food.

Big strides have been taken, but there is more to do as we search for the half a million living in the UK with undiagnosed coeliac disease. Huge numbers of people have already checked their symptoms on our online assessment, putting thousands on a path to diagnosis.

Check your symptoms today at www.isitcoeliacdisease.org.uk

A diagnosis of coeliac disease should come with a prescription, and not a price tag. So we're campaigning for continued access to gluten free staple food on prescription from the NHS to support adherence to the gluten free diet and to avoid costly complications.

Find out more about more at www.coeliac.org.uk/prescriptioncampaign



COELIAC UK'S HEALTHCARE PROFESSIONAL NETWORK

As a healthcare professional (HCP) or researcher with an interest in coeliac disease, you may wish to become a member of Coeliac UK's professional network. On joining, you will be able to access:

- early registration to the Coeliac UK Research Conference
- exclusive access to post Research Conference videos on our website
- our Electronic Food and Drink Directory, Gluten-free on the Move smartphone app, Recipe Database and Venue Guide
- hard copy of our Food and Drink Directory
- Crossed Grain magazine
- monthly electronic newsletter to support your patients with their condition and diet
- HCP quarterly electronic newsletter
- your own personalised online Scrapbook where you can save all downloadable documents and bookmark the webpages that you find the most useful
- access to our online publications
- an online forum where you can share best practice and information with other HCP Members.

To join, go to www.coeliac.org.uk/join-us/hcp and complete the online form.

In each edition of our quarterly electronic newsletter for HCPs, we have an article on 'People and Patients' written by HCPs to share best practice in the diagnosis and management of patients with coeliac disease. Past articles have covered the management of patients with the dual diagnosis of coeliac disease and Type 1 diabetes and five top tips for providing follow up care to children with coeliac disease. If you are interested in writing an article for the newsletter or would like to find out more we would love to hear from you, please contact Katie at: katie.stokes@coeliac.org.uk

'The gluten free diet; classic coeliac disease and more' has been approved by the Federation of the Royal Colleges of Physicians of the United Kingdom for 4 category 1 (external) CPD credits.

The Coeliac UK 2017 Research Conference is endorsed by:



BDA endorsement applies only to the educational content of the learning activity.




The Coeliac UK Research Conference 2017 is supported by the Primary Care Society of Gastroenterology.

Visit www.coeliac.org.uk/research for more information on Coeliac UK's:

- Research Strategy
- funding opportunities
- previous Research Conference videos
- funded research.

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