SMALLTALK

PAEDIATRIC NUTRITION MAGAZINE FOR HEALTHCARE PROFESSIONALS

SUSTAINABILITY AND TRANSFORMATION

Time for a rethink

103

Autumn/Winter 2023

Meet the Editors

Theresa Cole
Nutricia Medical Affairs Manager-Paediatric Faltering Growth

Jacqui Lowdon
Clinical Specialist Paediatric Dietitian, Cystic Fibrosis at Leeds Children's Hospital

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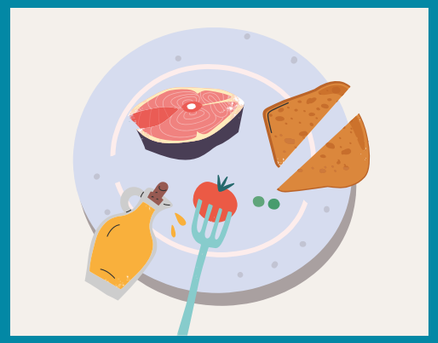

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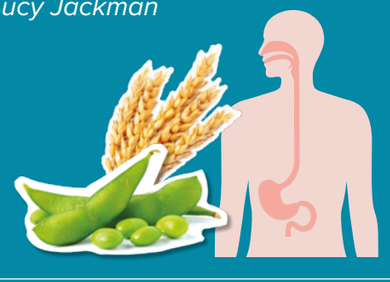

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NUTRICIA ACADEMY
Feed your mind

Welcome

to the autumn/winter edition of small talk.

We hope you had a wonderful summer, despite the fires that raged across the globe. The topic of driving to a more sustainable future has therefore never seemed more important. In this edition we will approach the subject looking at what can be done to support this initiative. In addition, we will address the transformations that are taking place in some areas of practice and what might be needed as we head into the future.

We open with an article by **Jacqui Lowdon** where she talks about the changing profile of children with Cystic Fibrosis from one of undernutrition to overnutrition and with significantly increased survival; thus, shifting from high-fat energy dense-diets to diets for lifelong health. She takes us through the advances in therapies and the impact of these advances on both patients and health care professionals.

Then **Victoria (Tori) Mullen** takes us through some of the challenges of dealing with Multiple Food Allergies (MFA) and how these conditions are managed at her centre, where they focus on early reintroductions to avoid lengthy exclusion diets.

Catherine Kidd shares her expertise on sustainability in health care and why it matters. She addresses some of the simple steps that dietitians can take, which combined, can make a big impact – considering people, the planet and profit.

Lucy Jackman then gives us an in-depth overview on the management of the complex allergic condition, eosinophilic esophagitis (EoE). Lucy, an expert in this area, describes EoE and its prevalence before focusing on the key considerations for optimal dietary management.

We also include a case study by **Lucy Paterson**, an update on why specific ingredients are added to infant formula by **Bahee van de Bor**, and an interview with **Victoria Wilkins** on her move from general paediatrics to a specialist in inherited metabolic disorders.

We hope you will enjoy reading and learning from these experts, and can advance your practice as a result!

Best wishes,

Theresa

Theresa Cole

Get in touch

If you have any feedback, any questions for our next edition, ask the expert, or would like to contribute to our next edition, we'd love to hear from you.

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15
NOVEMBER 2023

Paediatric Respiritory Problems

WHERE: Virtual

MORE INFO: Paediatric Events 2023 (nutricia.co.uk)

15-17
FEBRUARY 2024

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WHERE: Lisbon, Portugal

MORE INFO: https://nutrition-growth.kenes.com

20-22
MARCH 2024

38th Annual Meeting BSPHGAN

WHERE: Bristol, UK

MORE INFO: https://bspghan.org.uk/bspghan-annual-meeting-2024

24-25
APRIL 2024

4th World Congress in Pediatrics and Neonatology

WHERE: London, UK

MORE INFO: www.pediatrics-conferences.com/index.php

CAREER SPOTLIGHT

> My advice to anyone wanting to work in Metabolics is firstly to gain plenty of experience in general paediatric dietetics.

Victoria Wilkins Specialist Paediatric Dietitian in Inherited Metabolic Disorders and Barth Syndrome

How did you become a Metabolic Dietitian?

Prior to starting as a Metabolic Dietitian, I worked as a general paediatric Dietitian. My work involved managing a range of specialities including diabetes, home enteral feeding, food allergy and cystic fibrosis, all of which enhanced my dietetic skills enormously. However, I always saw metabolics as my dream job! Thanks to my love of biochemistry and the fact that diet therapy, for many patients with metabolic conditions, is considered the core treatment.

Did it require specific training?

I attended the BDA paediatric master's module 4 '*Dietetic management of inherited metabolic disorders (IMD)*'. The course included practical workshops, as well as lectures from medical and expert dietitians.

I took a lot from spending time with patient families, discussing their experience of managing the diet and the condition. The biggest challenge was having to write an essay at master's level, without any practical experience managing these patients, knowing that highly expert metabolic dietitians would critique my work. However, I managed to use my transferable skills from other areas of dietetics to help me succeed.

When did you find your "dream job" and did you experience any challenges initially?

In 2020 I was offered the role of Specialist Paediatric Dietitian in IMD and Barth Syndrome. Moving to a new speciality is both exciting and challenging. One of the challenges is the fact that there are so many rare conditions to learn about in metabolics, often with limited data. Luckily, we have an excellent *British IMD Dietitian Group* (BIMDG)[1] which provides unlimited support.

What is the most exciting part about your new role?

Working with such a motivating team of health care professionals, where dietitians are highly respected and can make a big difference to the lives of patients and families. The caseload is varied and includes patients with Phenylketonuria, glycogen storage disorders, urea cycle disorders, to name a few. I am also the only UK Barth Syndrome Specialist Dietitian, which means keeping up to date with international research in the area. There are also lots of opportunities to get involved in research. This year I published a poster for a BIMDG conference on '*A single centres approach to managing Ketotic Hypoglycaemia*' and I'm already working on a poster for a Barth Syndrome conference in Florida next year.

What advice do you have for others who might want to work in this area?

Gain plenty of experience in general paediatric dietetics first. Children with metabolic conditions may also have other medical conditions e.g., cow's milk protein allergy, iron deficiency etc. so it's important to know the basics. In addition, consulting with peers and attending international conferences is essential to learn (from others) how to manage the rare conditions.

1. BIMDG - https://www.bimdg.org.uk/site/index.asp

Nutritional management of children with Cystic Fibrosis

– considerations for advancing practice

Background

Cystic fibrosis (CF), the most common autosomal recessive disease in Caucasians, is caused by mutations of the gene encoding the cystic fibrosis transmembrane conductance regulator (CFTR). CF was traditionally associated with undernutrition, due to disease dysfunction causing recurrent infections, inflammation, malabsorption with resultant increased energy requirements[1].

Advances in detection, such as newborn screening (NBS) and treatments, such as modulators, have increased survival in people with CF (pwCF). Since 2000, children diagnosed with CF in the UK can expect a median survival of greater than 50 years[2]. In addition, the profile of pwCF is changing in the UK, from one of under to overnutrition. Consequently, we need to reconsider some of our traditional nutritional management practices in line with these advances.

> We need to reconsider some of our traditional nutritional management practices in line with these advances.

Life expectancy of pwCF

Several factors have contributed to improved survival in pwCF including multidimensional team working[3,4], NBS[5,6], specialised centre care[7], registries and clinical practice guidelines.[8,9] Improving and maintaining growth and nutritional status in children can also be accredited, adding up to 10 years to median survival.[10]

One recent, major, advancement has been the introduction of CF transregulator modulators (CFTRm). CFTRm is a revolutionary treatment, which partially restores function to the CFTR channels that cause disease dysfunction in CF. It is available to 90% of pwCF in the UK. Using a scenario analysis, the median projected survival for pwCF, who start the most effective CFTRm between the ages of 12-17 years was 82.5 years (~45-year increase) compared to best supportive care alone.[11] The consequences of these advancements in therapy have led to a change in the profile of pwCF with increasing numbers developing overweight and obesity as well as an ageing population.

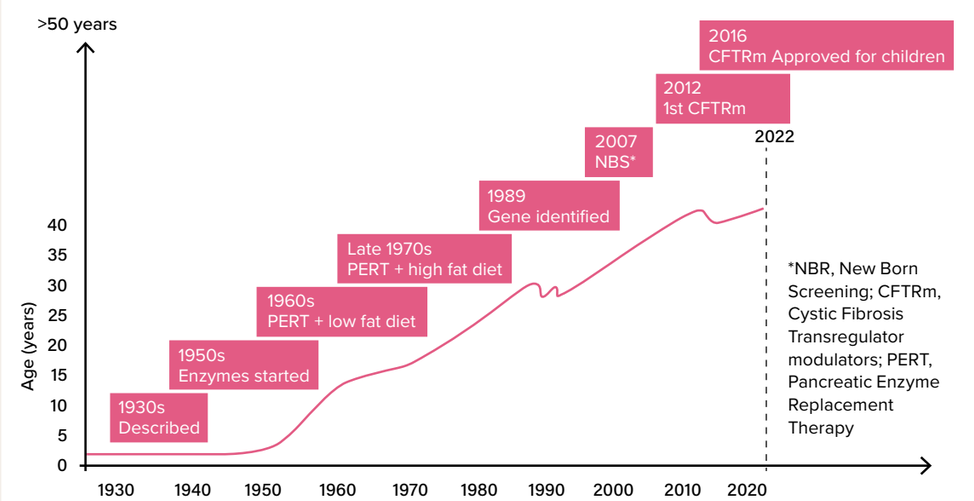
Adult populations have increased worldwide, with more adults now than children.[12] The European CF Society patient registry 2021 reported 54% adults with CF.[13] In the UK, 61.9% of the pwCF are >16 years of age.[14]

With increasing numbers of adults with CF living longer, we are potentially looking at an adult population who may go on to develop more CF comorbidities as well as age related comorbidities. For example, CF diabetes (CFD), which already is estimated to affect over 50% of the adult CF population.[15] In addition, increasing numbers of pwCF are living post-lung transplant. Not only does this increase the number of patients cared for in clinical practice but also increases the complexity of care with comorbidities associated with older age.

Change in patient profile

There is an increasing prevalence of overweight/obesity in pwCF being reported worldwide.[14,16,17,18], For example, a centre in the US reported seeing 8% obesity in 2-18-year-olds with CF.[19] CFTRm has been used in younger children in the US for longer, and although no recent evidence exits on obesity rates in UK CF children, we expect this to trend as we advance CFTRm therapy in progressively younger populations.

With improvements in the life expectancy of pwCF, along with the potential impact of overweight/obesity on health outcomes, such as on lung function, cardiovascular disease and diabetes, a review of our traditional nutritional interventions in pwCF is warranted.



Adapted from Garcia et al 2022; Kramer et al 2016

Traditional nutritional management

Nutritional interventions have traditionally focused on a combination of calorie-dense diets (achieved through added fats), vitamin supplementation, oral nutritional supplements (ONS) and enteral tube feeding (ETF), as well as optimising pancreatic enzyme replacement therapy (PERT); all with the aim of achieving and maintaining the desired BMI[8,20] which for children and young people was >/= 50th percentile. Achieving the desired BMI was considered essential owing to the data linking BMI with better lung-function, correlating with improved survival. For children and young people, achieving and maintaining the desired BMI was associated with an FEV1 at approximately 80% of predicted.

This was reflected in the nutritional guidelines for children with CF which proposed a daily energy intake of 110-200% of energy requirements, with 20-25% protein from energy, and higher salt intakes compared to healthy peer.[8,21] With the emphasis on weight gain and BMI, energy-dense (fat rich), nutrient-poor foods were promoted, rather than nutrient-dense foods, with little focus given to micronutrients. This dietary approach, also called the CF "legacy diet", contributed to a poor-quality diet lacking in nutrient dense foods[22] and dietary fibre.[23] Little consideration was given to the long-term health of pwCF as survival was low, commonly < 40yrs[24], so the impact of these diets on the development of non-communicable diseases (NCD) was not a focus.

Traditionally, high salt intake was also considered important, due to losses in sweat, especially for infants and children[8] where adequate sodium is crucial for optimal growth.[25] The guidelines therefore recommended salt intakes to account for these losses.[8, 26]

However, with the advances in CFTRm and other therapies we may no longer need to promote many of the traditional interventions.

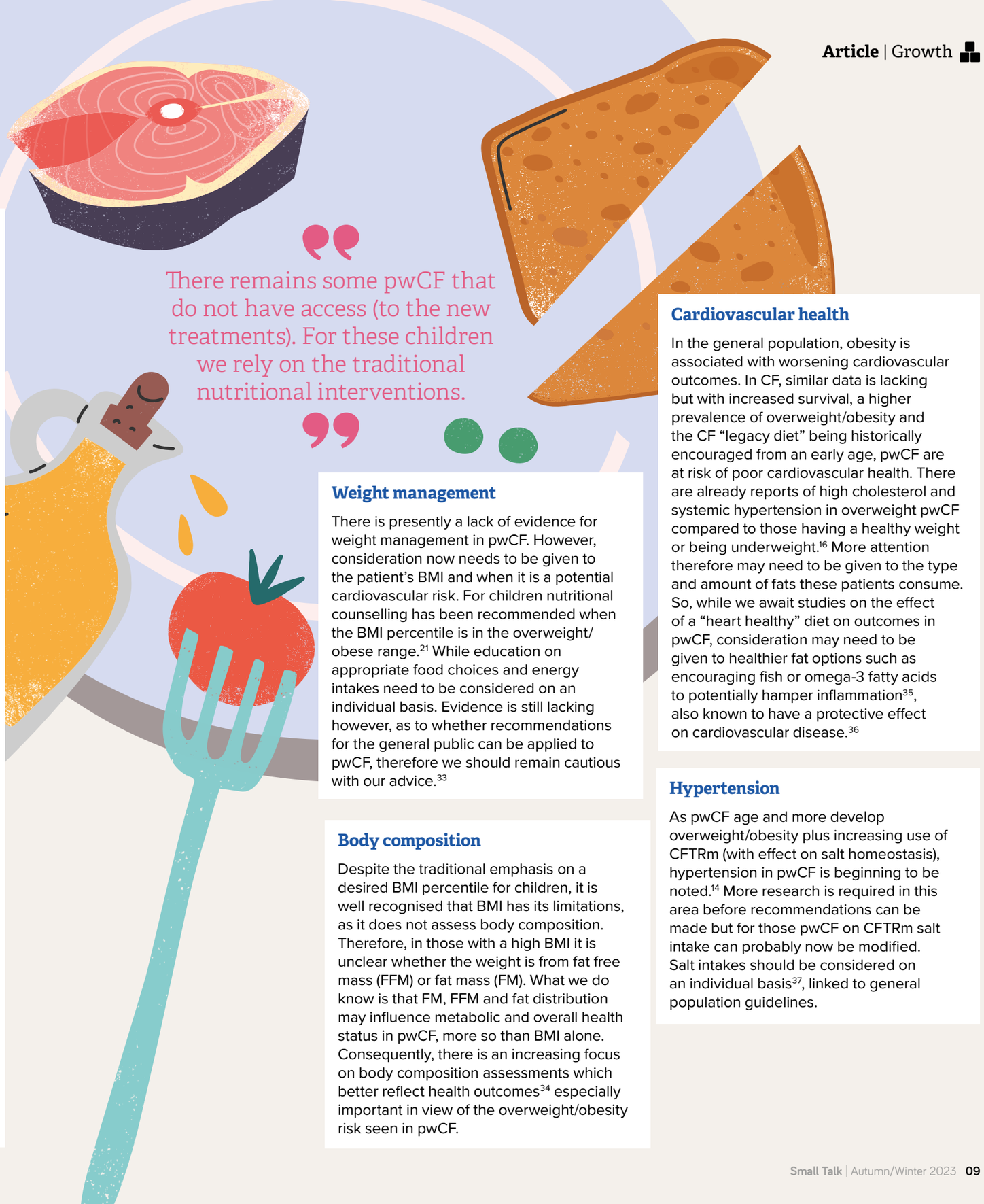
Looking forwards – important focus areas

Guidance is moving towards an age-appropriate healthy diet, associated with positive (long-term) health outcomes, similar to the general population.[27] So, in children with CF who are achieving their desired growth/target weight, food associated with optimal health benefits are now encouraged i.e. fruits, vegetables, low fat dairy products, lean meats (for omnivores), wholegrains, legumes. CFTRm affects sodium homeostasis so additional salt may not be required.

Furthermore, supplementation of fat-soluble vitamins (FSV) which has been part of standard CF management[8] may not be required; several studies have suggested this may be due to CFTRm.[28,29,30] In addition, there have been increasing reports of hypervitaminosis in pwCF. Therefore supplementation will need to continue to be monitored, but with a possible reduction in dose.

With the risk of overnutrition in some pwCF, there appears to be a lower dependence on the use of ETF. The UK CF Trust Registry 2021[14] reported the combined use of nasogastric, gastrostomy, and jejunostomy feeding accounted for ~5.1% of all supplements given to pwCF in 2021, compared to 24.2% in 2012[31]. Meanwhile, it would appear that ONS increased significantly over the same period from around 7% in 2012 to ~29% in 2021.[14,31] This would appear to indicate a shift in the type of nutritional support given to pwCF possibly associated with advances in therapy, such as CFTRm.

However, there remains some pwCF that do not have access to CFTRm, due either to it being unsuitable for their genetic mutation or to excess side effects making it unsuitable to continue. For these patients, undernutrition may still be an issue, therefore optimising energy intake remains a priority. For these children we rely on the traditional nutritional interventions, with focus on a calorie-dense diets (through added fats), FSV supplements, ONS and ETF. ONS have been an essential tool to help achieve the energy dense diet required by pwCF.[32]



> There remains some pwCF that do not have access (to the new treatments). For these children we rely on the traditional nutritional interventions.

Weight management

There is presently a lack of evidence for weight management in pwCF. However, consideration now needs to be given to the patient's BMI and when it is a potential cardiovascular risk. For children nutritional counselling has been recommended when the BMI percentile is in the overweight/obese range.[21] While education on appropriate food choices and energy intakes need to be considered on an individual basis. Evidence is still lacking however, as to whether recommendations for the general public can be applied to pwCF, therefore we should remain cautious with our advice.[33]

Body composition

Despite the traditional emphasis on a desired BMI percentile for children, it is well recognised that BMI has its limitations, as it does not assess body composition. Therefore, in those with a high BMI it is unclear whether the weight is from fat free mass (FFM) or fat mass (FM). What we do know is that FM, FFM and fat distribution may influence metabolic and overall health status in pwCF, more so than BMI alone. Consequently, there is an increasing focus on body composition assessments which better reflect health outcomes[34] especially important in view of the overweight/obesity risk seen in pwCF.

Cardiovascular health

In the general population, obesity is associated with worsening cardiovascular outcomes. In CF, similar data is lacking but with increased survival, a higher prevalence of overweight/obesity and the CF "legacy diet" being historically encouraged from an early age, pwCF are at risk of poor cardiovascular health. There are already reports of high cholesterol and systemic hypertension in overweight pwCF compared to those having a healthy weight or being underweight.[16] More attention therefore may need to be given to the type and amount of fats these patients consume. So, while we await studies on the effect of a "heart healthy" diet on outcomes in pwCF, consideration may need to be given to healthier fat options such as encouraging fish or omega-3 fatty acids to potentially hamper inflammation[35], also known to have a protective effect on cardiovascular disease.[36]

Hypertension

As pwCF age and more develop overweight/obesity plus increasing use of CFTRm (with effect on salt homeostasis), hypertension in pwCF is beginning to be noted.[14] More research is required in this area before recommendations can be made but for those pwCF on CFTRm salt intake can probably now be modified. Salt intakes should be considered on an individual basis[37], linked to general population guidelines.

Diabetes

Studies have demonstrated increased insulin resistance in pwCF who are overweight/obese compared to normal weight or underweight.[16,36] As pwCF become more overweight, insulin resistance may become more significant. Treatment of CFD may also be changing with CFTRm. In children with CFD, one study to date has demonstrated improved glycaemic control after commencing Elexacaftor/Tezacaftor/Ivacaftor, with some pwCF reducing or stopping insulin.[38] This intervention may reduce the incidence of CFD in the future, lessening the need for medical interventions through using diet alone.

Cancer

Recent research suggests a 5-fold increase in the risk of colon cancer in pwCF[39]. Whilst the aetiology is not fully understood, diet may be a contributory factor. For example, a low dietary fibre intake has been associated with an increased risk of colon cancer, and research has shown that fibre intakes in children with CF is low.[40,23] Dietary fibre intake in pwCF was never given priority due to the emphasis being put on weight gain, and the low-calorie density associated with high fibre foods. For pwCF, dietary fibre should be recommended in line with the dietary reference intakes for the general population.[27]

Obesity has also been linked to the development of colorectal cancer[41, 42] although this link has yet to be made in pwCF who are obese.

> More research is needed to look at the benefits of individualised healthy eating advice in pwCF who are living longer and presenting with age-associated comorbidities.

Conclusion

PwCF are living longer due to several important factors, most notably NBS and the introduction of CFTRm. This has led to a change in the profile of pwCF who more commonly present with overnutrition. As dietitians and health care professionals working with pwCF, particularly children, we need to adapt our practices to be aligned with these changes, from one of survival at all costs (e.g. BMI, high fat diets) to one of healthy survival, with a focus on a healthy diet and lifestyle. More focus is required on diet quality and healthy eating, plus micronutrient content, sodium intakes and dietary fibre.

It must be remembered however, that for some pwCF for whom CFTRm is not available and who remain undernourished, optimising energy/nutrient intakes in the traditional way will continue to be a priority. Finally, more research is needed to look at the benefits of individualised healthy eating advice in pwCF who are living longer and presenting with age-associated comorbidities.

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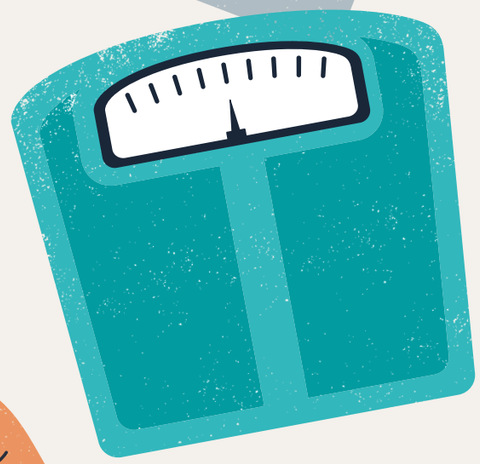

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BY VICTORIA (TORI) MULLEN
Senior Specialist Paediatric Allergy Dietitian
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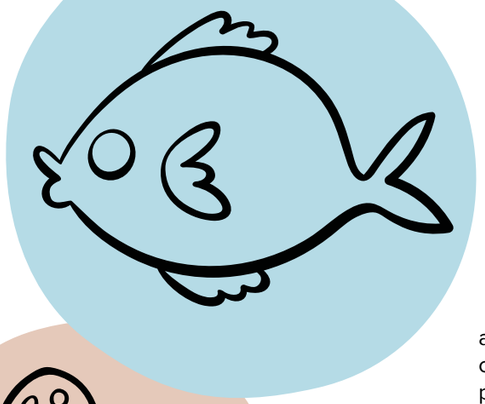
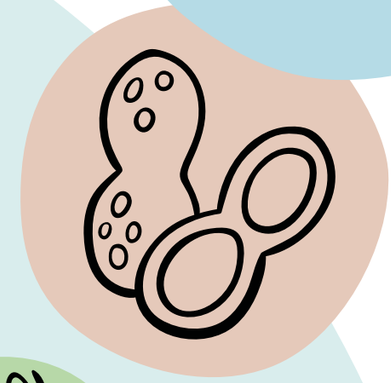
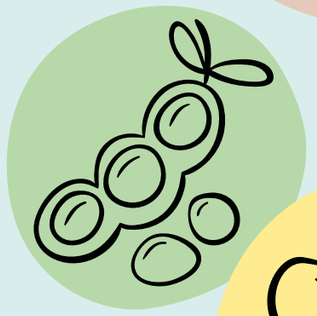
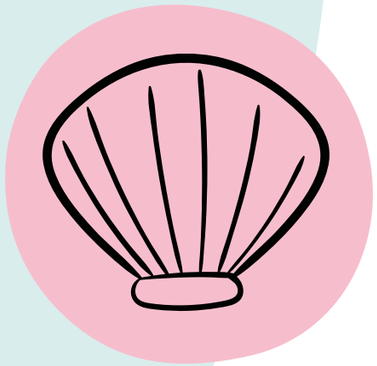
Managing Multiple Food Allergies in children: navigating the challenges









Introduction

Food allergy (FA) is defined as an adverse immune response to a food and is among the most common disorders of early childhood[1]. These can be immunoglobulin E (IgE)-mediated or non-IgE-mediated or a mix of both. IgE reactions typically occur within 2 hours and may involve the gastrointestinal (GI) and respiratory tracts, and skin. In serious cases breathing difficulties and anaphylaxis can occur[2]. Non-IgE allergies typically cause a delayed reaction and can take days to present[2,3]. Non-IgE allergies may cause skin and GI symptoms, but rarely respiratory[4]. In cases of mixed allergy, all systems can be affected. Children with multiple food allergies (MFA) can have a combination of both IgE and non-IgE-mediated reactions.

Prevalence of MFA in children

The prevalence of challenge proven FA in UK children under 5 years of age has been reported to be 4-6%[5]. Cow's milk protein allergy (CMPA) appears to be highest at (2.5%) followed by egg (1.5%) and peanut (1%)[4]. However, there is limited data on the prevalence of MFA in children. One issue is that MFA has no clear definition. In older data, reported rates of MFAs were around 47% in infants[6,7]; mainly milk allergy with other foods. In a recent US population-based survey by Warren and colleagues, they reported 40% of children had MFA, but only 2% were physician confirmed[8]. Additionally, in a Turkish study on children with allergic proctocolitis, they found that 34% were allergic to more than one food[9]. At UHL we consider multiple FA as being allergic to more than 3 staple foods, but others classify according to food type e.g. 4 possible phenotypes for MFA have been described, a milk and egg-dominant group (51%), a seafood-dominant (16%), peanut and tree nut-dominant (28%), and broadly MFA (5%)[8]. Using these criteria may help us better describe prevalence rates.

Causes

Although causes of allergy are not always known, certain factors may increase risk. For example, having a family history of allergic conditions or early-onset moderate to severe eczema are known risks. In terms of MFA, children with early-onset moderate/severe eczema and/or a diagnosis of FA prior to weaning are at higher risk, specifically to peanut and egg[10].

Diagnosis

The first step in the diagnosis of FA is understanding the nature of the reaction, performed by taking a detailed clinical and allergy-focussed history[11]. Here the physician will look for signs of atopy, such as eczema, asthma, and rhinitis, and take a detailed diet history[11]. The EATERS tool was developed to provide a concise way to take an allergy focused history[12]. This assessment tool can also be used in MFA[12].

The EATERS assessment should gather information on the following:

Exposure – consumption of a food allergen

Allergen – commonly known allergen

Timing – between the exposure and symptoms

Environment – where the reaction occurred

Reproducible symptoms – previous or future exposures to an allergen

Symptoms – typical reactions involving multiple organ systems

Tests to support the diagnosis

If the history and clinical presentation are consistent with IgE-mediated FA, the diagnosis can usually be confirmed using skin prick tests (SPT) and/or serum specific IgE (sIgE)[11]. The allergy focused history will help determine which foods to test. In non-IgE-mediated allergy no tests exist, therefore food exclusion followed by reintroduction is required. In children with suspected MFA, SPT/sIgE tests can be performed to the suspected foods[11]. A higher test result correlates to a higher chance of allergy but is not diagnostic.

Diagnostic food challenges- at home and in hospital

The gold standard for the diagnosis of FA is the double-blind, placebo-controlled oral food challenge (DBPCFC), but these are typically reserved for research, due to their complexity and resource demands and rarely needed[11]. For most children open food challenges are used in the supervised clinical setting, when there is concern that reintroduction may cause anaphylaxis[14]. In cases of MFA, several open challenges may be required to confirm or refute an allergy or to determine if the child has outgrown their allergy[11]. At UHL, we provide at least 10 ward-based food challenges per week, some of which will be to multiple foods. For example, we may challenge multiple-tree nut allergy in one biscuit, rather than separately. Re-challenging is especially important for children with MFA to avoid unnecessary lengthy restrictions.

> The EATERS tool was developed to provide a concise way to take an allergy focused history.

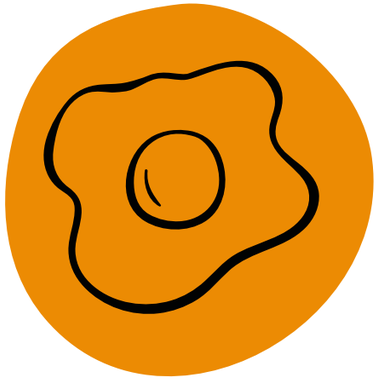
Management of children with MFA

The mainstay of management for MFA is removal of the implicated allergens whilst maintaining adequate nutrition[15]. Managing children with MFA can be complex for families, children and Health Care Professionals. Dietitians will need knowledge to be able to give ideas on recipes, supplements and best substitutes, as well as guidance on how to read product labels; and in those with severe reactions, education on using emergency devices or medications (e.g., self-injectable epinephrine, antihistamines). Support may also be needed on the safe reintroduction of foods.

At UHL, we hold a MFA clinic every other month. Patients are seen by the same allocated doctor, nurse, and dietitian at every visit, helping to build trust, while families are allotted more time e.g., 40-45 minutes instead of the usual 10-20 minutes. This allows the dietitian time for discussions around tolerance/food reintroductions, while the medical doctor focusses on medical management[17].

In breastfed babies with MFA, it is important to consider the mother's diet. If symptoms are occurring while breastfeeding, then advice may be given to mum on food exclusions. Depending on the foods excluded and length of the exclusion period, she may need supplementation of calcium and vitamin D. Foods will be removed for 2 to 4 weeks while monitoring the infant's symptoms then reintroduced to confirm or refute the diagnosis[15]. Breastmilk remains the ideal source of nutrition for infants[15]. When breastmilk is insufficient or not available, then infants should be prescribed an alternative hypoallergenic formula such as an extensively hydrolysed formula (eHF). Only in severe cases, including continued reactions while on eHF, anaphylaxis, and growth faltering with multisystem involvement, is an amino acid formula (AAF) indicated[15]. When families/parents prefer a plant-based formula, then soya-based may be offered from >6 months of age in the UK. While in the recent ESPGHAN position paper on the diagnosis and management of CMPA, hydrolysed rice-based formulas were considered an appropriate plant-based alternative (declaring arsenic content)[16]. These are not yet available in the UK but may be useful for MFA. Store bought plant-based drinks are not recommended as a main drink <2 years as they lack many essential nutrients needed for growth such as iron, protein, and fats. While some fortified plant-based drinks can be used, under dietetic supervision, from the age of 1 year, others may lack essential nutrients. Unfortunately, some of these plant-based drinks can contain allergens making them unsuitable for children with MFA. For these children a hypoallergenic formula is required[15]. Parents should also be advised that foods labelled vegan are not necessarily allergy friendly[21].

> The mainstay of management for MFA is removal of the implicated allergens whilst maintaining adequate nutrition.



When is strict avoidance necessary?

Depending on the initial reaction, strict avoidance of the allergens may be recommended[11]. However, complete avoidance of all forms of the allergen, may not always be necessary. For instance, baked forms of egg and milk (reduced protein allergenicity) appear to be tolerated by up to 70% of children with these allergies[15]. Therefore, incorporating baked milk or egg into the diet is useful, making diets less restrictive and helping with tolerance development. Note, however, that those with MFA tend to develop tolerance later than previously thought, sometimes into the teens rather than early school age[11].

Food reintroductions: a gradual approach

Food introduction ladders are used to systematically introduce allergens back into the diet.[22] At UHL, in those with an IgE milk and egg phenotype, we rarely recommend strict avoidance unless the patient has had a history of anaphylaxis or recent severe reactions to baked forms of the food. Families of children with a non-IgE-mediated MFA will be given guidance on reintroduction directly at diagnosis. All IgE-mediated milk allergic patients, without respiratory symptoms, who have a SPT weal of <8mm are considered for early baked milk reintroduction using the Leicester adapted milk ladder[17]. In high-risk milk allergic children, a clinic-based food challenge to baked milk will be offered. The Leicester ladder starts with a crumb of a malted milk biscuit daily for one week, slowly building up to 1 whole biscuit over 5 weeks. We use this ladder for both IgE and non-IgE MFA. Once tolerated, we advise that patients consume the tolerated quantity of the allergen in their diet, then continueon other foods containing baked milk. We follow the same procedure for other foods e.g., egg and soya.

In MFA, it is important to consider the food reintroduction order as certain allergens limit the diet more than others, but it should be individualised. At our centre, we discuss with the parents the foods they consider most important to reintroduce first, but we may guide them towards getting the most restrictive food in. For example, if a child is milk and egg allergic but is happily taking a milk alternative then I would suggest starting with egg as this allows a greater variety of foods. However, we may delay reintroductions when there is uncontrolled eczema.

Complementary feeding in infants with MFA

For allergic infants, complementary feeding should start between 4 and 6 months, but not before 4 months[15]. BSACI guidelines recommend that infants with moderate to severe eczema and/or FA may benefit from early introduction of egg and then peanut from 4 months[23]. At UHL, patients with moderate/severe eczema and/or FA, start with early weaning (from 4 months). They will start on pureed vegetables and fruits and then progress to blended family foods that contain allergens such as well-cooked egg (hard-boiled), peanut and tree nut spreads e.g peanut/cashew butter or peanut puffs[15]. In cases of MFA, parents may often be anxious to give new foods and the dietitian may need to give extra support.

Implications of the MFA diet

Whilst allergen avoidance is an important part of managing MFA, avoidance of several allergens can have nutritional, developmental, and social implications[11]. One study found that 25% of children with FA consumed less than 67% of the recommended dietary intake for calcium, vitamin D and vitamin E[24]. While another UK survey reported that when >3 foods were eliminated, the weight-for-age of the child was lower compared to when fewer foods were eliminated, with 11% having stunting[25]. For children with MFA, this can be especially problematic, as they may

be avoiding many food groups. At our MFA clinic, we ask for a 5-day food diary which is assessed at each appointment to determine macro and micronutrient intakes. We may request biochemical tests for some nutrients to confirm any deficiencies. Children with MFA may also require multivitamin supplementation depending on the level of dietary restrictions.

Conclusion

MFA has no clear definition and therefore no consensus exists on what optimal management is. It is difficult to determine overall prevalence, but at UHL and other UK centres, >50% of patients appear to have more than one FA. MFA diagnosis is complex and may require exclusion followed by reintroduction of multiple foods. The management is similar to that of individual FA, removing implicated foods but reintroducing may be more difficult to manage due to multiple restrictions. MFA can lead to nutritional deficiencies and growth restriction if not optimally managed, therefore, the role of the dietitian is essential. At UHL, we aim to avoid unnecessarily long restrictions which has long-term implications on the child's nutritional status and quality of life (QoL). We inform parents, at diagnosis, of the journey including on food reintroductions – and only in rare cases do we remove all forms of the food from the child's diet. We use an adapted milk and egg ladder to support structured food reintroductions, which we start as early as possible, using baked forms. MFA requires individualised care to optimise the health of the child while preserving QoL. Further work is needed in this area, including clear definitions and practice guidelines to further optimise care for these children.

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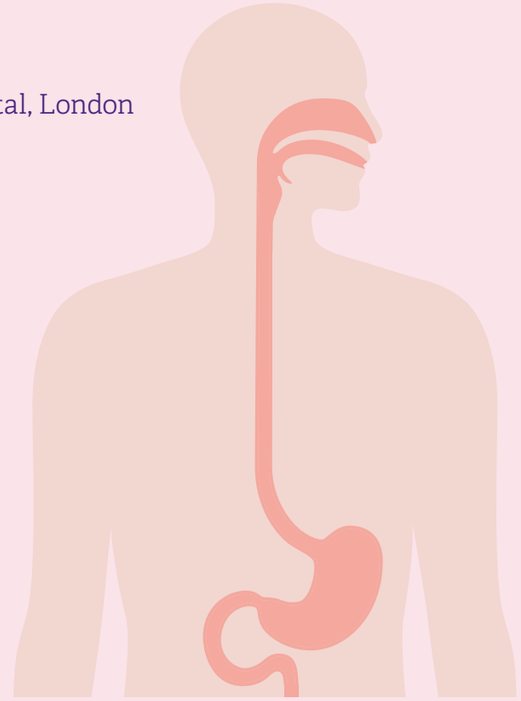


Nutritional management of **Eosinophilic Esophagitis**

What is EoE?

Eosinophilic oesophagitis (EoE) is an allergic condition characterised by the accumulation of eosinophils in the oesophageal tissue[1]. If not properly managed, it can result in oesophageal strictures and significant dysphagia. EoE is a chronic condition that often necessitates ongoing treatment.

How do you diagnose EoE?

The journey to a diagnosis can be lengthy, with evidence suggesting it takes ~3.5 years for a child with EoE symptoms to receive an accurate diagnosis[2]. The lengthy diagnostic process is attributed to the non-specific nature of the symptoms, which can vary with age. In children, a range of symptoms may be observed, including feeding aversion/difficulties, poor growth, vomiting, reflux, abdominal pain or distension, extended mealtimes, avoidance of certain textures (such as meat and bread), and instances of food impaction or difficulty swallowing[3]. In contrast, adults typically experience primary symptoms of dysphagia and food bolus impaction.

> If not properly managed, it can result in oesophageal strictures and significant dysphagia.

EoE is exclusively diagnosed through a biopsy, obtained during an upper gastrointestinal endoscopy. During this procedure, the gastroenterologist takes at least six samples from various locations along the oesophagus. These tissue samples are then analysed to determine the eosinophil count, which involves counting the number of eosinophils present (under the microscope) in a standardised area (approximately 0.3mm^2).[1] When the count exceeds 15 per high power field and is accompanied by specific associated changes, the diagnosis of EoE is confirmed.

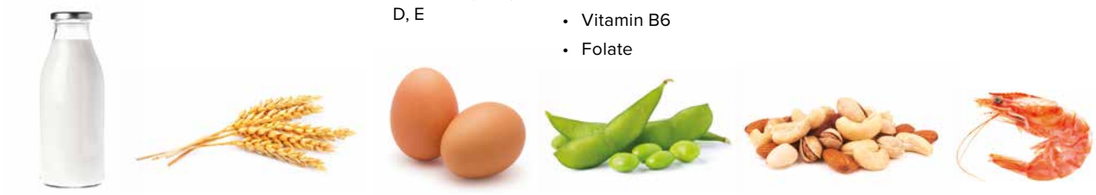
Currently no techniques exist that are less invasive for diagnosing/monitoring EoE. However, researchers are exploring alternative methods like awake nasal endoscopy, although not yet used in clinical practice.

Prevalence

EoE was first identified two decades ago and considered a rare condition. However, over the last decade, its prevalence has significantly increased in Western countries. Globally, prior to 2007, the reported prevalence (using author classified diagnostic criteria) was 15 cases/100,000 while in 2017 it was 63 cases/100,000 (children and adults combined).[4]

Table 1. Macronutrients and micronutrients at risk of deficiency whilst following the 2, 3 or 6FED[8]

Cows Milk	Wheat	Egg	Soya	Nuts	Fish/seafood
• Protein • Calcium • Magnesium • Phosphorus • Iodine • Riboflavin • Pantothenic acid • Vitamin A, B12, D	• Fibre • Zinc • Selenium • Calcium • Iron • Thiamine • Niacin • Riboflavin • Folic Acid	• Protein • Selenium • Iron • Iodine • Folate • Riboflavin • Pantothenic acid • Biotin • Vitamin A, B12, D, E	• Protein • Fibre • Calcium • Phosphorus • Magnesium • Iron • Zinc • Thiamine • Riboflavin • Vitamin B6 • Folate	• Protein • Selenium • Zinc • Magnesium • Phosphorus • Niacin • Vitamin E • Vitamin B6	• Protein • Iodine • Selenium • Boned fish: calcium and phosphorus • Fatty fish: Vitamin A, D, n-3 • PUFA



EoE exhibits a higher prevalence among males and is more frequently observed in individuals with other atopic conditions, such as immediate food allergy, atopic dermatitis, eczema, asthma, and allergic rhinitis[1].

Treatment

The aim of treatment is to reduce oesophageal inflammation, alleviate symptoms, prevent complications, and improve quality of life. There are currently 3 treatment pathways for EoE; pharmacological (proton pump inhibitors or swallowed topical steroids), dietary manipulations or surgical dilatation. It is important to highlight that after any treatment is initiated, repeat endoscopies will be required after 8-12 weeks to evaluate the treatment's effectiveness. Unfortunately, symptoms do not correlate with disease activity, making it incorrect to rely solely on symptoms to monitor EoE.

Regarding dietary management, there are six common food proteins known to trigger EoE: milk, egg, wheat, soya, fish/shellfish, and nuts/seeds (peanut and tree nut). However, not all cases require the elimination of all these foods. Initially, a 6-food exclusion diet (6FED) was recommended as the first-line dietary treatment, showing about 70% histological remission.[5] However, in clinical settings, implementing the 6FED proved challenging due to the high level of commitment required from patients/families, along with multiple repeat endoscopies to identify trigger foods.[1] This approach, known as the "top-down" approach, involved starting many food exclusions and gradually introducing foods between endoscopies.

More practical alternatives, the 4FED (milk, egg, wheat and soya) and 2FED (milk and wheat/egg) were proposed, achieving remission rates of between 50% and 40%, respectively.[1] In most cases, the disease can be effectively managed by excluding just one food, with milk being the most commonly implicated trigger (42%).[6]

Therefore, in clinical practice we use a "step up" approach, where we start with the least number of food exclusions possible, in most cases, 1 to 2; the most effective combinations being milk and wheat (37%) and milk and egg (33%). This approach has been shown to reduce the number of endoscopies by ~20%.[1] When the 2FED is not successful, the next step is a 4FED, then the 6FED. Despite a 90% histological remission rate with elemental diets (amino acid formula), it is only recommended in refractory EoE.[1]

Dietary eliminations for treatment of EoE cannot be guided by allergy tests, as EoE is considered a non-IgE medicated allergy. However, allergy tests may be considered in atopic individuals to assess the risk of it converting to an IgE mediated allergy following exclusion; this has been reported in the literature in a small number of cases.[7]

The role of the dietitian is fundamental in assessing the suitability, feasibility, and type of exclusion diet needed by patients with EoE, as well as monitoring compliance, success and nutritional adequacy. Initially a full dietetic assessment should be completed using the BDA Nutrition and Dietetic Care Process.[8] In depth consideration should be given to the following:

> The role of the dietitian is fundamental in assessing the suitability, feasibility, and type of exclusion diet needed.

- Growth history
- Primary endoscopy (eosinophil counts)
- Diet at diagnosis including current and previous food exclusions
- Allergy focused history (IgE and non-IgE mediated food allergies)
- Feeding behaviour and symptoms
- Medications, micronutrient supplements and oral nutrition supplements (ONS)

Elimination diets for EoE patients come with potential risks related to nutritional adequacy. In children, these diets can lead to feeding difficulties and impaired growth, and weight loss in adults. Elimination diets are inherently restrictive and involve the removal of staple food groups like milk or wheat. Both milk and wheat are important sources of essential nutrients: dairy provides calcium, protein, phosphorus, vitamin B12, and vitamin D, while wheat contributes iron, fibre, and B vitamins. As a result, cutting out these foods increases the risk of nutritional deficiencies.

The potential for nutritional deficiencies becomes even greater with additional dietary restrictions, such as in those with existing food allergies, or when following a vegetarian or vegan diet. In such cases, careful planning and guidance from a registered dietitian is crucial to ensure adequate nutrient intakes are achieved while managing EoE effectively.

Conclusion

As research continues to advance and our understanding of EoE improves, efforts to develop less invasive diagnostic methods and more effective treatments will become a priority. Ultimately, a multidisciplinary approach involving gastroenterologists, allergists, and dietitians is vital in managing EoE effectively, improving the quality of life for those affected, and fostering better outcomes for individuals living with this chronic allergic condition.

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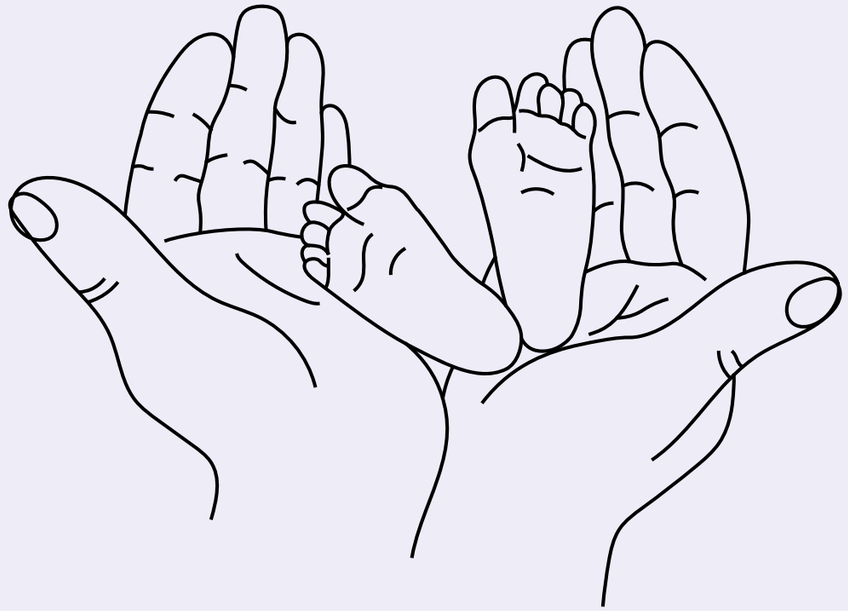
A case of **poor growth** and cow's milk protein allergy



Case presentation

Baby R was born at 34 weeks gestation. He was born via spontaneous vaginal delivery with a birth weight of 2.2kg (50th centile). His birth length was 45cm (50th centile) and his head circumference was 32.5cm (75th centile). During his hospital stay he had episodes of hypoglycaemia, and his blood glucose levels were monitored regularly. He required a short period of Nasogastric (NG) feeding on the Neonatal Unit then progressed onto oral feeds. He was fed with mum's own expressed breastmilk (EBM). He suffered from constipation so had an ultrasound to rule out any other complications. Following the diagnosis of constipation, he was started on laxative therapy. He was discharged at three weeks of age (37 weeks old) on EBM and weighed 2.9kg (50th centile).

At six weeks of age (term corrected) Baby R was transitioned onto standard cow's milk formula. From 10 weeks of age Baby R was unsettled and vomiting often with feeds. Initially Mum added *Carobel* (a thickener) to his feeds without success, then changed to a goat's milk formula. However, Baby R's symptoms did not improve. His weight dropped to the 25th centile (5.9kg) at 13 weeks, and he became orally aversive. He was on a standard infant formula and his intake declined, and he had infrequent bowel movements (every four days). Baby R's health visitor (HV) suspected milk allergy as his older sister had been diagnosed with non-IgE cow's milk allergy (CMA). She contacted the dietetic department for advice on a suitable dairy-free formula for Baby R. The GP prescribed Aptamil Pepti Syneo whilst Baby R waited to be reviewed by the dietetic team.

Baby R also presented with reflux but no medications were offered.

Management & outcomes

With suspicion of non-IgE-mediated CMA[1], baby R was trialled by the GP on Aptamil Pepti Syneo (an extensively hydrolysed whey-based infant formula, with the addition of synbiotics (GOS/FOS* and Bifidobacteria *Breve M-16V*) for 4 weeks, transitioning from his standard infant formula over a few days. He was now 14 weeks of age. After 2 weeks on his new formula his bowel movements became more regular and the laxative (Laxido) therapy was discontinued. His weight slowly improved and at 17 weeks Baby R was almost back on the 50th centile for weight (6.8kg) and 50th centile (61.5cm) for length. He then tracked this trajectory as his intake increased.

Follow-up care

At Baby R's first dietetic review at 6 months of age (5 ½ months corrected) mum reported that he had settled well on his new formula and his vomiting had improved. He had also been weaned onto milk-free solids from around 6 months.

To confirm his diagnosis, an oral food challenge was performed with cow's milk formula, which confirmed his allergy. He again became unsettled and his vomiting increased. Therefore, we proposed that he continue on Aptamil Pepti Syneo until his subsequent review at 9 months.

At his 9-month review Baby R was rechallenged with dairy using the milk ladder. Again, mum reported that he was unsettled, and his feed volume reduced. The reintroduction of dairy was therefore discontinued, and Baby R was not challenged again until 11 months of age. This challenge was successful with Baby R progressing through the milk ladder without issues, and at 13-months of age he was considered tolerant to dairy/cow's milk which had been fully reintroduced back into his diet.

Discussion

Baby R's symptoms were only detected at home when the HV and mum made the connection, knowing his sibling had CMA. This is not uncommon. Sladkevicuis et al (2010) reported that it takes up to 2 months for infants with suspected CMA before they receive a diagnosis[2].

Once a diagnosis is agreed then it is important that the correct type of formula is initiated when not breastfed. In this case Baby R received goats' milk which is not recommended in infants with a diagnosis of CMA. Only hypoallergenic formula should be prescribed which can be either Amino Acid-based Formula (AAF) or Extensively Hydrolysed Formula (eHF). However, in the majority of cases an eHF is considered first line. In infants with poor growth, it's been reported to take around 2-4 weeks on a cow's milk elimination diet before you see symptom resolution[3]. Baby R improved within 4-weeks in both symptoms and growth.

Baby R accepted Aptamil Pepti Syneo after only a few days increasing the volume (titrated) overtime. The addition of pre ("a substrate that is selectively utilized by host microorganisms conferring a health benefit")[4] and probiotics ("live microorganisms which when administered in adequate amounts confer a health benefit on the host")[5] may have helped with his constipation which resolved after two weeks. In fact, data suggests that the Syneo blend may reduce constipation [6,7]. Hubbard and colleagues (2022) reported that 14% of infants had an improvement in constipation when receiving Aptamil Pepti Syneo[6,7]. Baby R's mum was especially happy that he could stop his laxative therapy as his bowels now opened regularly. This could potentially be a cost-effective benefit of using this type of formula, although data is needed to confirm this.

Finally, Baby R became tolerant to dairy and milk by 13 months of age which is similar to what has been reported in other studies. In the EuroPrevall study for example, all infants with non-IgE-mediated CMA became tolerant to cow's milk at 1 year from diagnosis, similar to our finding here[8].

Conclusion

Baby R was born 34 weeks gestation with constipation, reflux and a late diagnosis for CMA (dietetic diagnosis at 6 months of age). He had symptoms of vomiting, oral aversion and poor weight gain/growth as well as constipation (treated with laxatives). Following initiation of Aptamil Pepti Syneo he had completed symptom resolution as well as catch up growth within ~4 weeks with normalisation of stools after just 2 weeks, which meant he could stop his laxative therapy. We believe that the inclusion of synbiotics in Aptamil Pepti Syneo may have contributed to the improvement in his bowel movements and may be an additional benefit when using this type of formula.

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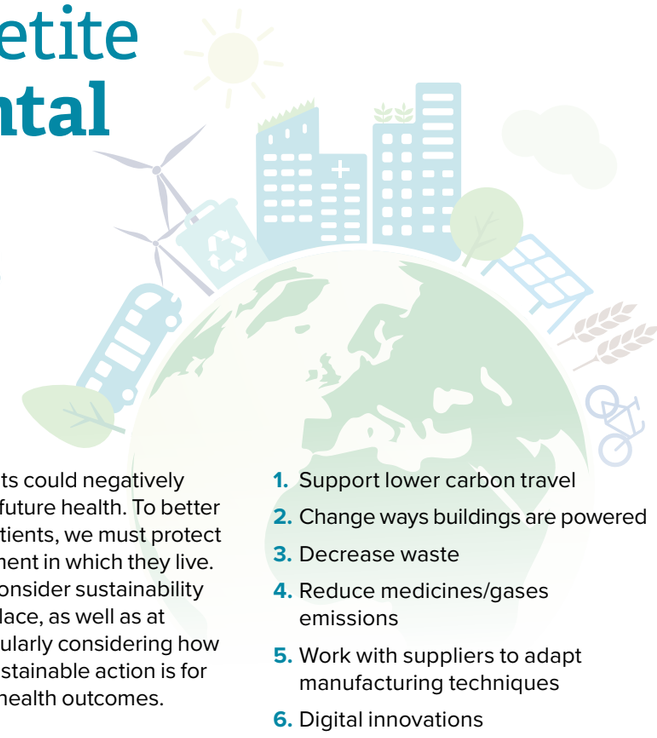
*GOS, galacto-oligosaccharide; FOS, fructo-oligosaccharide.

Building an appetite for **Environmental Sustainability** within dietetics



WHY DOES SUSTAINABILITY IN HEALTHCARE MATTER?

Environmental sustainability is a vision to safeguard our fragile planet and address the urgent climate crisis. There has never been a more poignant time to consider sustainability; as I write this article, heatwaves sweep southern Europe and the USA, and wildfires blaze in Greece, bringing home the need for urgent action.

The Lancet estimated that healthcare was responsible for 1-5% of the total global environmental impact[1]. While the NGO *Healthcare Without Harm* suggested that if healthcare were a country, it would be the fifth-largest emitter of greenhouse gases[2]. Why does this matter? The very healthcare we are providing to our patients could negatively impact their future health. To better serve our patients, we must protect the environment in which they live. We should consider sustainability in the workplace, as well as at home, particularly considering how important sustainable action is for longer term health outcomes.

I'M AIMING TO BECOME MORE SUSTAINABLE... WHERE DO I START?

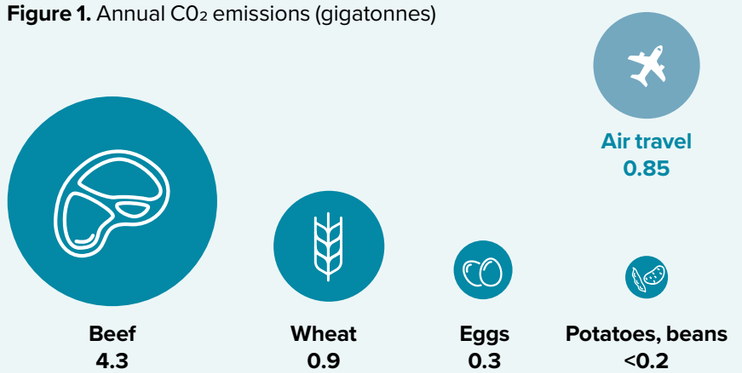
The NHS is the first healthcare service to commit to 'net zero' carbon emissions by 2045. Therefore, NHS staff can be empowered to make system-wide changes to improve sustainability. They suggested 6 ways to achieve this ambitious target:

1. Support lower carbon travel
2. Change ways buildings are powered
3. Decrease waste
4. Reduce medicines/gases emissions
5. Work with suppliers to adapt manufacturing techniques
6. Digital innovations

Diet has a huge impact on our lifetime carbon footprint; beef emits 31 times more CO_2 per calorie compared to tofu and is responsible for 5 times as much CO_2 as air travel (Fig 1). The British Dietetic Association has been instrumental in promoting sustainable diets, which we should all be conscious of, when advising patients. But this is just one area in which dietitians can impact sustainability! Other ideas include:

- Using public transport between patient homes/clinics
- Considering virtual appointments to reduce patient travel
- Considering digital alternatives to current processes
- Lobbying for improved visibility on carbon footprint of feeds and feeding equipment
- Factoring sustainability into feed prescription requests, enteral feed equipment or contract choices
- Supporting industry to develop greener manufacturing processes for feeds and equipment

Figure 1. Annual CO_2 emissions (gigatonnes)



Adapted from The Economist Oct 2, 2021

No change is too small. For example, moving from paper to electronic food record charts, considering powdered feeds over ready-to-use (which often have a lower carbon footprint due to a longer shelf-life, reduced transport emissions, and less plastic packaging) or reviewing your single-use plastic enteral feed equipment policy? Improvement is a collaborative effort, so getting sustainability onto your department's agenda is a great start!

I WANT TO START MAKING MY PRACTICE MORE SUSTAINABLE... WHAT AND HOW?

Quality Improvement (QI) is a systematic approach to designing, testing, and implementing changes. How many times have you heard: '*I know it's not perfect, but that's just the way we do it ...*'? QI is all about challenging the status-quo, driving system-wide change to support improvement in patient care. Marrying sustainability with QI approaches (termed SusQI) is the newer horizon for QI. When thinking about your own projects, reaching out to your trust's QI team for support or attending QI training is a fantastic place to start.

Here are a few tips to start:

1. Start by working out what the problem is, and what you are aiming to achieve. The Acronym SMART (Specific, Measurable, Achievable, Realistic/Relevant, Timebound) is really helpful. For example, rewrite 'making enteral feeding more sustainable' into 'this project aims to reduce single-use plastic enteral feeding equipment in long-term PEG patients by 30%, by December 2024.'
2. Measurement is crucial – otherwise how do we know if the change we implement constitutes an improvement? Consider the 'triple bottom line' – where each project is measured in terms of people (patient outcomes, staffing requirements), profit (financial impact) and planet (environmental impact) (Fig 2).
3. Start small, experiment often and build your QI project gradually. We recommend using iterative '*Plan, Do, Study, Act*' (PDSA) cycles. Plan one small 'experiment' at a time, carry it out, then study if it worked using your measurement tool. Finally, implement the change if it worked, and ditch it if it didn't! Experimenting on a small scale makes it OK to fail, provided we learn.

RESULTS FROM OTHER SUSTAINABLE PROJECTS AT GOSH

In 2021, GOSH declared a Climate and Health Emergency. Earlier this year, I moved from my role as a clinical dietitian to the QI team. This is an exciting area in which AHPs can develop skills in mentoring, facilitation, project and change management – skills we develop through our dietetic practice. One of the main focusses of my current role, is around integrating sustainability into our trust-wide QI projects.

A project doesn't have to have sustainability as a primary outcome to show great environmental benefits. Before I moved roles, I carried out just such a project on the cardiac ICU, around standardisation of enteral feeding. The primary goal was to reduce rates of necrotising enterocolitis, but I wanted to pull in the 'triple bottom line' and integrate sustainability into the secondary outcomes. The ward nursing team highlighted challenges around ordering '*out of hours*' feeds, and high volumes of wastage. This seemed like a great place to start! We changed to powdered feeds and they were made up in bulk for the whole ward, rather than individually for each patient; auditing pre- and post- change. From this one small change, we saw a **62% reduction** in feed volume ordered from the special feed unit (2.2 swimming pools of milk annually!) and a **49% reduction** in single use plastic bottles (1 blue whale of plastic!). This was a very encouraging start on my journey to improving environmental sustainability in my own practice.

Figure 2. The triple bottom line

LET'S GET GOING!

Becoming sustainable at work has never been so important, but it can feel like a minefield. Start slowly, and never think an idea is too small – subscribing to the Greener NHS bulletin or visiting the Centre for Sustainable Healthcare website is a manageable place to start. Because small changes over time can change the world...and every small change counts!

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UKKIDSNUTRITION

Research and understanding of Oligosaccharides in infant health with a focus on scGOS/lcFOS

Breastfeeding is always best, and breastmilk has long been established as the gold standard for infant nutrition due to its unique composition, providing essential nutrients and bioactive components that support infant growth and development, gut microbiome, and immunity. Infant formula should be recommended (by a Health Care Professional) when breastfeeding is not available/possible.

Among these bioactive components are the human milk oligosaccharides (HMOs), the 3rd largest solid component of human milk after lactose and lipids. They are one of the two main carbohydrates found in breast milk (the other being lactose) and are predominantly non-digestible, or minimally digestible (1-2%)[1].

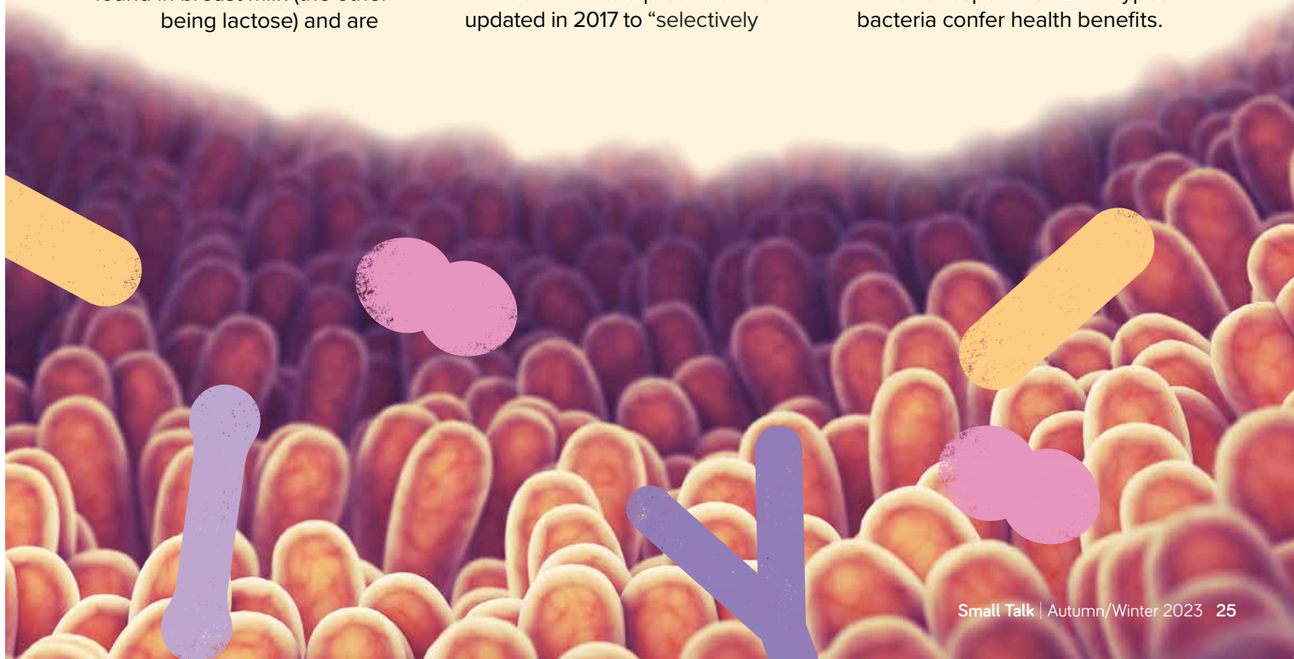
HMOs are complex glycans with a lactose backbone and a chain length of 5 monosaccharide units[2], while most other oligosaccharides have between 5-10 monosaccharide units. To date, researchers have identified 200 different HMOs.

As these non-digestible components reach the large gut, they are selectively fermented by healthy bacterial species particularly Bifidobacteria and Lactobacillus[3-4] that reside in the gut. HMOs are anti-viral and pathogen-binding prebiotics that improve gut barrier function and play a role in maintaining immune homeostasis[5].

The definition of a prebiotic was updated in 2017 to "selectively fermented ingredients that allow specific changes, both in the composition and/or activity in the gastrointestinal microflora, that confer benefits upon host well-being and health"[6].

Following birth, Bifidobacteria typically becomes the dominant species in the colon of breastfed infants, with "infant type" Bifidobacteria specifically abundant[7]. It was first isolated from the faeces of breastfed infants in 1899 by Tissier and has since been found in the oral cavity and the gastrointestinal tract of various mammals[8].

Lactobacillus play a vital role in balancing gut barrier integrity, mucosal barrier defence, and host immune responses[9]. Both types of bacteria confer health benefits.



The link between HMOs and other oligosaccharides

A unique prebiotic oligosaccharide mixture of short-chain (sc) galactooligosaccharides (scGOS) and long-chain (lc) fructooligosaccharides (lcFOS) was developed by Nutricia Research in 1994. The composition and ratio (90%:10%) of the mixture of scGOS to lcFOS was specifically selected to closely resemble the molecular size composition of HMOs[10].

FOS, derived from chicory inulin, is a plant-based prebiotic that occurs naturally in onion, garlic, asparagus, banana, and artichoke, among many others[11]. Dietary FOS are not hydrolysed by small intestinal glycosidases so reach the caecum structurally unchanged[11].

In contrast, GOS is obtained from lactose, a natural sugar in milk, at a degree of polymerization of 3-8 and is animal-based[12].

ScGOS and lcFOS are among the most studied oligosaccharides in infant formula[3]. Just like HMOs, they have been shown to increase the production of short-chain fatty acids (SCFAs), including acetate, propionate, and butyrate, which contribute to a reduced colonic pH[11, 13]. This reduction in colonic pH in turn fosters the growth of beneficial Lactobacillus and Bifidobacteria while inhibiting the growth of undesirable bacteria (e.g. E. coli and enterococci), promoting gut health[2, 3, 8]. Moreover, SCFAs act as a vital energy source for the colonocytes. They also help regulate the immune system, reducing inflammation within the gut[13, 14].

The European Commission agreed to a concentration of up to 0.8g/100ml scGOS/lcFOS in infant formula at a ratio of 9:1. This is also included in the European Directive 2016/127/EC on infant and follow-on formulae[15].

Table 1. Health benefits of using formula with scGOS/lcFOS (9:1 ratio) for term and preterm infants

Health Benefits	References
Increased levels of bifidobacteria in stool and reduced stool pH	14, 16, 17, 18, 19
Reduced incidence of gastroenteritis with less antibiotic use	20
Reduced infectious episodes, particularly respiratory infections	20, 21
Softened stools, improve stool viscosity and defecation frequency	22, 23, 24, 25

Benefits of scGOS and lcFOS when added to infant formula

scGOS/lcFOS at 0.8g/100ml (9:1 ratio) has been shown to improve stool consistency, frequency, gut microbiota, and immune function in healthy term infants[14, 16-19], resembling that of breastfed infants[3, 14].

In one study, comparing 0.4g/100ml to 0.8g/100ml scGOS/lcFOS (9:1) versus formula without prebiotics, they found that the bacterial composition of stools in those taking 0.8g/100ml was closer to stools of the breastfed (control) group[14]. This was one of the reasons for choosing this quantity and combination.

Other studies have demonstrated similar benefits of this combination (scGOS/lcFOS) in fermented formula[3].

> SCFAs act as a vital energy source, helping regulate the immune system, reducing inflammation within the gut.

Increased levels of Bifidobacteria in the stools

Several studies have demonstrated increased levels of Bifidobacteria in stools of infants fed scGOS/lcFOS (9:1)[14, 16-19]. Knol et al (2005) found significantly higher levels of Bifidobacteria stools of infants fed formula with 0.8g/100ml scGOS/lcFOS (9:1) compared to a control group without scGOS/lcFOS[18]. After 6 weeks, changes in SCFA and pH of stools in scGOS/lcFOS-fed group were more similar to that observed in the breastfed group compared to the control.

Reduced incidence of gastroenteritis, with less antibiotic

A randomised controlled trial (RCT) with 342 term infants revealed that the addition of scGOS/lcFOS (9:1) at a lower concentration of 0.4g/100ml of formula, reduced the incidence of gastroenteritis in the supplemented group compared to the control. Additionally, the supplemented group required fewer antibiotics, suggesting potential protection against infections in these infants[20].

Reduced infectious episodes, particularly respiratory infections

Another prospective RCT by Arslanoglu et al[21] in healthy, term infants showed that 0.8g/100ml scGOS/lcFOS (9:1) when added to a hypoallergenic formula resulted in fewer episodes of upper respiratory tract infections with less antibiotic use.

Softened stools and improved stool viscosity and defecation frequency

Several studies have demonstrated softer stools and improved defecation frequency, in both term and preterm infants using the 0.8g/100ml scGOS/lcFOS (9:1) mix[22-25].

Conclusion

Human milks' unique composition, with essential nutrients and bioactive components such as HMOs, make it the ideal nutrition for infants or the "gold standard" of infant nutrition. HMOs provide many benefits to the infant including anti-viral and pathogen-binding, improve gut barrier function and play a role in maintaining immune homeostasis. When breastfeeding is not possible or available, a replacement infant formula is required. Studies have demonstrated that formula containing 0.8g/100ml scGOS/lcFOS (9:1), provides numerous clinical benefits to term and preterm infants including reduced gastroenteritis and respiratory infections, less antibiotic use and promotes softer, more frequent stools.

Thus, scGOS/lcFOS (9:1) at 0.8g/100ml represents a valuable innovation to support gut health and immunity in formula-fed infants, when breastmilk is not available.

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NUTRICIA ACADEMY
Feed your mind

Ask The Expert

Jacqui Lowdon

Do you have a question for our expert? Ask your question to Jacqui **resourcecentre@nutricia.com** and your question might be answered in our next edition!

How can dietitians facilitate better self-management in children with Cystic Fibrosis Diabetes (CFD) so that they are better able to cope later in life?

With the improvement in survival of Cystic Fibrosis (CF) patients, there has unfortunately been an increase in co-morbidities, with CFD being the most common.

CFD shares some features of Type 1 and Type 2 diabetes mellitus (T1/2 DM) but there are some important pathophysiological differences requiring different approaches to management. For example, the first symptom may be a reduction in weight and lung function while reactive hypoglycaemia is not uncommon. Clinical guidelines exist in the UK and internationally (Cystic Fibrosis Trust 2022), with a strong focus on nutritional status. Within these guidelines, the importance of patient knowledge on self-management of their CFD is emphasised.

So, what can we do to help facilitate better self-management in our children with CFD? Well, the guidelines also recommend collaborative multidisciplinary team (MDT) approaches to care; understanding who does what, and the role of each team member. Having these (roles) defined early on will help.

The patient's voice is also particularly important. This should include their experiences of self-management, and the challenges they face (Brunzell et al. 2015).

A recent paper by Collins et al 2023 suggests that the management of CFD is challenging and, although people with CFD experience many adaptation and management processes similar to people with T1 DM, they struggle with the additional complexity of balancing CF and CFD. The provision of appropriate education, support and person-centred care is therefore important.

We need to ensure that our patient cohort receive sufficient support from us, highlighting any gaps in knowledge and offering information on how to make appropriate self-management decisions.

Written, take-away information and/or online resources about CFD and its management should be made available at diagnosis. A good example is the CF charities such as the CF Trust, which have resources written by dietitians along with online discussion groups for peer support.

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Do you find that lack of resources in dietetics is impacting patient care? And do you have any simple solutions you use in practice?

Lack of resources will always impact patient care but there are no simple solutions. I believe the initiation of the Long-Term Workforce Plan published by the NHS in England and backed by the government, will help. This will set out how the NHS will tackle existing vacancies and how it plans to meet the needs of a growing and ageing population, over the next 15 years. It will also provide ideas on new ways of working. It aims to improve patient care, plan for a sustainable workforce, focus on retaining staff and improve the use of technology. Emphasis is on train, retain and reform! This new announcement will hopefully also support the BDA to develop apprenticeships and advanced practice, as well as achieving independent prescribing. And it has already started with the first dietetic apprentice course, offered by Coventry University. It involves a mix of on and off-the-job training for individuals whilst employed by their NHS trust and is run over 2 years.

Key benefits of the apprentice scheme include:

1. Reduced turnover

2. Upskilled work force

3. Sustainable recruitment tool

The previous NHS Long Term Plan (2019) highlighted that the NHS's greatest strength is its staff. In 2020, the NHS People Plan highlighted that developing skills and increasing capabilities improved morale and supported career progression. The publication of the Long-Term Workforce Plan is therefore one of the most seminal moments in the NHS history and aims to future proof staffing and improve patient care.

www.england.nhs.uk/publication/nhs-long-term-workforce-plan

Up2Date

by **Theresa Cole**

An ESPGHAN position paper on the diagnosis, management and prevention of cow's milk allergy.

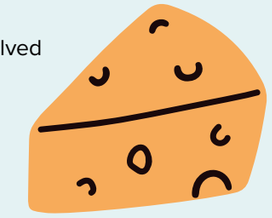
Vandenplas Y et al. 2023

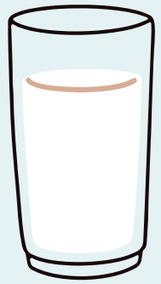
The European Society of Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) published guidelines on the *diagnostic approach and management of cow's milk allergy* (CMA) back in 2012. A new position paper has been accepted for publication in the JPGN, which includes additions such as *prevention, food reintroduction/milk ladder* and *feed choice*.

Some of the key statements that were agreed:

- Overdiagnosis of CMA occurs more commonly than underdiagnosis, but both have negative long-term health consequences for the child. Food challenges/elimination diets help overcome misdiagnosis.
- The prevalence of genuine CMA is <1%.
- CMA can exist in exclusively breastfed infants but is uncommon. An example is food protein induced allergic proctocolitis (FPIAP).
- Maternal cow's milk (CM) exclusion for 2-4 weeks whilst continuing breastfeeding may be considered when CMA is suspected in a breastfed baby. In such cases, mum's diet should be monitored, and may require vitamin D and calcium supplements.
- Non-IgE CMA can manifest as different gastrointestinal (GI) disorders such as FPIAP, food protein induced enterocolitis syndrome (FPIES), eosinophilic disorders. Family history of allergy, and involvement of several organ systems (digestive, cutaneous, respiratory), increases its likelihood.
- In those not responding to conventional therapies for functional GI disorders (FGIDs), colic and GORD, CMA could be considered with a trial on a CM elimination diet for 2-4 weeks; followed by oral food challenge (OFC) or food reintroduction.
- In formula fed infants, a CM derived extensively hydrolysed formula (eHF) either whey or casein, is the first choice; Hydrolysed rice-based formula can also be used. All should comply with the food for special medical purposes (FSMP) guidance, and have been tested (and confirmed safe) in randomised controlled trials.
- Soy-based infant formula is not recommended as first line in CMA, but can be considered for economic, cultural and palatability reasons.
- When CMA presents with severe diarrhoea and/or severe malnutrition, the transient use of a formula without lactose for 2-4 weeks may be preferred.
- There is insufficient evidence to recommend amino-acid based formula (AAF) as first choice in the diagnostic elimination – AAF are reserved for severe cases of CMA and/or those with severe malnutrition.
- The Atopic patch test, IgE-antibodies, component resolved diagnostics, or the basophil activation test (BAT) are not recommended in routine practice to support the diagnosis.
- The open OFC is considered more feasible and practical in the clinical setting and is sufficient to confirm diagnosis or tolerance in CMA.
- The milk ladder (adapted to local dietary habits) is recommended for food reintroductions at home; home challenges are safe for non-IgE-mediated CMA.
- Changes in stool characteristics or occasional hematochezia (blood in stool) is common in infants and should not be considered diagnostic of CMA. In infants with constipation not responding to laxatives (in optimal dosages), an elimination diet for 2-4 weeks followed by food reintroduction can be considered.
- The Cow's Milk Related Symptom Score (CoMiSS) tool can be used to support the diagnosis of CMA but is not a diagnostic tool.
- Complementary feeding in CMA infants should start at the same age as non-allergic infants and follow the same recommendations, excluding only CM/dairy.
- There is (currently) insufficient evidence to recommend "biotics" [pro-, pre- or synbiotics] as part of the therapeutic elimination diet in CMA and no evidence in prevention.

This position paper contains sections on nutrition, growth, cost and quality of life.





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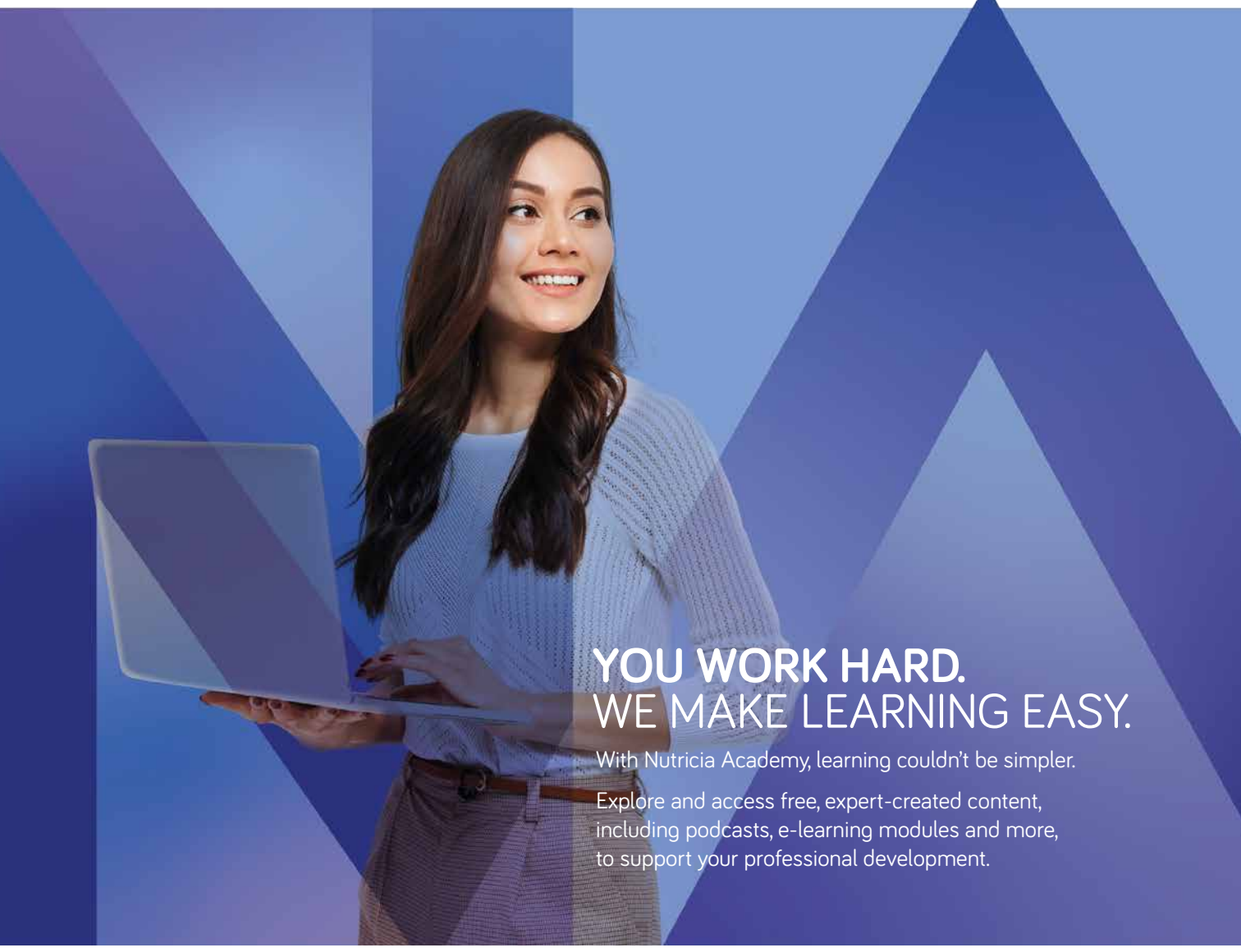

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