Food Allergies and Eating Disorders: A Review of Current Literature

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1. INTRODUCTION

CASE REPORT

S.B, age 22, reported gluten intolerance in the absence of celiac disease (CD) upon admission to a residential eating disorder facility. As a child, S.B. reportedly underwent a food elimination diet as directed by her pediatrician due to gastrointestinal complaints. In the reintroduction phase of the elimination diet, S.B. found that gluten triggered gastrointestinal symptoms. These symptoms resolved after removing gluten from diet. S.B. later developed anorexia nervosa- restricting type in her teen years. S.B.'s residential treatment team reported difficulty in separating the reported food intolerance from her eating disorder (ED). Since the timeline of S.B's gluten intolerance symptoms preceded the onset the ED, as evidenced by patient medical records, the treatment team decided that the reported gluten intolerance was not part of S.B's ED symptomology. This case resulted in robust communication between treatment providers and brought to question the clinical course of action for handling self-reported food allergies or intolerances in the context of ED treatment.

The case of S.B is not a stand-alone observation. Observation at residential ED treatment center showed nearly 30% of the patient population self-reporting food allergies or intolerances at the time of the registered dietitian (RD) intake interview upon admission. Gastrointestinal (GI) stress elicited by lactose-intolerance, dairy allergy, or gluten-intolerance were the most commonly cited issues. On average, however, only 1 out of 14 patients had clinical evidence of a true food allergy (FA). This mirrors observation beyond the ED population. Currently, about 20-35% of the western population self-reports adverse reactions to one or more food items, but true FA is the culprit for only about 2-10% of the population (Bischoff & Crowe, 2005; Lied et al, 2011; Young, 1994). The discrepancy between self-reported data versus actual diagnostic data brings to question the role food allergies play in individuals with EDs. Is report of the FA a manifestation of ED pathology or is there an underlying mechanism common to ED patients that elicits an allergic response to food? The former possibility is speculated by a number of popular media sources that claim FAs are the new ED, including *The Times* (2000), *Forbes Magazine* (2014), *Psychology Today* (2015), *Huffington Post* (2009), and *Cosmopolitan* (2014). A possible reason for the rise in FA popularity, as evidenced by the 48% global increase in the sale of gluten free food items from 2013 to 2016, is that they are used as an excuse for food restriction (Forbes). Moreover, alternative health media and celebrity culture perpetuate the erroneous belief that certain food items, particularly gluten, are unhealthy, "allergenic", and that avoiding these items can aid in weight loss and eliminate a number of symptoms (DiGiorgio, 2015, Finlay, 2009;). We know that the media plays a large role in how individuals susceptible to EDs value their bodies and food choices. The media's influence on the popularity of food allergies has likely affected the ED population. To determine the relationship between FAs and EDs, it is

important that we first discuss the general signs and symptoms of both pathologies. Next, we will discuss how GI distress presents in ED patients versus in those with true food allergies—demonstrating how GI symptoms in ED patients and FAs can be easily confused. From there, we will discuss what the current literature says about FAs and EDs as comorbidities. Finally, we will discuss our conclusions and provide treatment recommendations to ED professionals. The hope is that this investigation will help professionals understand how to best diagnose and treat a) ED patients with reported FAs or b) individuals with FAs who may develop an ED.

EATING DISORDERS

Eating disorders are mental illnesses characterized by persistent feeding disturbances that significantly alter the consumption or absorption of food (Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-V)). Inadequate feeding over a period of time can lead to severe malnutrition that can significantly impair physical and mental health (DSM-V). The DSM-V characterizes and provides diagnostic criteria for anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), avoidant/restrictive food intake disorder (ARFID), Other Specified Feeding or Eating Disorder (OSFED), pica, and rumination disorder.

AN is characterized by persistent energy intake restriction in effort to maintain a bodyweight below the minimum weight for age, height, sex, and developmental trajectory (DSM-V). This behavior is coupled with an intense fear of gaining weight or of becoming fat that persists even with weight loss. Individuals with AN use perceived weight and shape in order to evaluate their worth and self-esteem. Weight loss is viewed as an impressive feat requiring self-discipline, while weight gain is viewed as a personal failure (DSM-V). Individuals with AN are often in denial of the seriousness of their low body weight or of the physiological implications of their disordered behaviors. One physiological consequence of AN, which is used as a diagnostic criteria, is the absence of at least three consecutive menstrual cycles. Other physiological consequences include protein energy malnutrition, cardiac abnormalities, electrolyte disturbances, osteoporosis, and impaired gastrointestinal motility. There are two types of AN: (1) restricting type, with no regular binge/purge cycle, and (2) binge-eating/purging type, with regular binge/purge cycles (George, 2003). The lifetime prevalence of AN is 0.3%, but this figure is likely higher due to recent adjustments in DSM-V criteria (Hoek, 2006; Mustelin, 2016). AN is the deadliest of all mental illnesses with a mortality rate with 5.1 deaths per 1000 person-years—One in 5 of these deaths are due to suicide (Smink, 2012).

BN is characterized by three main features according to the DSM-V: (1) recurrent episodes of binge eating, (2) recurrent inappropriate compensatory behaviors to prevent weight gain, and (3) body shape and weight as influencers of self-esteem or self-worth. Binge eating episodes are defined as eating a large amount of food in a discrete period of time, and are accompanied by an out of control feeling, where it is nearly impossible to refrain from

eating once started. Binges are typically comprised of foods that are usually restricted (i.e. sweet or high fat foods). Due to the high level of shame associated with this behavior, binges usually occur in secrecy. Triggers for binges include interpersonal stressors, dietary restriction, negative body image, and boredom. In BN, binges are coupled with compensatory behaviors in order to prevent weight gain, otherwise known as 'purging behaviors'. Vomiting is the most common and most accessible purging behavior of BN. It acts to provide relief from the physical and psychological discomfort from the binge. Other purging behaviors include misuse of laxatives, diuretics, enemas, thyroid hormone, insulin, as well as prolonged fasting and excessive exercise. Individuals with BN are similar to those with AN in their intense fear of weight gain and bodily dissatisfaction. To receive a BN diagnosis, binging and purging behaviors must occur at least once per week for three months (DSM-V). The lifetime prevalence of BN is 1%, and there is some evidence that this rate may be decreasing over time (Smink, 2012).

BED is characterized by recurrent episodes of binge eating at least once per week for 3 months. However, unlike BN, BED does not involve the use of inappropriate compensatory behaviors. In contrast to AN and BN, BED occurs in normal-weight, overweight, and obese individuals. However, BED is distinct from obesity. Not all those who are obese engage in binge eating, yet the obese individuals who have BED experience greater functional impairment, lower quality of life, more subjective distress, greater psychiatric comorbidity, and greater calorie consumption (DSM-V).

ARFID is an eating disturbance that usually first presents in childhood. It is mainly characterized by avoidance or restriction of food intake that manifests in the failure to meet adequate nutrition and energy requirements through oral food intake (DSM-V). Reasons for food avoidance may be based on the sensory characteristic of food (i.e. appearance, color, smell, texture, temperature, taste). Other contributors to food avoidance include generalized anxiety, gastrointestinal symptoms, a history of vomiting/choking, and *food allergies* (Fisher, 2014). AFRID distinguishes itself from the other EDs by a lack of disturbance around weight and body image (Fisher, 2014).

OSFED includes a variety of feeding disorders that cause significant social, psychological, and occupational impairment, but do not meet the full diagnostic criteria for the EDs presented in the DSM-V. Examples of OSFED include atypical anorexia, where all the criteria for AN are met besides BMI; bulimia nervosa of low frequency or limited duration; BED of low frequency or limited duration; purging disorder that occurs in the absence of binge eating; and night eating syndrome (DSM-V). Other less prevalent EDs include pica, involving the ingestion of inedible objects, and rumination disorder, involving re-ingesting regurgitated food.

FOOD ALLERGIES AND INTOLERANCES

The public, patients, doctors, and other health professionals frequently confuse non-allergic food reactions with a true FA (Turnbull, 2014). A *food allergy*, as defined by the National Institute of Allergy and Infection Disease, is an adverse immune-mediated response that is reproducible upon exposure to a given food and absent during avoidance, while a *food intolerance* is a non-specific, non-immune response to certain foods due to metabolic or undefined mechanisms (Sicherer & Sampson, 2014; Turnbull, 2014). The most allergenic food substances worldwide include eggs, milk, fish, shellfish, peanut, soybean, tree nuts, and wheat, and they account for 85% of all food allergies (Björkstén, 2004; Turnbull, 2014). Although a wide number of diseases fall under the umbrella of food allergies, this paper will focus on how food allergies manifest in the gastrointestinal tract (Sicherer & Sampson, 2014).

The current incidence of food allergies is under speculation. 20-35% of the western population self-reports adverse reactions to one or more food items. However, likely a fraction of those events is due to actual food allergies (Bischoff & Crowe, 2005; Lied, 2011). Recent journal articles estimate that "food allergy affects more than 1-2% but less than 10% of the population" (Young, 1994). Self-reported data from the National Health and Nutrition Examination Survey (NHANES) from 2007-2010 with 20,686 US participants mirrors that approximation: 8.96% of adults and 6.53% of children reporting FA. Objective insight from oral food challenge studies estimate FA rates at ~5% for adults, ~8% for children, and ~2% for peanut allergy alone (Turnbull, 2014). Some research indicates that less than 50% of reported reactions are confirmed (Young, 1994). The discrepancy between self-reported and confirmed FA rates may have to do with the understanding of "food allergy" by the lay public (Björkstén, 2004). Many view perceived food intolerance as a "food allergy", when only a small fraction of the world population experiences immune-mediated reactions to food. Since there are few epidemiological studies that use the gold standard double blind placebo controlled food challenge (DBPCFC)—essential for objective assessment—the true prevalence of food allergies remains unknown (Turnbull, 2014; Young; 1994)

A FA develops due to an abnormal response of the immune system to ingested antigens (Sampson, 2004). Each day, various food particles and pathogens bombard the physical barrier of our digestive system. In the intestines, a thick mucosal layer traps harmful substances, while brush-border enzymes, bile salts, and changes in pH destroy harmful antigens (Sampson, 2004). Our innate (natural killer cells, macrophages, epithelial cells) and adaptive (lymphocytes, sIgA, cytokines) immune system provides another layer of attack against harmful invaders. When there are deficiencies of the gut barrier and immune system, food allergies can result (Sampson, 2004).

According to the National Institute of Allergy and Infectious Disease Expert Panel, there are four categories of immune-mediated food reactions: IgE-mediated, non-IgE-mediated, mixed IgE/non-IgE, and cell-mediated reactions (Sicherer & Sampson, 2014). IgE-mediated food allergies are those where a sensitization to certain food items develops, meaning the body develops a heightened response to intake. When food-specific IgE antibodies bind with circulating food allergens, mast cells and basophils are activated to release cytokines and other immunologic mediators (Sampson, 2004). This usually occurs in the first three years of life when serum-specific IgE antibodies are likely to develop for a given food allergen (i.e. peanuts, milk) (Björkstén, 2004; Turnbull, 2014). Technically, this response can happen with any protein, but the most common IgE reactions in adults are to peanuts, tree nuts, and seafood. Reproducible adverse signs and symptoms will develop immediately upon ingestion, inhalation, or skin penetration in the span of 1-2 minutes up to two hours (Turnbull, 2014). These symptoms include burning, tingling, itching of the mouth, throat constriction, shortness of breath, rhinitis, atopic eczema, urticarial and gastrointestinal reactions (nausea, vomiting, abdominal pain, diarrhea) (Kelsey, 2003; Bischoff & Crowe, 2005; Lied, 2011). The most severe reactions affect the whole body and can lead to fatal anaphylaxis. An example of an IgE mediated allergic response that effects the GI tract is oral allergy syndrome and gastrointestinal anaphylaxis.

Non-IgE reactions are predominated by a T-cell-mediated process (Sicherer & Sampson, 2014; Turnbull, 2014). In non-IgE reactions, there may be histological evidence of underlying immune response, such as eosinophilic inflammation (white blood cell) of the GI tract or intestinal cell destruction in CD (Turnbull, 2014). The immune mechanisms underlying these conditions are not well understood, though TNFα, an inflammatory immune signal, is involved (Turnbull, 2014).

Examples of conditions resulting from mixed IgE/non-IgE mediated FA are cow's milk protein allergy, eosinophilic esophagitis (EO), and eosinophilic gastroenteritis (EG). These conditions can present like other IgE reactions, with immediate symptom onset and potential anaphylaxis (Turnbull, 2014). In these conditions, the non-IgE mechanisms are harder to recognize due to a poor temporal distinction between exposure and symptoms. The ingestion of culprit foods may not be closely associated with the onset of symptoms (Turnbull, 2014). Clinically, this can look like treatment resistant GERD, eczema, diarrhea, and constipation (Turnbull, 2014). In fact, cow's milk allergy may account for nearly 40% of childhood GERD, since the milk protein, casein, can induce an immune response that disturbs peristalsis (Turnbull, 2104). EO and EG involve white blood cell infiltration of the respective GI organ, and there is increasing evidence that FA is involved in its pathogenesis (Turnbull, 2104).

Food allergies can be mistaken for food intolerances because they can both cause similar respiratory symptoms, rhinitis, and hives upon ingestion of culprit foods (Turnbull, 2014). Unlike food allergies, food intolerance reactions do

not directly involve the immune system and are harder to explain since symptoms are often delayed until several hours after an ingested meal (DiGiorgio, 2015; Turnbull, 2014). These symptoms, which often are attributed to irritable bowel syndrome, may be a result of altered small intestinal motility (due to stress or malnutrition), malabsorption of carbohydrates (i.e. lactose intolerance), enzyme deficiency, altered bacterial flora of the colon, or psychosomatic mechanisms (Berstad, 2005, Lied, 2011, Turnbull, 2014).

GASTROINTESTINAL SYMPTOMS OF FOOD ALLERGIES AND EATING DISORDERS

A large proportion (50%) of food reactions manifest in the gastrointestinal tract, yet they can be difficult to recognize, diagnose, and treat, and even more difficult to distinguish from EDs (Sampson, 2004). It is important to understand how food allergies manifest in the gastrointestinal tract, since many ED patients report impaired digestion related to suspected food allergies. I will discuss how EDs can affect the gastrointestinal tract and compare these physiological changes to what happens in the GI tract during a food reaction.

All EDs greatly affect normal gastrointestinal function. AN, BN, BED, ARFID, and OSFED have the capability of affecting all parts of the GI tract. Non-specific gastrointestinal complaints such as bloating, heartburn, nausea, early satiety, and constipation are common in ED patients; however, improved nutrition commonly reverses underlying physiologic issues (Bern, 2013; Salvioli, 2013; Zipfel, 2006). Furthermore, some argue that ED patents have a distorted perception of digestive functions, and may be experiencing a psychosomatic reaction that are not founded in physiologic dysfunction or FA (Salvioli, 2013; Robinson, 2000). This overview will focus on specific impairments in the esophagus, stomach, small intestine and large intestine leading to adverse gastrointestinal symptoms.

Esophagus

Functional esophageal complaints such as dysphagia and heartburn are common complaints in patients with AN and BN (Bern, 2013). Wang et al (2014) investigated the prevalence of adverse GI symptoms in 100 women with AN or BN, and found that 22% of patients complained of heartburn, 8% of chest discomfort, and 6% of dysphagia. Esophageal abnormalities are commonly linked with BN, but evidence confirming that relationship is limited (Bern, 2013). In a 60-year retrospective literature review of BN and other EDs, only four studies described an association with GERD. One study reported that 51% of the 101-patient ED unit experienced functional heartburn in connection with binge eating, while another study showed that 23 out of 37 long-term BN patients had normal endoscopic evaluations.

Esophageal inflammation or gastroesophageal reflux disease secondary to FA can closely resemble purging disorders (Bern, 2016). Vomiting, pain, dysphagia, and food refusal are main signs and symptoms of eosinophilic esophagitis (EoE) (Bischoff & Crowe, 2005, Bern, 2013). EoE is a mixed IgE/non-IgE condition in which the esophageal mucosa is marked by a dense infiltration of eosinophils, also known as disease-fighting white blood cells (Bischoff & Crowe, 2005). About 70% of patients with EoE show elevated serum IgE levels and have a history of FA (Bern, 2013; Turnbull, 2014). The most common allergens associated with EoE are cow's milk, soy, eggs, and wheat. EoE differs from GERD based on treatment strategy. EoE will not respond to proton-pump inhibitor (PPI) therapy, but will respond to food elimination and corticosteroids (Bischoff & Crowe, 2005). Individuals with EoE may employ compensatory behaviors that look like disordered eating behaviors. These include drinking excessive amounts of fluid to wash food down, chewing for prolonged periods of time before swallowing, or avoiding foods like bread, rice, and meat for choking risk (Bern, 2016)

Stomach

Gastric symptoms commonly described by ED patients include bloating, nausea, epigastric discomfort, and fullness (Bern, 2016). In the previously mentioned Wang et al (2014) study, specific gastric complaints of ED patients included gastric distention (45%), cyclic vomiting syndrome (17%), excessive belching (14%), nausea (10%), and rumination syndrome (7%). Evidence points toward impaired gastric motility as an explanation for these symptoms. The development of gastroparesis in malnourished adults with EDs has been cited a number of times, with the degree in delay of gastric emptying correlating with the degree of malnutrition (Bern, 2016; Georg, 2003; Robinson, 2000). Severe protein energy malnutrition from AN and BN results in the breakdown of muscle tissue in order to generate substrates for gluconeogenesis to keep blood glucose stable and provide energy for cells (Bern, 2013; Georg, 2003). This muscle breakdown weakens all bodily muscles, including those involved in the GI tract. Studies have found a higher rate of abnormal myoelectrical activity in the gastric muscles in patients with EDs, and with greater abnormalities in the delay of emptying in BN than AN (Bern, 2016). Reasons for this delayed emptying in BN patients could be impaired postprandial cholecystokinin release (altering satiety), increased gastric capacity, and diminished gastric relaxation from impaired autonomic response—not allowing "rest & digest" of the parasympathetic nervous system to take place. ED patients describe sensations of gastric distention, gas, and nausea as more numerous and intense than in healthy subjects, which can interfere with refeeding (Georg, 2003). Robinson (2000) describes the concept of distorted body image extending to the interior body, influencing an exaggerated response to normal postprandial physiology. However, these GI manifestations of malnutrition are not permanent—studies show that gastric emptying accelerates with the normalization of BMI (Georg, 2003).

Symptoms related to impaired gastric motility in ED patients mirror the symptoms of allergen exposure. The immediate IgE response is marked by nausea and vomiting as part of the anaphylactic response to the allergen (NIAID, 2010).

Small Intestine

Wang et al (2015) also described the intestinal complaints of ED patients. 53% of patients were identified with having irritable bowel syndrome (IBS), functional bloating, or constipation, while 16% had anorectal complaints. IBS-like symptoms and bloating arise in ED patients due to small bowel adaptations from malnutrition (Bern, 2016). The small intestine epithelial cells undergo apoptosis and reduce cell reproduction, but attempt to maintain a high ratio of villous cells in order to absorb as much as possible. Fewer cells overall, despite the adaptive changes, can lead to malabsorption and bacterial translocation from the large intestine-- a major contributor to bloating (Bern, 2016). Impaired motility extends beyond the stomach and into the small intestine-- another factor contributing to bloating in patients with EDs. A study found that duodenal transit time is significantly delayed in patients with AN compared to healthy controls (Bern, 2016).

IgE and non-IgE food allergies elicit intestinal symptoms that are similar to those experienced by ED patients. A specific IgE response can manifest in the intestines as abdominal pain and/or diarrhea, which is triggered by a rapid influx of fluid into the intestinal lumen (Berstad, 2005). A study utilizing endoscopy, transabdominal ultrasound technology, and MRI showed that direct duodenal administration of suspected allergens yields a rapid influx of fluid into intestinal lumen, intestinal wall thickening, and increased peristalsis (Lied, 2011). This suggests that local IgE reactions can generate adverse GI symptoms even in the absence of elevated total IgE serum concentrations (Lied, 2011). A specific example of a non-IgE FA that manifests in the small intestine is CD. Epidemiologic studies in Western nations suggest that CD occurs in 0.5-1% of the population (Bern, 2013; Bischoff & Crowe, 2005). Gastrointestinal symptoms of CD include diarrhea, steatorrhea, vomiting, abdominal pain, constipation, and bloating, which can lead to significant weight loss and failure to thrive (Bern, 2013; Berstad, 2005). Ingestion of the protein gliadin, found in wheat, rye, and barley induces intestinal inflammation via a T-cell response leading to the destruction of villi and impaired absorption of nutrients (Bern, 2013). Small intestine destruction is reversed with strict adherence to a gluten-free diet (Bern, 2013; Bischoff & Crowe, 2005).

Large Intestine

Constipation is a common problem in ED patients, and the uncomfortable sensations related to constipation can interfere with the refeeding process (Georg, 2003). Smooth muscle atrophy and electrolyte abnormalities, namely hypokalemia, are largely to blame (Georg, 2003). Potassium is the primary electrolyte in the stool (70-90 mmol/L), and hypokalemia, or low serum potassium, can be induced by frequent vomiting or laxative abuse. Laxatives are used to relieve constipation while also trying to promote weight loss. However, the loss of weight from laxative use is mostly due to water loss, not the expulsion of nutrients (Georg, 2003). A study of 24 patients with BN observed a significant inverse relationship between the rate of bowel emptying and serum potassium concentration, which was low in four patients and low-normal in eight patients (Georg, 2003). The delay in intestinal transit in patients with BN who abuse laxatives may also be due to "cathartic colon". In this case, the colon loses its ability to effectively expel intestinal contents due to loss of colonic neurons, loss of haustrations, decreased volume of interstitial cells, and loss of enteric neurons (Georg, 2003). The mechanism behind this loss of function is likely due to the release of neurotoxins and unidentified toxic compounds present in laxatives. This can be complicated by the abuse of magnesium-containing laxatives can lead to hypermagnesemia-induced neuromuscular injury, another factor delaying intestinal transit. For ED patients without laxative abuse, pelvic floor dysfunction may explain for the inability to expel bowel contents. Refeeding normalized intestinal transit time, but anorectal muscle contraction remained abnormal, perhaps because of malnutrition on muscle atrophy (Georg, 2003).

Allergic reactions that extend to the large intestine do not generally present with constipation, except sometimes in the case of the mixed IgE/non-IgE cow's milk allergy, which can delay colonic motility (Turnbull, 2014). Rather, IgE allergic reactions will manifest in the gut as abdominal pain and diarrhea. These are also symptoms of eosinophilic gastroenteritis (EG), which has been strongly associated with food allergies (Bischoff & Crowe, 2005). Much like EO, this uncommon disorder is characterized by eosinophilic inflammation. Non-immune food intolerances may also explain uncomfortable lower-bowel symptoms. Many patients with GI complaints think their symptoms are a result from adverse reactions to food, since symptoms are typically worse after meals (Turnbull, 2014). However, the caloric content of meals, the ratio of carbohydrate to fat, and the fiber content may influence postprandial colonic contractions more than the specific food item itself. Nevertheless, foods high in carbohydrates and fat, coffee, alcohol, and spices are cited as contributors of negative symptoms (Turnbull, 2014). Specific food intolerances that manifest in the intestines include lactose intolerance, fructose malabsorption, and gluten sensitivity. Symptoms of lactose intolerance result when unabsorbed lactose, due to a lactase enzyme deficiency, reaches the large intestine. There, it causes osmotic diarrhea and gut bacteria ferments the lactose, which releases gas. However, chronic ingestion of lactose can allow for colonic bacteria adaptation over time (Turnbull, 2014). Fructose is a

monosaccharide found in fruits, and does not require enzymatic breakdown in order to be absorbed. Instead, it gets absorbed through facilitated diffusion, and glucose aids in its absorption (Turnbull, 2014). However, some individuals are unable to completely absorb fructose, allowing it to ferment in the large intestine. Lastly, gluten sensitivity can occur even without confirmed CD or wheat allergy. These individuals do not have allergenic immune markers, but will still report a variety of symptoms in response to ingesting gluten. At this time, the mechanism behind non-celiac gluten sensitivity remains unknown, but it has been hypothesized that gluten-containing foods act as high FOD-MAP food items, resulting in IBS-like symptoms (Biesiekierski et al, 2013). FODMAPS include fructans (vegetables, wheat, barley, rye), galactans (beans), polyols (fruits), lactose, fructose, and artificial sweeteners. These food groups contain poorly absorbed short chain carbohydrates that induce luminal distention, which can induce osmotic diarrhea and colonic gas production (Turnbull).

Eating disorders and food allergies result in similar symptoms across the major organs of the gastrointestinal tract. Based on these similarities, it is understandable a) why an eating disorder patient with impaired digestion would be apt to self-diagnose a FA or b) why someone with a FA might develop a disordered relationship to food in effort to avoid such adverse reactions.

2. CURRENT LITERATURE

Several procedures were used to retrieve published and unpublished articles. A computer search was performed on PubMED, ResearchGate, Google Scholar, Web of Science, and UNC Library using the following key words: *eating disorders, eating pathology, anorexia nervosa, bulimia nervosa, binge eating, ARFID, food allergy, food intolerance, food hypersensitivity, somatization, adverse food reactions.* 27 of articles regarding food allergies (including gluten allergy of CD) were initially retrieved. Upon review, articles were eliminated if they were outside the scope of this review (e.g., discussed other psychiatric comorbidities at length but made no mention of EDs) and articles not in the English language due to inability to translate.

Chronological progression of FA and ED research show a number of themes. Early case studies established a possible relationship between the two pathologies. The next wave of studies looked at the prevalence of clinical and subclinical EDs in the adolescent and adult population with food allergies. From there, authors looked at screening for food allergies in ED patients, and debated if that was a useful practice. Meanwhile, other studies investigated predetermining characteristics for ED development in those with food allergies.

Examining the co-existence of food allergies and eating disorder

Case Studies

Terr (1986) was the first to establish the relationship between food allergies and EDs by looking at the predisposing factors leading to these comorbities. At the time, suspected reactions to allergenic foods that were not reproducible or failed the gold standard double-blind placebo controlled food challenge (DBPCFC) were assumed to be psychological in nature (Terr, 1986). Terr analyzed several case studies and found two major themes: 1) concern about body weight may be an initiating increased preoccupation with food consumption; and 2) the power of suggestion can create an irrational fear over certain food items. In all cases, food diaries and DBPCFC did not confirm suspected allergy. Although the cases included in Terr's study did not meet the diagnostic criteria for AN or BN, they did exhibit symptoms of frequent weight fluctuations, body image concerns, preoccupation with food and eating, and depression, all of which are classical manifestations of disordered eating.

Other early case studies reveal similar themes surrounding preoccupation with food, body image, and/or weight disturbances, while also bringing to question the timeline of the onset of FA and ED. Does the FA influence the development of an ED or vice versa? The literature shows evidence for both interactions. For example, Kosky et al (1993) found a case where the diagnosis of FA preceded the development of bulimia nervosa. From an early age, this individual was shamed for her allergy, and thus developed strong emotions around food that lead to disordered eating. Ricca et al (2000) also unveiled a case where diagnosis of CD preceded the development of AN. Perhaps the strict and necessary restriction of gluten in order to avoid gastrointestinal disturbances and villous atrophy triggered a pathological restraint in eating. Additionally the burden of chronic illness on normal growth and weight gain can focus attention unduly on body size, which may be a contributing factor to the pursuit of thinness in vulnerable individuals (Ricca, 2000). While some individuals with food allergies may go on to develop an ED, others may go on to develop subclinical disordered eating patterns that nonetheless have the ability to interfere with daily life. For example, a case study followed five children (9-18 years old) after a confirmed diagnosis of peanut allergy (King et al, 2009). While no case developed a full-blown ED, they all developed abnormal eating behaviors that included a strict refusal to eat away from their mother or family home. However, obsessions about contaminated food items can develop into a full ED in vulnerable individuals. In one case, an individual with multiple psychiatric comorbidities developed OSFED after a CD diagnosis (Sharma et al, 2011)

While some initial case studies suggest that diagnosis of a FA can lead to the development of an ED, just as many unveil the opposite relationship. In fact, the same Ricca et al (2000) study featured another case where the diagnosis of CD came several years after the onset of AN, binge/purge type. This suggests that the new CD diagnoses could represent a complication from years of disordered food behaviors affecting the gastrointestinal tract. However, another possibility is that this case presented with subclinical CD for years, and went undiagnosed. Yucel et al (2006) brought to question the possibility that ED behavior may be masking underlying food allergies and CD. In a case study by Yucel el al, a patient with an atypical ED presented with a low BMI and ritualistic food behaviors, yet did not complain of poor body image, fear of gaining weight, or of amenorrhea. This patient was later diagnosed with CD, which would explain the weight loss and avoidance of foods if they were causing gastrointestinal distress. This parallels an OSFED case with a BMI of 15.8 that saw the resolution of all disordered eating behaviors and 10% weight restoration two months after removing allergenic food items (in this case, gluten) from her diet (Pynnonen, 2002). Prior to CD diagnosis, this patient was preoccupied with thinness and perfectionism, yet weight restoration did not elicit anxiety. This suggests that the allergic reaction to gluten was a contributor to disordered thoughts and behaviors, and that the disordered thoughts and behaviors masked the root of the problem. This idea is further supported by a case where a less common eating disorder, pica, developed due to iron deficiency secondary to malabsorption related to CD (Korman, 1990).

A case report by Leffler et al (2007) provides a more nuanced investigation of the interaction between food allergies and EDs, beyond exploring which disease elicits the other. An interdisciplinary research team at Brigham and Women's hospital in Boston, MA explored 10 cases of individuals diagnosed with both an ED and CD. Themes from previous case studies persisted, such as a diagnosis of CD/FA leading to development of an ED (Kosky, 1993; Ricca, 2000), and undiagnosed CD/FA mimicking an ED (Pynnonen, 2003; Yucel, 2006). Leffler et al (2007) observed how a CD diagnosis could improve ED outcomes, but also how an ED diagnosis can impair CD outcomes due to intentional gluten exposure in effort of promoting diarrhea and weight loss.

Prevalence Studies

While these case studies offer insight into the complexities between disordered eating and FA or intolerance, the data is lacking objective analysis. Recent research has moved to more sophisticated study designs in order to elucidate more information on the relationship between EDs and food allergies, particularly gluten allergy. A large systematic study by Karwautz et al (2008) looked at a large sample (n=283) of adolescents aged 10-20 years diagnosed with CD. CD patients were assessed for ED by BMI, the Eating Disorder Inventory (EDI-2), Eating Disorder Examination

Questionnaire (EDE-Q), and an Eating Disorders Examination Diagnostic Interview (Karwautz, 2008). In the analysis, Karwautz et al. looked at lifetime history and current presence of disordered eating, considering both clinical EDs and sub-clinical disordered eating (SED). They found that compared to the general population, there was a higher rate of disordered eating among adolescent celiac patients and that all those affected by disordered eating were female. 4.8% of the cohort was affected by a lifetime history of an ED (one AN, four BN, and six OSFED), while 3.9% was affected by a current ED (all OSFED). 10.2% reported a lifetime history of sub-clinical disordered eating, while 10.7% reported current subclinical disordered eating. While a celiac diagnosis preceded the onset of an ED or SED in most cases (86%), this study does provide evidence that ED or SED can also precede a celiac diagnosis. Compared to those adolescents with CD who did not develop an ED, those that did had a higher rate non-compliance with the gluten-free diet (32% vs 16%) (Karwautz, 2008). The lack of a control group in this study makes it difficult to determine if ED is related to FA or non-specific burden of chronic disease (Leffler, 2007; Ricca, 2000; Yucel, 2006l)

Adolescents with not just CD, but other food allergies as well, are affected by higher rates of EDs. Cross-sectional analysis of the longitudinal Great Smokey Mountain Study (GSMS) recruited a representative sample of children (ages 9, 11, and 13) from 11 counties in western North Carolina for detailed interviews and were assessed annually until age 16 (Shanahan et al, 2014). 5,231 interviews took place, and 5,165 had complete FA data. The incidence of food allergies was assessed by self-report from the parent, while the participants were interviewed separately. The presence of EDs was assessed through DSM-IV criteria for underweight status, fear of gaining weight, body image disturbances, amenorrhea, binges, and other compensatory behaviors used for weight loss. Results showed that, even when controlling for sex and age and adjusting for psychiatric comorbidities, food allergies were associated with symptoms of anorexia nervosa ($p<0.01$). Authors concluded that the changes in food intake due to FA likely alter feelings about food and body image (Shanahan et al, 2014).

Karwautz et al (2008) and Shanahan et al (2014) showed that adolescents with CD are at increased risk for developing an ED, and Passananti et al (2013) looked to see if this association persisted in the adult population. Passananti et al (2013) developed a case-control study where celiac patients and controls completed a dietary interview, the Binge Eating Staircase, Eating Disorder Inventory (EDI-2), and Eating Attitudes Test (EAT-26) to determine degree of eating pathology. They found that celiac patients had a higher amount of pathological EAT-26 scores compared to controls (16% vs. 4%; $p=0.01$). Surprisingly, the level of pathological eating did not correlate with the presence of adverse gastrointestinal symptoms. This is a surprising finding as some studies cite digestive complaints as a reason for screening those with EDs for CD (Basso, 2012; Kaltsa, 2015; Saldhana, 2016).

Food Allergy Screening in Eating Disorders

A group of recent studies has looked at the benefits of screening individuals with AN for CD. A prospective study that took place in the Neuropsychiatry Unit at Children's Hospital in Rome collected data from screening procedures used with 177 patients with AN from January 2005-December 2010 (Basso et al, 2012). Patients were screened for CD via anti-transglutaminase antibodies of IgA class (tTGA) and by anti-transglutaminase antibodies of IgG class (tTGG) in case of IgA deficiency (IgA < 10 mg%). 32% of AN patients reported abdominal complaints, however only 1 case had a positive tTGA and small intestinal biopsy results. The prevalence of CD in this cohort was 0.6%, which is similar to what is observed in the general population (~1%). Basso et al (2012) also noted that the digestive complaints of AN patients subsided during the follow-up period, however the degree of regression and period of follow-up was not specified. This suggests that GI symptoms are part of the ED, as GI complaints improved over a period of psychiatric intervention.

Similar results were found through a retrospective chart review evaluating all patients (7-22 years old) of an inpatient adolescent medicine department (2011-2014) for an ED (Saldanha et al 2016). Of the 1,160 evaluated for an ED, 494 (42.6%) were also screened for CD by tissue transglutaminase IgA antibody (tTGA) and anti-endomysial IgA antibody (aEAb). Total IgA was also measured to rule out IgA deficiency. Of those screened for CD, 2% (10 patients) had positive serum tTGA results. Of the 10 with positive serum values, only four patients were diagnosed with CD as confirmed by small intestine biopsy. This yields an overall 0.8% prevalence of CD in the ED cohort—quite similar to what was observed by Basso et al (2012).

The large sample sizes and objective screening methods are strengths for these two studies. However, one weakness is that the overall gluten intake of AN patients was not specified and may not have been enough to yield a positive serum tTGA or tTGG, given the overall reduced intake in AN. This potential confounder was accounted for in the study design of another study that screening for CD in patients with EDs. A prospective study of 154 patients with a mean age of 16.7 were assessed by a medical history intake that focused on GI symptoms and daily gluten intake (Kaltsa et al, 2015). Gluten intake was self-reported, and averaged 10-15 g/day. 50 patients dropped out of the study due to needing a higher level of care or death, so 104 patients were reassessed after weight gain, which took on average 18-19 months. Constant psychiatric supervision on the unit and a dietary regimen of at least 15 g of gluten per day not only assisted in weight gain, but also resolved GI complaints of post-

prandial distention in 32% of the patients (Kaltsa et al, 2015). No reports of CD were cited in this cohort (Katlsa et al, 2015). For this patient population, it is likely that GI complaints were manifestations of the ED, and not due to FA.

Disordered Eating Predisposition in Food Allergy

Two articles in the past year sought to define which personality characteristics that predispose those with FA to EDs in both adolescents and adults. A cross-sectional study by Wagner et al (2015) used the same cohort of adolescent females with CD as described previously (Karwautz et al 2008). A total 259 adolescent females with CD were used in this analysis aimed at determining levels of depression, personality characteristics, and coping strategies present in cases with comorbid EDs. Analysis found that 15.5% of adolescents had comorbid CD and ED. Overall, patients in this group were older, more non-compliant with gluten-free diet, and had a higher BMI (Wagner, 2015). Within this group, 31.25% had elevated depression scores, compared to 6.9% of elevated depression scores in those with CD without comorbid ED. Another cross-sectional study looked at the predictors of disordered eating in adult women with CD (Satherley et al, 2016). Online versions of EAT-26 and the Binge Eating Scale (BES) were distributed via online forums to patients with CD (n=157), IBD (n=116), type-2 diabetes (n=88), and healthy controls (n=142). The celiac group was comprised of patients self-reporting a positive small intestine biopsy result. Results showed that the celiac group had higher EAT-26 scores compared to the type 2 diabetes and healthy control group, but not the IBD group (Satherley et al, 2016). EAT-26 identified that dietary management was the most important factor for those with CD, and cluster analysis identified binge eating and AN-restrictive type as the main EDs evident in the study's celiac patients. Moreover, cluster analysis identified features of "low risk" individuals compared to "critical" or "high distress" individuals (Satherley et al, 2016). Of the celiac cohort, 60 were categorized into the low risk group that reported low psychological distress, few GI symptoms, good dietary management, and low EAT-26 and BES scores. Another group of 25 participants were identified as being "critical", with moderate stress, poor dietary management, many GI symptoms, and high EAT-26 scores. The final group of 11 participants was labeled "high distress", who scored the highest on psychological distress, high BES scores, but have good dietary management (Satherley et al, 2016). The online recruitment of this study may have over-sampled from the younger population more apt to be online, creating a bias that may distort the magnitude of eating concerns in the celiac population.

While both of these studies provided useful analysis on the characteristics leading to disordered eating in CD, the cross-sectional study design does not allow for a causal relationship between FA and EDs to be established.

Furthermore, self-report measures used for CD diagnosis in the Satherley et al. (2016) is not a reliable measure for recruiting CD patients, especially when individuals are apt to self-diagnose food allergies or intolerances. A promising abstract from the 2009 British Society for Allergy and Clinical Immunology Conference (Knibb & Smith, 2009) showed that self-diagnosis of FA or intolerance was reported by 35.5% of 102 adult participants who completed a study-specific eating attitudes questionnaire, Stirling Eating Disorder Scale (SEDS), and a Body Shape Questionnaire (BSQ). Those who self-diagnosed had significantly higher scores on all sub-scales of the SEDS ($p<0.05$), had greater concerns about body shape ($p<0.05$), and admitted to have dieted in the past or currently ($p<0.05$) (Knibb & Smith, 2009). While this study did not identify EDs by DSM-V criteria, the data does suggest that disordered eating is a factor in self-diagnosis of FA as evidenced by increased attention on body shape and food intake. Authors do think it is possible that FA or intolerance is used, either consciously or unconsciously, to mask eating disordered behavior.

3. Discussion and Recommended Practice Guidelines for Interdisciplinary ED Treatment Teams

The current literature on food allergies and EDs establishes a connection between the two pathologies. Early case studies observed the ways in which the two pathologies intersected. A main theme discussed was which comes first, the FA or the ED? There was evidence for both progressions, with much of the literature focusing on CD.

Food allergy → Eating Disorder

Kosky (1993) and Ricca (2000) discussed cases in which FA preceded development of the ED, and a few cross-sectional and case-control studies provided prevalence data showing that disordered eating may occur at a higher rate in individuals with food allergies (Karwautz, 2009; Passananti, 2013; Shanahan 2014). Moreover, a couple of studies sought to determine predictive factors towards the development of an ED in the face of a FA (Satherley et al, 2016; Wagner, 2015). Overall, much of the current literature is supportive of the notion that having a FA can lead to the development of an ED, as opposed to the opposite association. There are varied hypothesized reasons as to why an ED would develop in the context of FA. Whether an individual is dealing with an immediate IgE peanut allergy or CD, the approach to treatment is the same-- complete avoidance of the offending food item. The necessary vigilance it takes to make safe food choices at every meal and snack may elicit a permanent preoccupation with food, leading to an overall increase in attention on eating behavior. An increased focus on eating can lead to pathological restraint in eating or an increase in binge eating and compensatory behaviors (Kaltsa et al, 2015; Ricca, 2000). Additionally, the burden of a chronic condition on normal growth and weight gain can lead to an unduly focus on body size, which may be a contributing factor to the pursuit of thinness in vulnerable individuals (Ricca, 2000).

Not all CD patients experience abdominal discomfort, but for those that do, painful symptoms could lead to a severe decrease in calories, potentially triggering disordered eating patterns by associating certain foods with pain. CD is a unique manifestation of FA because the effects of small intestinal villous atrophy are not immediate, but malnutrition can be seen over time in the form of growth interference, pubertal delay, and weight loss. Removal of gluten from the diet tends to reverse the consequences of malnutrition, leading to necessary weight gain. However, this often-sudden change in body size can trigger disordered thoughts and behaviors around food and body image. Patients may compensate for this change by fixating on food intake. This is especially of concern among the adolescent celiac population who are already in a vulnerable position based on pubertal changes taking place (Karwautz, 2009; Wagner, 2015).

Eating Disorder → Food Allergy

Case studies by Ricca (2000), Yucel (2006), Pynnonen (2003), and Korman (1990) suggest that EDs can precede food allergies. The reasons why EDs may lead to the development of FA are numerous. However, one has to first determine if an individual with an ED is dealing with situation #1, an *actual* FA, or situation #2, a "food allergy". The measures needed to diagnose and treat actual food allergies in the context of EDs is discussed in the next section. There is some speculation that actual food allergies, situation #1, can result as a complication of years of disordered eating behaviors negatively affecting the GI tract. As discussed previously, it is true that EDs compromise gastrointestinal function, which can feel similar to allergic responses. The lack of inflammatory response in most ED patients, however, hints that the impaired gastrointestinal abilities frequently cited in this population are functional losses due to malnutrition and not the consequence of an immune-mediated process. Research shows that true food allergies are no more frequent in the ED population (Basso, 2012; Kaltsa et al, 2015; Saldanha et al, 2106), so the likelihood of an ED patient developing a FA is very unlikely. Nevertheless, it is possible that long-term EDs mask underlying CD since a restrictive food intake prevents an accurate diagnosis Treatment providers should keep this in mind for patients with a variety of unexplained symptoms (Kaltsa et al, 2105).

It is far more likely that the reported food-related gastrointestinal complaints of ED patients fall under situation #2: "food allergy". From the literature, it appears as though "food allergy" is either a) a misnomer for *food intolerance*, a non-immune response to food, b) impaired gastrointestinal function, or c) psychosomatic symptoms. Food intolerances, such as lactose intolerance, may develop in ED patients from a lactase enzyme deficiency secondary to the effects of malnutrition. In practice, dairy is often cited as a difficult food to digest in the ED population. Another reason for this difficulty, beyond lactase deficiency, is a deficient population of gut flora due to starvation. A healthy gut flora can combat against the ill effects of dairy consumption, such as gas, bloating, and altered bowel

movements. An enzyme deficiency and altered gut flora can also be accompanied by reduced stomach, small intestine, and large intestine motility. This causes food to “sit” in the GI tract, leading to early satiety, abdominal pain, gas, bloating, and constipation. Luckily, improved nutrition provides the substrates for enzymatic production, feeds healthy gut bacteria, and “wakes up” the GI tract, allowing for smooth digestion. In this case, food is not the enemy, but rather the solution to gastrointestinal complaints amongst the ED population.

Even so, American media loves to make an enemy of food. The 1980’s and 90’s created a fear around fat, spreading the erroneous idea that eating fat leads to excess fat. Then the low-carb craze hit in the early 2000s as a form of extreme weight loss, and now gluten is being cited by journalists and celebrities as the cause for a wide variety of ailments—including excess weight. The power of suggestion can spread irrational fear around certain foods, only propelling our fat-phobic culture and amplifying disordered thoughts among the ED population. Popular media speculates that food allergies are “the new eating disorder,” and this may be possible. Confirmed FA diagnosis remains stable in recent years, yet self-reported “food allergies” are on the rise. The literature is not currently able to confirm if this rise in self-reported food allergies results from a disordered mentality, but it questions the role of psychosomatization. Adverse food-related gastrointestinal symptoms that go unconfirmed by FA testing may occur more in individuals with psychiatric features such as hypochondria, somatization, and anxiety (Bischoff & Crowe, 2005). In a qualitative study of patients with food hypersensitivity symptoms, 57% of patients fulfilled DSM-V criteria for a psychiatric disorder, including depression, generalized anxiety disorder, panic disorder, and somatoform disorders (Parker, 1991). Moreover, a review article by Teufel et al (2007) looked at seven studies investigating the psychiatric profile of patients with self-reported food hypersensitivity. Food sensitivity correlated with mood disorders (i.e. anxiety, depression), and was reported more frequently in women with neurotic symptoms. Patients with anxiety or excessive worry tend to have a heightened vigilance of their internal and external environments. This increased attention to physical symptoms may affect central nervous system mechanisms, and just the thought of ingesting a food can trigger allergic symptoms in the absence of antigens (Bischoff & Crowe, 2005; Lied, 2011; Kelsay, 2003; Teufel, 2007). Specifically, psychological stress (along with any other type of stress) leads to the release of corticotropin releasing hormone (CRH) from the anterior pituitary gland. CRH acts on the vagus nerve and suppresses its activity, which results in gastroparesis and delayed gastric emptying, leading to early fullness and abdominal discomfort after eating. A vicious cycle can develop when an anxious mind misinterprets abdominal sensations as harmful, causing more stress and more GI symptoms (Berstad, 2005; Teufel, 2007). This is not necessarily surprising, given knowledge of the extensive neural networks connecting the central nervous system, the enteric nervous system, and the immune system (Bischoff & Crowe, 2005). In fact, electron microscopic examinations show that the enteric nervous system and intestinal mucosal mast cells are in direct contact (Berstad, 2005).

Intestinal perfusion studies show a release of histamine and tryptase from mucosal mast cells in response to stress. This response resulted in an increase of fluid into the intestinal lumen, similar to the mechanism of a FA (Berstad, 2005). Thus, it may be very difficult to differentiate the actions of the mind versus the body in ED patients.

Literature Gaps & Future Directions

The current literature provides us with essential information on the prevalence, screening, and predictive factors of comorbid food allergies and EDs. However, the current literature only begins to make connections between the seemingly complicated relationship between disordered eating and food allergies—whether the allergies themselves are validated or self-reported. Since the majority of the literature is comprised of stand-alone cases and cross-sectional studies that are unable to determine causality, future investigation requires a more objective analysis of the relationship between food allergies and EDs. This can be accomplished through the creation of larger systematic prospective trials. Larger studies with sophisticated sampling techniques, as opposed to convenience sampling at health clinics (i.e. Karwautz, 2008), can help establish study cohorts that are representative of the entire US. Future studies should aim to accomplish this, since much of the current literature was conducted in either Europe (Karwautz, 2008; Wagner, 2015) or specific American communities (Shanahan et al, 2016). Moreover, larger studies may provide for better figures, considering how the overall prevalence of both food allergies and EDs is very low. Although self-report measures are conveniently implemented and frequently used throughout the literature (Passananti, 2013; Satherley, 2016; Shanahan et al, 2016; Wagner, 2015), future studies should consider more valid diagnostic methods for food allergies, such as the DBPCFC, considering the notoriously inaccurate nature of self-report. Currently there are no long-term studies investigating the development of EDs in individuals with food allergies and vice versa. Such longitudinal studies are needed in order to determine the timeframe between FA onset and disordered eating (or vice versa).

There are several themes to incorporate into future research. For instance, prospective studies investigating the role of screening for CD in ED patients found no association between the two pathologies, despite highly prevalent digestive complaints. Future prospective research studies are needed in order to understand the significance of digestive complaints in ED patients—are they psychological or functional in nature? What threshold of psychological or functional disturbance is enough to elicit a self-diagnosed “food allergy”? Although there is some evidence that digestive complaints may subside after nutritional rehabilitation, the current literature is lacking conclusive evidence of the efficacy of nutrition therapy in the normalization of digestive function in ED patients and how this may be confounded by the high relapse rate in this population. Longer-term prospective studies may be required to account for this factor.

A couple of recent cross-sectional studies (Wagner, 2015; Satherley, 2016) looked at the predictors of ED development in individuals with food allergies, and more research is required to develop the association between psychopathology, BMI, gastrointestinal symptoms, personality characteristic and the development of an ED. The current literature discussing food allergies and EDs failed to look at the influence of external environmental factors commonly observed in clinical ED practice such as familial and personal relationships, trauma history, identity crises, bullying, and societal repression. Future research should account for these influencing factors, and also consider the factors that may lead to the development of one type of ED over the other. Much of the literature focused on AN and BN, and a few hinted at the role of binge and OSFED (Kaltsa et al, 2015; Ricca, 2000; Satherley, 2016). However, no large systematic studies have exclusively looked at the relationship between BED or OSFED and FA. Future research should also consider the function of the new ARFID diagnosis in assessing self-reported food allergies amongst the ED population. AFRID may be the appropriate diagnosis if patients present with food avoidance in the absence of body image or weight disturbances. It would be interesting to determine if ARFID is more or less apt to develop in individuals with food allergies, as opposed to other EDs.

Overall, the literature has just started to untangle the relationship between food allergies and EDs, but much more information is needed in order to better understand how and why each pathology manifests, and to further guide treatment recommendations.

Steps for Eating Disorder Treatment Providers

While the literature has yet to establish a cause and effect between food allergies and EDs, it does provide a collection of helpful information to guide clinicians working with the ED population. If an individual seeks treatment for disordered eating and complains about gastrointestinal symptoms, then the first step is to rule out a true FA or intolerance (Sicherer, 2104). The 2010 Expert Panel recommends that all patient reports of FA must be confirmed due to the high likelihood (50-90%) that the reported complaints are not due to allergy (NIAID). Diagnosis of FA is a serious medical complication in the treatment of EDs. Having an accurate diagnosis at the start of treatment can prevent unnecessary dietary restriction, which would confound and likely prolong the recovery process (Teufel, 2007). Each member of the ED treatment team (physician, nurse practitioner, RD, psychiatrist, and social worker) plays a role in this diagnosis (Leffler, 2007).

Physical Exam: Diagnosis of a FA starts with a physical exam and medical history (NIAID). The goal of the physical exam and medical history is to determine the likelihood of an immunologic adverse reaction to food and to rule out differential diagnoses based on the symptoms of concern (Turnbull, 2014). Food allergies and/or EDs present similarly to other gastrointestinal diseases, deficiencies, toxicities, or infections (Sampson, 1999). The physician,

nurse, or RD can review the patient's weight and growth history to establish nutritional sufficiency. A patient's growth curve showing deceleration, delayed puberty, and weight loss is evidence for FA or ED (Bern, 2013). Physical signs indicative of either ED or malnutrition secondary to FA include hair loss, dry skin, lanugo, jaundice, pallor, and digital clubbing (Bern, 2013). Generally, the patient's history is notoriously inaccurate, and cannot be used in isolation to determine allergy risk. Certain characteristics have been associated with increased risk of FA and they include a family history of allergy, non-Hispanic black ethnicity, Asian ethnicity, comorbid allergenic conditions (i.e. severe atopic dermatitis, asthma), vitamin D insufficiency, male sex, and young age (Savage, 2015; Sampson, 2004; Turnbull, 2014). It is important to be aware that few foods are responsible for the vast majority of reactions, and it is very rare to be allergic to more than one food (Sampson, 1999). This provides an interesting contrast to ED demographics, mostly comprised of young women, who may report an intolerance to many food items.

RD Interview: The RD interview should be conducted at the time of the physical exam to obtain diet-specific history. Dietetic input in ED treatment was previously neglected, as the disordered eating behaviors were viewed as secondary to the psychopathology of the disease (Hart, 2011). However, this has shifted since learning about cognitive side effects of malnutrition (i.e. Minnesota Starvation Study) that prevent ED patients from engaging in mindful therapy (Hart, 2011). During the initial RD interview, several questions should be asked to get a better understanding of the relationship the patient has with their FA or intolerance. The RD will want to confirm how and when the FA was diagnosed, and by whom. How do the patients feel when they eat these foods, and has food caused these symptoms more than once? What quantity of this food elicits symptoms? What was the timeline of food exposure and symptoms, and how long did they last? Can food ever be consumed without causing symptoms (i.e. baked versus raw), and have symptoms occurred without consuming food? It is important to keep in mind other foods eaten at the same time, the potential of contaminated foods, and of "hidden ingredients" (Sampson, 1999). For instance, milk and soy protein are added to processed foods into increase protein content and enhance flavor, while nut products are used to flavor and thicken sauces (Sampson, 1999). To evaluate mind/body interaction, the RD can inquire about their emotional state surrounding ingestion of that food item, as well as the physical response that it elicits. It might be helpful in this case to start a diet diary with the patient in effort to establish a less biased recall of foods, the timing of symptoms, and the corresponding emotional state (Sampson, 1999; Bischoff & Crowe, 2005). This can lay the foundation for the emotional work that patients encounter with their therapists, as there is the possibility that the symptoms are psychological in nature (Teufel, 2007). In addition, assessment of other food restrictions for religious or ethical reasons (vegan, vegetarianism) can provide useful data on the patient's history of restriction. Other factors such as exercise, alcohol, and use of aspirin and NSAIDs can be assessed. If a FA is not yet

diagnosed and is suspected, the RD can work with the physician, NP, and nurse team members to acquire proper diagnosis and documentation for this patient.

Labs: Obtaining basic laboratory work at the time of physical exam can help determine likelihood of FA in the context of EDs. A complete blood count with differential can provide information about anemia, iron deficiency, inflammatory marker elevation (CRP, platelet count), and peripheral eosinophilia (WBC). Abnormalities in these values are usually seen in conditions resulting from food allergies (i.e. CD, EoE, EG), but not EDs. Surprisingly, many ED patients present with completely normal inflammatory markers, CBC, serum albumin and iron—even when malnourished (Bern, 2016). Food allergies may lead to low iron levels for a number of reasons—low iron intake, inadequate absorption, or chronic blood loss from chronic mucosal irritation. An abnormal electrolyte panel is telling in ED symptomology, rather than FA diagnosis. AN and BN can be associated with hypokalemia, hyponatremia, metabolic alkalosis, hypomagnesemia and hypocalcemia.

Food Allergy Screening: Beyond basic lab work, the treatment team can pursue a number of tests if FA is suspected. It may be helpful for the treatment team to categorize reactions based on allergic mechanism (i.e. IgE mediate, non-IgE mediated) because that drives the course of action of further investigations. The 2010 Expert Panel Guidelines recommends skin prick tests, allergen-specific blood tests, oral food challenges (OFC), and elimination diets for the treatment of IgE food allergies (NIAID). The double-blind placebo controlled food challenge remains the gold standard for objective diagnosis of FA, however it is not commonly implemented in the clinical setting due to high risk of allergic reaction, inconvenience, and cost (Berstad, 2005; Bjorksten, 2004; Turnbull, 2014; Sampson, 1999). Upcoming miniaturized technology involving protein and peptide microarrays may allow for future screening of IgE mediated food allergies to multiple foods at once with just a few drops of blood. This technology could also determine cross-reactivities, the severity of the reaction, and the likelihood of outgrowing the allergy (Sampson, 2004).

To date, no laboratory tests have been shown to identify foods in non-IgE mediated reactions (Sampson, 1999). For gastrointestinal allergies, histology of biopsy samples confirms the diagnosis, but cannot indicate specific foods responsible for the reaction. In non-IgE conditions such as EoE or EG, high white blood cell count may be evident in the blood and stool, but cannot be used to diagnose FA. Furthermore, food-specific IgG antibodies can be elevated in patients with non-IgE allergy affecting the GI tract, but IgG mostly reflects the types of food typically ingested and are not specific to “food related pathogenesis” (Sampson, 2004). Although not yet recommended by the Expert Panel, other potential measures for non-IgE mediated FA diagnosis includes measurements of fecal matter, mucosal biopsy, and provocation (Berstad, 2005). The fecal biomarkers calprotectin and lactoferrin have been used to identify patients with inflammatory conditions involving the gut. Both of these biomarkers are derived from neutrophils in the mucosa

of actively inflamed intestines (Bern, 2016). A promising new screening method, known as colonscopic allergen provocation (COLAP) test, involves a colonoscopy-guides submucosal injection of the offending allergen. Initial investigation shows that 77% of individuals with suspected FA or intolerance had a positive COLAP test, and the COLAP test was consistently negative in control subjects (DiGiorgio, 2015). More research is needed to determine if gut provocation is the diagnostic tool of the future.

Celiac Disease: The available literature on food allergies and EDs discusses CD at length. There are a specific set of measurements used for the screening of CD. According to the latest guidelines, serum anti-tissue transglutaminase immunoglobulin A (IgA tTG) and immunoglobulin A (IgA) are used for screening purposes (Kaltsa, 2015; Bern, 2013). For individuals who are IgA deficient, immunoglobulin G deaminated gliadin peptide antibody (IgG DGP) are used instead (Bern, 2013). A gastroenterologist can also test for endomysial IgA antibody levels, which can be a more sensitive and specific screening measure, albeit expensive (Bern, 2013). Official diagnosis of CD is made through small bowel biopsy via endoscopy showing villous atrophy. The ED treatment team can screen for CD in patients exhibiting gastrointestinal symptoms, weight loss, and growth failure—although statistically, it is likely that these symptoms are a consequence of the ED and not presence of CD. If patients express interest, they can be screened for genetic markers of CD—HLA-DG2 or DQ8. Because the literature showed the pathological presentation of ED and CD going either way, it is important for professionals caring for patients with CD to routinely discuss body image and weight concerns (Bern, 2013).

Not Recommended: For IgE or non-IgE mediated food reactions, the 2010 NIAID Expert Panel does not recommend intradermal tests, total serum IgE blood tests, atopy patch testing, or tests that have not been standardized, including: measurement of basophil histamine release, lymphocyte stimulation, facial thermography, gastric juice analysis, hair analysis, applied kinesiology, allergen-specific IgG4 measurement, electrodermal testing, cytotoxicity assays, and mediator release assay (LEAP diet) (NIAID; Sicherer, 2014).

Elimination diets are usually implemented in order to support positive screening measures and establish a FA diagnosis. However, elimination diets are not recommended in the context of ED treatment. A successful elimination diet requires the complete exclusion of the suspected allergen(s) in all forms from the diet, so that a lack of response to the suspected allergen upon reintroduction rules out a FA (Sampson, 1999). Following the strict protocols required of an elimination diet goes directly against dietary liberalization efforts in ED treatment. Turnbull et al (2014) noted how strict adherence to elimination diets places significant stress on patients, and how vigilance about accidental exposure can lead to a reduced quality of life. The ED population is particularly vulnerable to this diet-related stress, and following an elimination diet at any point in ED treatment poses the risk of triggering ED symptoms.

Clinical Management of Eating Disorders with Comorbid Food Allergy

If an ED patient has a confirmed FA diagnosis, then the only proven form of treatment for both IgE and non-IgE mediated food allergies is strict avoidance of the offending food item (Bischoff & Crowe, 2005; NIAID; Sampson, 1999; Sicherer, 2014). Nutrition education on strict allergen avoidance, including how to interpret ingredient lists and food labels, is recommended (NIAID; Turnbull, 2014). Some allergens are known to cross-react with common species, and the restriction of cross-reactive foods is not recommended in the ED population due to the nutritional and psychological implications of extra restriction (unless the patient is at high risk for a severe reaction based on screening and diagnostic measures) (Turnbull, 2014). Severe allergic reactions can result from cross contamination, so there should be designated areas of the treatment facility kitchen and separate kitchen tools for the preparation of allergen-free foods. Nutrition education for the patient, as well as all treatment team members, should also encompass anaphylaxis prevention and treatment (NIAID). A lack of knowledge of the consequences of IgE or non-IgE food allergies (including CD) on the part of an ED clinician can compromise patient trust and health (Leffler, 2007). Patients should be provided with a medical alert bracelet, an adrenaline injector, and action plan in preparation of an accidental exposure (Turnbull, 2014; Sampson, 1999). There are no medications currently recommended to prevent IgE or non-IgE reactions from occurring (NIAID). The use of antihistamines can only lessen the severity of the reaction, but cannot treat anaphylaxis (Turnbull, 2014). Immunotherapy aimed to induce allergen tolerance is currently being researched as a potential treatment for IgE and non-IgE food allergies, but more information is needed before these therapies can be recommended to the already compromised ED population (NIAID). The clinical reactivity to allergens diminishes over time in 1/3 of adults and children with food allergies (Sampson, 1999). Thus, yearly assessment of allergy status via screening procedures previously discussed is recommended in hopes of potentially liberalizing the diet (Savage, 2015; Sampson, 1999). Especially for the psychologically vulnerable ED population, dealing with a FA can be particularly burdensome and can affect quality of life (Sicherer, 2014). Necessary food restriction may feed into already-established disordered beliefs, and can cause internal conflict during the seemingly simple task of feeding oneself, which can make any food-related digestive symptoms worse. ED patients with a comorbid FA require a high level of psychological and nutritional support. Remaining considerations for the treatment of individuals with a comorbid ED and FA is described in the next section.

Clinical Management of Eating Disorders without Comorbid Food Allergy

If the ED patient's FA screening tests come back negative, then treatment goals focus on nutritional rehabilitation and challenging disordered food beliefs in a safe environment. Gastrointestinal complaints during weight restoration are often confused as FA symptoms and are very common. One study reported that 97% of a hospital ED unit reported

gastrointestinal complaints upon admission (Boyd, 2010). Such complaints include heartburn, dysphagia, nausea, vomiting, bloating, gas, and constipation. Some discomfort during the refeeding period is to be expected, however guidance from an RD can help to avoid further discomfort that may interfere with food consumption. The RD can educate patients on the adjustment their body has to go through in order to properly digest food again. The body may feel overly full for about 2-3 weeks, but it is essential that patients keep eating despite their fullness or bloating (Hart, 2011). The body needs time to produce sufficient digestive enzymes and repopulate the gut flora to support digestion. In order to reduce symptoms, patients will have a gradual, structured introduction to all foods in the form of small frequent meals (Hart, 2011). Since enzymatic and micronutrient deficiency is suspected at this point, it is recommended to incorporate a wide variety of foods, but to also limit raw fruits and vegetables (Hart,2011). In general, raw foods are not dense enough in calories for this population and the high fiber content of these foods can worsen delayed gastric empting, pain, and bloating. If a client complains of lactose intolerance, lactase enzyme can be consumed with meals and snacks that contain dairy. Intolerance to dairy is a common side effect of malnutrition, but the body can adjust to handling dairy again if it is maintained as a component of their diets (Hart, 2011). Dairy not only provides potentially weak bones with much-needed calcium and vitamin D, but it also is a rich source of healthy probiotics. The ingestion of yogurt may improve immune markers and help to repopulate gut bacteria, which can help ameliorate gas and normalize stool movements (Hart, 2011). Therefore, it is not recommended for ED patients to exclude dairy from their diet, despite any initial discomfort it may bring. If constipation is a problem for the patient, moderate consumption of whole grains, bran, fluid, and prune products can help to relieve discomfort (Hart, 2011). The use of laxatives for constipation treatment is strictly avoided in the ED population. If patients abused laxatives prior to treatment, it is important to understand that acute cessation of laxatives can trigger severe sodium and water retention, which can result in serious cardiac consequences. It is important to be in communication with medical team members regarding electrolyte status of the patient, not only to assess cardiac risk, but to also make sense of gastrointestinal complaints. Many gastrointestinal symptoms occur due to the imbalance in electrolytes, and the normalization of electrolytes during the weight restoration period has the potential to reverse many gastrointestinal complaints, however the evidence is inconclusive (Bern, 2013). Some studies suggest that gastrointestinal complaints resolve after a period of nutritional rehabilitation (Perez, 2013), while others report that gastrointestinal symptoms persist even after weight rehabilitation (Boyd, 2010; Porchelli, 1998). Certain personality and psychiatric features may be predictive of prolonged gastrointestinal complaints in the ED population. Patients with higher levels of anxiety, somatization, neuroticism, and binge eating were all predictive of functional gastrointestinal disorders (Boyd, 2005; Perez, 2013). Given that gastrointestinal complaints may be amplified or more frequent in individuals with greater amounts of psychiatric distress, it is important for patients to continue to work with a therapist and

psychiatrist in effort to treat underlying issues. To support the patient's emotional state at the dinner table and beyond, it is important that they refeed in an environment that is predictable and low stress, with high amounts of support and encouragement.

4. Conclusion

An undefined relationship exists between food allergies and EDs. Research indicates that EDs are more prevalent in individuals with FA, but the opposite association may not be true. This brings to question why self-reported FAs are frequently observed in ED treatment centers, if FA incidence is not greater in the ED population. These self-reported "food allergies" may be psychosomatic, a consequence of impaired digestion, part of the ED pathology, or a misnomer for food intolerance. Future longitudinal studies are needed in order to better understand the progression and predictive factors between the two pathologies.

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