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The Gluten-Free, Casein-Free Diet In Autism: Results of A Preliminary Double Blind Clinical Trial

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This study tested the efficacy of a gluten-free and casein-free (GFCF) diet in treating autism using a randomized, double blind repeated measures crossover design. The sample included 15 children aged 2–16 years with autism spectrum disorder. Data on autistic symptoms and urinary peptide levels were collected in the subjects' homes over the 12 weeks that they were on the diet. Group data indicated no statistically significant findings even though several parents reported improvement in their children. Although preliminary, this study demonstrates how a controlled clinical trial of the GFCF diet can be conducted, and suggests directions for future research.

KEY WORDS: Autism; diet; gluten; casein; GFCF diet.

INTRODUCTION

Autism, the most classic form of the autism spectrum disorders (ASD), is a complex, life-long condition that appears to be increasing in prevalence (Fombonne, 2003). It can be devastating to families who are often ill-equipped to meet the challenges of caring for a child with autism (Hasting & Brown, 2002). Particularly difficult is communicating with the autistic child whose disorder is hallmarked by limited reciprocal social interactions, marked impairments in functional communication abilities, and a variety of unusual and sometimes dangerous behavioral patterns (Carr, 1985; Kanner, 1943; Rutter, 1978).

Exploration into the role of the immune system, genetic susceptibility, and environmental factors such as infections, vaccines, and diet has led to the emergence of numerous theoretical models to explain the biological basis for autism. In turn, a growing number of parents are implementing a variety of still unproven treatment modalities with their autistic children.

Testimonials throughout the United States and a number of other countries in addition to work by Cade and colleagues (Cade, Privette, Fregly, Rowland, Sun, Zele, Wagemaker, & Edelstein, 1999) have supported the efficacy of the GFCF diet that is free of wheat (which contains gluten) and milk (which has casein). Most noteworthy are parental and teacher reports of ASD children who, after being placed on the restricted diet, have been "cured" of their autism, having acquired language and showing marked improvement in social relatedness. To date, however, there is insufficient empirical data supporting these claims and no published reports of double blind controlled clinical trials that use the most widely accepted diagnostic instruments and direct behavioral observation to test dietary effects.

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Because autism has no clear etiology or cure, there is an urgent need to further science on this challenging, life-long disorder. Working collaboratively with the UF General Clinical Research Center (GCRC), our research team at the UF College of Nursing has identified and overcome a number of methodological issues and incorporated knowledge gained from three prior NINR/NIH-funded studies of ASD children to conduct an initial investigation regarding the effects of a gluten-free casein-free (GFCF) diet using well-accepted and standardized instruments in a randomized double blind clinical trial. Results of this study not only yield preliminary data regarding diet efficacy, they also provide important information on developing and implementing a GFCF diet and clarify a number of feasibility issues.

DIETARY TREATMENT OF CHILD BEHAVIORAL DISORDERS

Dietary treatment of children is not new. Beginning in the 1920s (Shannon, 1922), literature has reported on restricted diets and their effect on child behavior. Most famous is Feingold's work in the 1970s, in which he reported that at least 50% of hyperactive and learning disabled children improved when placed on diets free of salicylate and additives. In the early 1980s, other researchers reported adverse effects of sugar on hyperactive and aggressive behavior (Prinz, Roberts, & Hantman, 1980; Wolraich, Stumbo, & Millch, 1986). Over time, those who promote dietary treatment have combined these recommendations of Feingold (1975) and the later investigators, to restrict additives, preservatives, and sugars. Even though reports of dietary effects have public appeal, Wolraich (1996) urges clinicians to use caution when considering dietary restrictions. He notes that even though some parents firmly believe that diet greatly affects their children's behavior or learning, the evidence to date is not convincing. Wolraich also mentions that the power of suggestion on the part of parents can be very strong in situations affecting their children's behavior. For example, Hoover and Millch (1994) gave children a drink with artificial sweetener but told half of the parents that the drink was sweetened with sugar. The parents who thought their children had received the sugar-drink rated their children's behavior as significantly worse than those who were told their children received artificial sweetener. Thus, further sound, well-controlled research regarding dietary restrictions and their effect on child behavior is needed.

Background of the Gluten-Free and Casein-Free (GFCF) Diets

Initially focusing on schizophrenia, Cade and colleagues (Cade *et al.*, 2000) conducted a series of studies to test Dohan's hypothesis that schizophrenia is in some way associated with the absorption of "exorphins" contained in gluten and casein (Dohan, 1966). Dohan formed his hypothesis after studying the dietary habits of societies in New Guinea and other South Pacific Islands. Inhabitants of these areas ate diets free of wheat, rye, barley and oats and reportedly had fewer and less severe cases of schizophrenia than more developed western cultures with diets rich in gluten and casein-containing foods. Seeking to explain this observation, Dohan asserted that individuals with schizophrenia may have genetic defects that lead to an overload of peptides from milk protein (casein) and/or gluten. Normally, proteins found in milk and wheat products are metabolized into peptides, and then into amino acids which are absorbed by capillaries in the intestine. High peptide levels may be caused by excess production of peptides in the intestine resulting from abnormal intestinal permeability (D'Eufemia *et al.*, 1996). Focusing attention on β-CM, smaller peptides found in milk. Sun, Cade, Fregly, and Privette (1999) found that β-CM7 could cross the blood-brain barrier in rats and induce C-Fos activity in various regions of the brain, and alter behavior. The researchers assert that these effects can be reversed by an opioid antagonist (Sun & Cade, 1999).

Results of GFCF Dietary Clinical Trials in Autism

Expanding laboratory testing in applied settings, Cade *et al.* (2000) conducted a study of 270 individuals. One hundred and twenty of these participants were diagnosed with schizophrenia, and 149 met the DSM III criteria for a diagnosis of autism. All of the children with autism were treated with a GFCF diet, a synthesis of the *Milk Free Kitchen* by Kidder (1988) and the *Gluten-Free Gourmet* by Hagman (1990). During the study, parents, physicians and some teachers independently assessed the children for the presence and severity of the diagnostic manifestations of autism using a four-point Likert scale. These ratings were done initially and repeated after 1 month of treatment and then every 3 months for 1 year. Parent and physician reports were averaged, with

variability of individual observer scores reported as less than 10%. Blood samples were examined to measure the absorption of peptides contained in wheat products (gluten) and dairy (casein) and the associated antibodies IgA and IgG for each of these food products. The study found that 87% of the children with autism had high titer IgG antibodies to gliadin and 30% had high titer transindolylacryloylglycine (IgA) antibodies to gluten or casein. Treatment with a GFCF diet was accompanied by reports of improvement in 81% of children within 3 months. A strength of this work was the combined use of physiological and behavioral measures. The behavioral results are limited, however, by the heavy reliance on reports from parents and teachers who knew that the children were on the GFCF diet.

Recently published are three studies examining various hypotheses related to the GFCF diet in autism. Vojdani, Pangborn, Vojdani, and Cooper (2003) measured the antibodies IgG, IgM, IgA against CD26, CD69, streptokinase (SK), gliadin, casein, and ethyl mercury in 50 children diagnosed with autism. Analysis of blood samples revealed that a significant number of the children developed antibodies against casein and gliadin. In addition, SK, gliadin, casein, and ethyl mercury were shown to bind to the lymphocyte and tissue enzyme (CD26), and is thought to perhaps trigger inflammatory and immune reactions in children with autism.

In comparative study, Arnold, Hyman, Mooney, and Kirby (2003) evaluated amino acid patterns of 26 children with autism on a regular diet, 10 on a gluten–casein free diet, and 26 children with developmentally delays who served as controls. The children with autism in the study had higher deficiencies in essential amino acids compared to the control group. Although preliminary, these findings suggest that children with autism are at high risk for amino acid deficiencies and may benefit from a structured diet. Clearly, this is an area that warrants further investigation. The authors note that a major limitation in the study was the small sample. An additional concern is the lack of strict dietary control for children on gluten–casein free diet, a commonly encountered problem in conducting dietary research in children.

Knivsberg, Reichelt, Hoien, and Nodland (2002) and others conducted a randomized single blind study with 20 subjects to assess the effect of a gluten–casein free diet on children with autistic syndrome and urinary peptide abnormalities. The children in the control and experimental group were matched according to severity of autistic symptoms, age, and cognitive level. Changes were observed in both the control and experimental group; however, the experimental group showed more significant changes. There was statistical difference between the experimental and control group, demonstrating that the experimental group had improvement in autistic behavior, non-verbal cognitive level, and motor problems.

While results from these recent studies provide interesting information regarding hypothesized GFCF dietary effects on physiology, behavior and cognition, they are limited by small sample sizes. A need still exists for rigorous controlled clinical trials evaluating both physiological and behavioral effects.

Study Purpose and Specific Goals

Thus, the purpose of our overall research program is to provide critical feedback loops for families of children with ASD including the most current scientifically sound information and incorporating family observations into treatment planning and development. The goals of this pilot study were:

1. To evaluate the effects of a GFCF diet on the severity of autistic symptoms as measured by the Childhood Autism Rating Scale (CARS), Ecological Communication Orientation Scale (ECOS), and direct behavioral observation frequencies.
2. To evaluate the effects of a gluten-free, casein-free diet on urinary peptide levels.
3. To evaluate the role of parent behavior in therapeutic and placebo effects of a gluten-free, casein-free diet in children with autism.

METHOD

Participants and Settings

A total of 15 children with ASD (chronological age range 2–16 years; mean age 7.32 years, SD 4.1 years) were chosen as participants by purposive sampling from the Center for Autism and Related Disabilities (CARD) and/or Child Psychiatry Services at the University of Florida's Department of Psychiatry and Brain Institute. Inclusion was based on a diagnosis of autistic disorder according to DSM IV criteria (American Psychiatric Association, 2000) and a score above cut-off on each symptom domain of the Autism Diagnostic Interview Revised (ADI-

R). The sample included 12 boys and 3 girls. Thirteen of the children completed the 12-week protocol. One child was Asian and the remainder, Caucasian. Parents of all participants provided signed informed consent. Children were excluded from the study if their medical histories and/or physical examinations indicated that they had physical or sensory-impairments or significant medical problems including celiac disease. There were two settings: the General Clinical Research Center where the children were weighed weekly and the children's homes.

Instrumentation for Description of Subjects

Two instruments were used to describe the subjects: the CARS, and the Autism Diagnostic Interview-Revised (ADI-R).

Childhood Autism Rating Scale (CARS)

Used to assess autistic features, the CARS consists of 15 items and a 7-point likert scale. Each item contributes equally to the total score, which can range from 15 to 60. CARS items cover the following behaviors: relationships with others; imitation, emotional expression, body use, peculiarities in object use; resistance to change; visual, auditory, and tactile responsiveness; anxiety; verbal and nonverbal communication; activity level; and intellectual ability. The instrument's validity has been assessed as good under various conditions. Its interrater reliability has been well documented (Schopler, Reichler, DeVellis, & Daly, 1980; Schopler, Reichler, & Renner,1986), and the CARS manual reports a test–retest reliability of .88 on total CARS scored based on 91 cases over a 12-month-period. The α coefficient for the total score is reported as .94. After observing the child during a structured activity, an evaluation team completed the CARS 7-point scale for each of the 15 items and reached consensus.

Autism Diagnostic Interview—Revised (ADI-R)

The ADI-R is a semistructured interview for caregivers of individuals with autism; it is based on the ICD-10, which is used for the differential diagnosis of pervasive developmental disorders. The ADI-R covers the following three areas: Impairment in reciprocal social interaction (SI), communication (CO), and repetitive behaviors and stereotyped patterns (RB). A diagnosis of autism is established if an individual is scored at or above the cut-off score in the three ICD-10 symptom domains, and the abnormality is present at or before 36 months. Lord, Rutter, and Le Couteur (1994) report interrater reliability from .62–.89. The mean weighted κ levels across all items ranged from .73–.78. For six rater pairs the mean weighted kappa for algorithm items were above .75. The mean percentage agreement across all the items for each pair ranged from 90 to 93%. The Cronbach α was calculated to assess internal consistency. The item-total correlations for social area ranged from .54 (direct gaze) to .77 (quality of social overtures) with α .95. The item-total correlations for restricted and repetitive behaviors ranged from .30 (compulsions & rituals) to .53 (unusual sensory behaviors) with reported α of .69. The item correlations for the 11 verbal subjects ranged from −.06 (inappropriate questions) to .77 (instrumental gestures) with α .85. Communication items for all subjected showed item-total correlation ranging from .45 (imitative social play) to .70 (conventional, instrumental gestures) with α .84. The first author was trained by, and established reliability with, the instrument developers prior to conducting this study.

Instruments and Procedure for Measuring Dependent Variables

The following instruments were used to measure dependent variables. (See Table I for instrument administration sequencing.)

Childhood Autism Rating Scale (CARS)

Along with using the CARS to screen participants, we also used it to make a baseline assessment of the participant. This rating was compared with the CARS ratings from both the control and experimental dietary conditions.

Urinary Peptide Levels (UPL)

Urine samples were obtained at baseline (Day 1), on Day 21, on completion of D1 (Day 42), on Day 53, and at the end of D2 (Day 84). Each sample was analyzed to determine levels of the urinary casein and gluten peptides, casomorphin and gliadorphin, respectively. Normal results for the peptide creatinine ratio are $<.95$.

Ecological Communication Orientation (ECO) Language Sampling Summary

The ECO Language Sampling Summary (MacDonald, Gillette, & Hutchinson, 1989), or "ECO

Table I. Sequence of Dietary Intervention and Measurement

		WK1	WK2	WK3	WK4	WK5	WK6	WK7	WK8	WK9	WK10	WK11	WK12
Group A	BL	D1	D1	D1	D1	D1	D1	D2	D2	D2	D2	D2	D2
Group B	BL	D2	D2	D2	D2	D2	D2	D1	D1	D1	D1	D1	D1
Instruments:													
UPL	X (day 1)			X			X			X			X
CARS		X					X						X
ECOS		X					X						X
IHO		X					X						X

BL, Baseline period after consent was obtained and the start of the first intervention condition (either D1 or D2). Used to obtain behavioral profile of child.
D1, Administration of the regular diet.
D2, Administration of the gluten-free, casein-free diet.

Scales'', was employed as in Elder (1995) to record child behavior and collect interactive samples. Reliability and validity of the ECO Scales has been established by MacDonald, Gillette, and Hutchinson (1989) who report content, construct, and concurrent validity for various subsets of their ECO Scales. Concurrent validity has been assessed using Bzoch and League's (1970) *Receptive-Expressive Language Scale*, (REEL). Using .40 as a minimum criterion, MacDonald and his associates noted a moderately high correlation between the REEL and the ECO Scale for four different occasions following onset of treatment. ECO Scale reliability is also reported: interrater agreement (91.2), stability (overall, for all components = .60; range = .28–.86), and internal consistency using split halves method (overall r's = .93).

In-Home Observation

Behavioral response categories from the author's previous studies (Elder, 1995; Elder & Goodman, 1996; Elder, Valcante, Won, & Zylis, 2003) were combined to provide a more comprehensive constellation of behaviors for the proposed research questions. The child behaviors included *child initiating, child responding*, and, *intelligible words spoken*. Also coded were *parent initiating, parent responding*, and *parent expectant waiting* (an operationally defined measure of parental signaling and waiting for a specific child response). A research assistant trained in videotaping techniques and maintaining unobtrusiveness during home visits, videotaped each child interacting with his/her primary care-taking parent for 15 minutes during unstructured sessions. Using these videotapes, trained coders obtained behavioral counts of parent and child behaviors. Coders did not know the status of the subjects with regard to treatment phase (placebo vs. GFCF diet). Data were collected at three points in time: immediately before the diet began, at the end of the first dietary condition (Week 6) and again at the completion of the study (Week 12). Ratings of the parent and child were conducted independently and behavioral frequency counts determined by the number of occurrences divided by the length of the session (10 minutes). Coding and analysis of target behaviors was facilitated using the Multi-Option Observation System for Experimental Studies (MOOSES) (Tapp, 1996) program for the field collection and analysis of observational data. To minimize the potential for bias, two independent raters coded the tapes. Interrater agreement ranged from .82 to 1.0.

Sequencing of Baseline, Experimental and Control Conditions (See Table I)

After obtaining parental consent and following a screening evaluation, the GCRC data manager randomly assigned participants who met inclusion criteria to either the GFCF or a placebo diet. Children, parents, and all of the investigative team except for the data manager and dietician were blind to the dietary order. Dietary assignment was counterbalanced across subjects. Unlike most crossover clinical trials, the diets in our study did not require a washout period according to our dietary consultants. Table I is a timeline for the protocol that we have described in the text.

Development and Monitoring of Dietary Intake

Under the direction of the University of Florida's GCRC Bionutrition staff, participants were provided all meals and snacks from the GCRC's Metabolic Kitchen for 12 weeks. The gluten- and casein-free experimental diet was adapted to each child based on individual food preferences. Parents

received a 3- to 4-day supply of food twice each week from the GCRC. In addition, parents were given a list of allowed foods in case of emergencies, and they were asked to record their child's diet intake to monitor compliance. Each participant's regular diet was provided during the baseline period and for 6 weeks as the control diet. The experimental diet consisted of gluten- and casein-free counterparts food items in the control diet to insure that the parents and observers were blinded. Menus were analyzed for nutritional adequacy, and if necessary, a vitamin and/or mineral supplement was provided in order for all of the children to meet the Recommended Dietary Allowances (RDA) for his or her age. Nutrient calculations were performed using the Nutrition Data System for Research (NDS-R) software version 4.05, developed by the Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN, Food and Nutrient Database 33, released July 2002.

Statistical Design and Methods

Design

As previously noted, this pilot study was a randomized, double blind repeated measures cross-over design of 13 children comparing two diets (regular diet vs. GFCF diet) in terms of autism symptoms as measured by the CARS, ECOS, frequencies of social initiating, social responding, intelligible words spoken, and non-speech vocalizations. The primary measure of efficacy was the CARS, which was measured at baseline, week 6 (at the end of D1) and week 12. Urinary peptide levels (UPLS) were measured at five points (See Table I).

Analysis

This study was primarily a pilot study. For each treatment comparison, we used a two-sided, two sample *T*-test constructed as follows. Irrespective of the treatment order assignment, the dependent variable was the value in period 2 minus the value in period 1. The mean for the AB ordering was then compared to the mean for the BA ordering. The primary dependent measures were ECO and CARS. Half the difference in the group means is an unbiased estimate for the main effect of treatment, whether or not there is treatment by period interaction. This has an advantage over using a one-sample *T*-test on the Treatment1−Treatment0 difference that ignores order, since treatment by period interaction (carry-over effects) have a negative impact on the one-sample version, both in terms of bias when sample sizes for each order differ, and in terms of a larger variance. We employed a missing at random model for the three of 15 subjects whose week 12 or week 6 data were incomplete on a major variable (CARS or ECOS).

RESULTS

Frequencies and standard deviations of the dependent variables are presented in Table II. While several of the mean scores within each treatment condition appear discrepant, group analysis results indicated no significant differences with CARS ($p = .85$), ECOS ($p = .29$), or behavioral frequencies ($.32 < p < .45$) and no significant differences in grouped data for urinary peptide levels of gluten ($p = .44$) and casein ($p = .11$). In addition, there were no statistically significant differences in observed parent behaviors ($.97 < p < .98$), indicating no detectable parental behavioral influence and/or confound. Interestingly, some anecdotal reports varied from the non-significant findings. For example, parents of seven children reported that there were marked improvements in child language, decreased hyperactivity and decreased tantrums. Further, parents of nine children decided to keep the children on the GFCF diet even though there was no empirical support for continuing. Also interesting were the unsolicited reports of one teacher and one respite worker who claimed to observe language and behavioral improvements in two of the children. Before unblinding, parents were asked to comment on whether they thought their child was on the GFCF diet the first or second 6 weeks. Five were correct, two had "no idea", and six were incorrect.

DISCUSSION

It is important to mention that the statistically non-significant findings may be the effect of small sample size, and/or large within-group variance. Clearly, this indicates the need to replicate the study with larger and less heterogenous samples. It is also interesting that even though grouped data were non-significant for each of the dependent variables, behavioral and language improvement could be seen in individual children. A common problem in the study of children with autism is the wide variability among the children regarding behavioral and developmental traits and levels. Clearly this was a heterogeneous sample in reference to age, severity of autism, and

Table II. Mean and Standard Deviation by Group

Group variables	Week 6	Week 12	Difference (week 12−week 6)
A	No Treatment	Treatment	
ECO	174.4 + 86.0 ($n = 7$)	162.9 ± 108.8 ($n = 8$)	−4.1 ± 68.0 ($n = 7$)
CARS	31.2 + 8.7 ($n = 7$)	33.5 ± 8.4 ($n = 8$)	2.0 ± 7.7 ($n = 7$)
Behavioral response frequencies:			
Child initiating	7.5 + 6.1 ($n = 8$)	10.8 ± 8.4 ($n = 8$)	3.3 ± 8.4 ($n = 8$)
Child responding	14.3 + 6.5 ($n = 8$)	11.9 ± 4.5 ($n = 8$)	−2.4 ± 7.7 ($n = 8$)
Intelligible words	24.0 + 43.5 ($n = 7$)	30.9 ± 36.0 ($n = 7$)	6.9 ± 17.0 ($n = 7$)
Parent initiating	71.6 + 34.9 ($n = 7$)	64.3 ± 30.4 ($n = 8$)	−3.4 ± 26.3 ($n = 8$)
Parent responding	20.1 + 13.6 ($n = 7$)	20.3 ± 7.3 ($n = 8$)	−0.8 ± 16.4 ($n = 8$)
Expectant waiting	1.9 + 2.7 ($n = 7$)	2.3 ± 2.2 ($n = 7$)	0.4 ± 3.0 ($n = 7$)
B	Treatment	No Treatment	
ECO	175.8 ± 86.4 ($n = 6$)	111.6 ± 46.6 ($n = 5$)	−42.2 ± 38.1 ($n = 5$)
CARS	33.6 ± 8.6 ($n = 7$)	37.5 ± 6.6 ($n = 5$)	1.2 ± 6.0 ($n = 5$)
Behavioral response frequencies:			
Child initiating	9.5 ± 9.6 ($n = 6$)	5.2 ± 3.2 ($n = 5$)	−0.6 ± 4.5 ($n = 5$)
Child responding	27.7 ± 21.8 ($n = 6$)	15.0 ± 15.0 ($n = 5$)	−8.4 ± 7.0 ($n = 5$)
Intelligible words	26.8 ± 35.1 ($n = 6$)	12.4 ± 14.2 ($n = 5$)	−2.6 ± 12.4 ($n = 5$)
Parent initiating	61.2 ± 37.0 ($n = 5$)	56.8 ± 48.7 ($n = 4$)	−3.3 ± 24.2 ($n = 4$)
Parent responding	15.2 ± 11.6 ($n = 5$)	11.0 ± 4.8 ($n = 4$)	0.8 ± 1.9 ($n = 4$)
Expectant waiting	2.3 ± 1.4 ($n = 6$)	2.0 ± 2.9 ($n = 5$)	−0.8 ± 2.2 ($n = 5$)

cognitive abilities and thus it was difficult to draw meaningful conclusions about the group as a whole.

While planning this study, many were skeptical regarding whether it would be possible to develop and implement a double blind condition using GFCF and placebo diets. Interestingly, we found that eight of the parents were unable to correctly distinguish the placebo and experimental diets. This indicates that we were able to successfully produce and implement a GFCF placebo diet and has important implications for future dietary research.

LIMITATIONS

As mentioned, results from our work are preliminary and indicate the need for future research to address some of the limitations we encountered. For one, the sample size was small and heterogeneous, thus, possibly contributing to a Type 2 error. Future study should include either larger, more homogeneous samples, or in-depth individual study using rigorous intrasubject, single subject experimental measures with replication across subjects.

Also, even though most parents were conscientious regarding the dietary restrictions, there were several reports of children "sneaking food" from siblings or classmates. Ideally, it would be important to replicate the study in a more controlled educational or inpatient setting and/or extend data collection for a longer period of time, anticipating that there may be non-compliance incidents and thus a need for wash-out periods during the course of the study. Another reason for possibly extending the 12-week protocol is that there are clinical reports of some children who respond to the GFCF diet quickly, while others take several weeks before behavioral effects are detectable. Furthermore, labels should be read carefully as there is some indication that soy products may affect urinary peptides and thus introduce confounds (Schakel, Sievert, & Buzzard, 1988).

We also discovered that while CARS was a useful screening instrument, it may not have detected some of the more subtle changes reported by families. Future research should include a combination of direct observational methods as well as several additional instruments suited to repeated measures designs that have well-established psychometrics.

Finally, there has been some speculation regarding parental placebo effects related to the GFCF diet effectiveness. This may account, at least in part, for families reporting improvements that were not empirically supported by our work and choosing to continue the diet even after being told the results of our study. This is certainly another area needing further exploration because parents, and the

decisions they make, are critically important to their children's welfare.

SUMMARY

In summary, although grouped data from this preliminary study were statistically non-significant, results have revealed several interesting findings and methodological discoveries. Clearly, this was a difficult study to conduct but undoubtedly viewed as important by the participating families. Several suggestions have been made regarding the need for additional research to help these families who are desperately searching for answers.

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