Dietary treatment for Epilepsy

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Dietary Treatments for Epilepsy

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Health and Culture

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Abstract

Dietary treatments for epilepsy have been used since the early 1920s, however, the use of these treatments has been replaced by anticonvulsant drugs. In the past ten years there has been a reemergence of the use of dietary treatments for epilepsy. These dietary treatments are referred to as Ketogenic Diets. There are three types of ketone diets: Classic Ketogenic Diet (KD), Medium-chain-triglyceride Ketogenic Diet (MTC), and the Modified Atkins Diet (MAD). These dietary treatments utilize a high-fat, adequate protein and very low carbohydrates diet to control seizures. The purpose of this report is to provide a comprehensive overview of dietary treatments for epilepsy. I will discuss the history of dietary treatments for epilepsy, the three types of treatments, clinical application, and the patients currently using the treatment. In addition to scholarly discourse about these treatments, I will provide a detailed account of my personal experience using one of these dietary treatments to for my epilepsy.

Introduction

Altering a patient’s diet to reduce seizure activity precedes modern medicine. The Greek royal physician Erasistratus wrote “One inclining to fits should be made to fast without mercy and be put on short rations” (Fredricks, 2012). This concept of fasting to reduce seizures continued into the 20th century. By 1921, fasting as a form of treatment for epilepsy became a common medical practice (Fredricks, 2012). The use of fasting eventually evolved into the Ketogenic Diets (KD) used today. However, the use of dietary treatments did not last long.

The use of Ketogenic diets began to decline in 1938, when H. Huston Merritt and Tracy Putnam discovered Phenytoin1. Since 1938 the use of Antiepileptic Drugs (AED)2 has become

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1 Phenytoin, marketed as Dilantin, is an enzyme producing AED.
2 For complete list of AEDs used see Appendix Table.
the first line of treatment for most seizure disorders. AEDs can have serious side effects\(^3\), including but not limited to: central nervous system toxicity and hypersensitivity reactions (Schmidt, 2009). In addition to adverse side effects, the use of AEDs increases the risk of drug interactions. These side effects and drug interactions are increased when more than one AED is used. AED “cocktails” are commonly used to achieve seizure control. Approximately 15-30% of people treated with AEDs require two or more AEDs to achieve seizure control (Schmidt, 2009). AED “cocktails” also increase the risk of plasma concentrations to toxic levels and tremors (Schmidt, 2009). The health risks related to the long term use of AEDs includes liver and kidney failure, and cognitive degeneration (Eddy et al., 2011).

Currently, despite their proven effectiveness, dietary treatments are used as a last resort. Ketogenic diets are usually used in children with refractory epilepsy, only after two or more AEDs and surgery have been proven ineffective. In an article published in the journal of Epilepsy & Behavior, Schmidt (2009:56) suggests “…if refractory epilepsy is confirmed, surgical options should be considered in suitable candidates.” Surgery is invasive and the long term use of AEDs is detrimental to a person’s health. AEDs do not favorably affect the course of underlying epilepsy (Dieter, 2009).

Dietary treatments are equally, if not more, effective in achieving seizure control, with limited adverse side effects. Ketogenic Diets have been found to be highly effective in children, 30-60% of children experience at least a 50% reduction in seizures (Lee & Kossoff, 2011). Recent studies have shown that dietary treatments also significantly reduce the incidence of seizures in adults. The use of Ketogenic diets as a treatment for epilepsy can provide a more comprehensive and safer long-term treatment for seizure disorders. Ketogenic diets should be considered in all cases, not just individuals with refractory epilepsy. The documented efficacy of

\(^3\) For complete list of side effects see Appendix Table 2.
ketogenic diets as a treatment for epilepsy supports the use of KD as a clinical treatment for all epilepsies. The purpose of this report is to provide an overview of the three KD treatments for epilepsy and present their clinical applications.

**Methods**

This research paper is a synthesis of scholarly articles, popular media, field research, interviews, and participation observation. I used online libraries such as, JSTOR, Wiley Online and Elsevier to obtain relevant scholarly articles. In addition to reviewing scholarly materials I used informational web sites such as the CDC to obtain statistical information. The field research conducted for this paper consisted of monitoring online communities, and “yahoo groups” that focused on the use of a Ketone based treatment for epilepsy. Three interviews were conducted for this research paper. The first interview was a parent who is utilizing the MTC-KD diet for her 9-year-old son; the second is a 20-year-old college student who is using the MAD; lastly a nurse practitioner at Emory University Hospital’s neurology department. To maintain the anonymity of the people I interview I refer to them as Interviewee1, Interviewee2 and EmoryNP, respectively. For the purposes of participant observation I started the MAD.

My use of the MAD was conducted without the supervision of a healthcare professional. I used the diet parameters established by Kossoff & Dorward (2008). To monitor my daily fat, protein and carb intake I used an online food log called myfitnesspall.com. I used ketone test strips to monitor daily ketone levels in my urine. I maintained a diary of my weight, ketone levels and experiences. Vital statistics such as lipids, cholesterol, micronutrients, weight and blood pressure were taken on February 31, 2014. There was no follow up blood work done at the end of the participant observation period.

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4 Interviews were conducted through yahoo messenger, an online chat application.
Epilepsy

Epilepsy is a chronic neurological disorder characterized by reoccurring seizures. These seizures are caused when neuronal activity becomes disturbed. During a seizure, disturbances of nerve cell activity produces symptoms that vary depending on which part, and how much, of the brain is affected (De Boer et al., 2008). Seizures may produce changes in awareness or sensation, involuntary movements, or other changes in behavior. Usually, a seizure lasts from a few seconds to a few minutes (Center for Disease Control, 2013).

Seizures can be placed into two broad categories: 1) Focal, where the abnormal activity is in just one area of the brain and 2) Generalized, these seizures result from a disturbance throughout the entire brain (Mayo Clinic, 2013). Each category has a specific electroencephalogram (EEG) signature. Within each of these categories there are different types of seizures. Focal seizures can manifest in two ways, simple focal⁵ and dyscognitive focal⁶. Generalized seizures can manifest in six different ways, Absence⁷, Tonic⁸, Clonic⁹, Myoclonic¹⁰, Atonic¹¹ and Tonic-Clonic¹² (Mayo Clinic, 2013). Seizure types and/or categories are not mutually exclusive, focal seizures can become generalized and/or a person can have multiple types of seizure within a specific category.

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⁵ Simple focal seizures may alter emotions or change the way things look, smell, feel, taste or sound. They may also result in involuntary jerking of a body part, such as an arm or leg, and spontaneous sensory symptoms such as tingling, dizziness and flashing lights (Mayo Clinic, 2013).

⁶ Dyscognitive seizures alter consciousness or awareness and may cause you to lose awareness for a period of time. Dyscognitive focal seizures often result in staring and purposeless movements — such as hand rubbing, chewing, swallowing or walking in circles (Mayo Clinic, 2013).

⁷ Absence seizures, also called petit mal seizures, are characterized by staring and subtle body movement. These seizures can cause a brief loss of awareness (Mayo Clinic, 2013).

⁸ Tonic seizures cause stiffening of your muscles. These seizures usually affect muscles in your back, arms and legs and may cause you to fall to the ground (Mayo Clinic, 2013).

⁹ Clonic seizures are associated with rhythmic, jerking muscle movements. These seizures usually affect the neck, face and arms (Mayo Clinic, 2013).

¹⁰ Myoclonic seizures usually appear as sudden brief jerks or twitches of your arms and legs (Mayo Clinic, 2013).

¹¹ Atonic seizures, also known as drop seizures, cause a loss of muscle control, which may cause you to suddenly collapse or fall down.

¹² Tonic-clonic seizures, also called grand mal seizures, are characterized by a loss of consciousness, body stiffening and shaking, and sometimes loss of bladder control or biting your tongue.
Epilepsy is the fourth most common neurological disorder (Neal & Cross, 2010). In 2012 the CDC estimated that about 2.3 million adults and 467,711 children in the United States have epilepsy (Center for Disease Control, 2013). Nearly 150,000 Americans develop the condition each year (Center for Disease Control, 2013). New cases of epilepsy are most common among children and older adults (Center for Disease Control, 2013). Despite the use of AEDs 30% of epileptics have refractory epilepsy\(^{13}\) (Lee & Kossoff, 2011). In addition to people who have refractory epilepsy, it is estimated that one-third of all people with epilepsy will develop intractable epilepsy in their life time (Johns Hopkins Medicine, 2012). Approximately half of the people diagnosed with epilepsy have no identifiable cause of the disease (Mayo Clinic, 2013). If a cause can be determined it is likely to be: genetic influence such as heredity, head trauma, brain conditions such as tumor or stroke, infectious disease, birth defect, and/or developmental disorder (Mayo Clinic, 2013).

Epilepsy is diagnosed, treated and monitored by a neurologist. Epilepsy is not considered a disease that can be “cured”. A person can be considered “seizure free” but is not considered cured of epilepsy. There are three major treatments for epilepsy, anticonvulsant medicine, surgery, and dietary treatments. Anticonvulsants and surgery are the most common forms of treatment; dietary treatments are used as a last resort.

**History of Dietary Treatment for Epilepsy**

Dietary treatments for epilepsy have been around since ancient Greece. Ancient Greek physicians treated epilepsy, by altering their patient’s diet. In the book “Epidemics,” Hippocrates describes the case of a man whose epilepsy was cured with drastic diet and fasting (Fredricks, 13 Refractory epilepsy refers to seizures that are not controlled by Antiepileptic drug. Refractory epilepsy is sometimes referred to as, “drug resistant” or “intractable”.}
Using fasting as a treatment continued into the 20th century. The first recording scientific study of dietary treatment of epilepsy was in France in 1911 by Guelpa and Marie (Wheless, 2004). In the study twenty epilepsy patients were given a low-calorie vegetarian diet combined with periods of fasting and purging. Due to lack of participant compliance, the study did not yield successful results. Despite the failure of the French study in 1911 the use of fasting continued to be used. In the early 20th century fasting had become common medical treatment for epilepsy. This is reflected in Dr. McMurray’s letter to the New York Medical Journal describing digestive disturbances as an impressive finding in his patients with epilepsy and the use of fasting followed by a starch- and sugar-free diet as a treatment beginning in 1912 (Wheless, 2004). In 1920 osteopathic physician Hugh Conklin, treated his epileptic patients by recommending thy fast for eighteen to twenty-five days (Fredricks, 2012). Conklin’s fasting treatment had promising results, 20% of his patients experienced complete retraction of seizures and 50% saw a marked reduction in seizure frequency (Wheless, 2004). Although fasting was being used to treat epilepsy, the medical community did not know why the process worked.

One year after Conklin published the results of his work with fasting as a treatment for epilepsy, Lennox and Cobb conducted a small study of five children that would lead to the development of the KD. In 1921, during a study of the efficacy of fasting, conducted by Lennox and Cobb chemical analysis of the blood and urine were performed. The participants of Lennox and Cobb’s study all showed an increase in serum uric acid and acidosis, which typically developed after two or three days (Wheless, 2004). The increase in serum uric acid and acidosis and was accompanied by a decrease in seizures (Wheless, 2004). Lennox and Cobb deducted that dehydration, ketosis, and acidosis were used to explain the efficacy of fasting (Wheless, 2004). Although their assumptions were wrong, the evidence collected was invaluable towards the
development of the Ketogenic diets.

At about the same time as the study conducted by Cobb and Lennox, Dr. Woodyatt published an article about diet adjustments and diabetes (Wheless, 2004). Wheless (2004) provides an excerpt from Woodyatt’s article.

…acetone, acetic acid, and beta-hydroxybutyric acid appear in a normal subject by starvation, or a diet containing too low a proportion of carbohydrate and too high a proportion of fat. It [ketoacidosis] appears to be the immediate result of the oxidation of certain fatty acids in the absence of a sufficient proportion of ‘oxidizing’ glucose.

This article by Woodyatt, linked high-fat/low-carbohydrate to fasting.

Inspired by Dr. Woodyatt’s findings, Dr. Wilder at the Mayo Clinic proposed the benefits of fasting could be obtained by a diet “containing too low a proportion of carbohydrate and too high a proportion of fat” (Wheless, 2004). Wilder suggested that a ketogenic diet should be as effective as fasting and could be maintained for a much longer period (Wheless, 2004). By 1928 the Mayo Clinic was using the Ketogenic diet as a treatment for epilepsy (Wheless, 2004). In 1928, Dr. Talbot, of the Mayo Clinic, wrote that “the best therapeutic results in epilepsy are not obtained until the ratio has approached 4:1…” (Wheless, 2004:34) Talbot’s suggested ratios are still used today.

From 1911 to the mid-1930s dietary treatments for epilepsy were widely used, with a focus on the ketogenic diet in the 1920s and early-1930s. However, the discovery of diphenylhydantoin by Merritt and Putnam in 1938 shifted focus from dietary treatments to new AEDs. Pediatric neurologists and epileptologists were led to believe that better understanding of central nervous system neurotransmitters and rationally designed AEDs were the hope for the
future. Fewer children were placed on the ketogenic diet, resulting in fewer dieticians who were trained in the initiation and maintenance of the diet (Wheless, 2004:42). Although, no longer popular, research with KIDs continued at Johns Hopkins. In 1971, Huttenlocher et al., introduced a medium-chain triglyceride (MCT) oil diet that was more ketogenic per calorie, allowing less restriction of other foods. Apart from Johns Hopkins, the use of the Ketogenic diet drifted into obscurity until the early 1990s.

In October 1994 NBC’s Dateline reported on a 2-year-old child named Charlie Abrahams. Charlie, the son of Hollywood producer Jim Abrahams, suffered 60-100 seizures weekly (Wheless, 2004). Charlie’s seizures remained uncontrolled despite the use of multiple AEDs, however, Charlie was able to achieve total seizure control using the KD (Wheless, 2004). The Abraham’s experience inspired them to create The Charlie Foundation. The Charlie Foundation promotes the diet and research. In 1994, the efforts of the Charlie foundation helped spearhead a multicenter study. The KD received more Hollywood attention in 1997, when Abrahams produced a TV movie called First do no Harm, starring Meryl Streep (Wheless, 2004). Since the 1990s the KD has experienced a significant reemergence, and modern clinical studies have established the treatment as significantly effective. The renewed interest in the KIDs, has led to the development of the Modified Atkins Diet (MAD) as an alternative to the Classic Ketogenic Diet (CKD) and the medium-chain triglyceride ketogenic diet (MTC).

**Dietary Treatments**

Since the revival of ketogenic diets there are seventy-five centers in over forty-five countries (Johns Hopkins Medicine, 2012). Studies in children show the CKD reduces seizures by more than 50% in half of the patient, and by more than 90% in a third of patients who use the treatment (Vining & Hartman, 2007). KIDs are usually administered to children with refractory...
epilepsy. However, recent studies have shown KDs provide comparable seizure reduction in adults as they do in children. Unfortunately, adults have higher rates of noncompliance with the CKD and MTC (Sirven et al., 1999). Research indicates that adults comply with the less restrictive regimen of the MAD. All of the KDs require vitamin supplements to compensate for the unbalanced diet. The efficacy of the KDs are without question, however, the mechanisms that make the diet effective is still unknown.

Ketogenic diets simulate the same effect as fasting, by producing ketone bodies, \( \beta \)-hydroxybutyrate, acetoacetate, and acetone (Bough & Rho, 2007). An increase in ketone bodies produces a state of ketosis. Once the body shifts into a state of ketosis, the brain begins to use ketones from the metabolism of fats as energy rather than glucose (Bough & Rho, 2007). This metabolic process has an anticonvulsant effect. It is believed, but unconfirmed, that ketone bodies and responsible for the anticonvulsant effect. All of the KDs produce ketone bodies, but the administration and structure of the diets differ. Each type of KD diets has been shown to be more effective with specific types of seizure disorders than with others.

**Classic Ketogenic Diet**

The Classic Ketogenic Diet was the first ketone based dietary treatment for epilepsy. It is designed as a high-fat, adequate-protein, low-carbohydrate diet. The CKD is based upon consumption of long-chain triglycerides, in a 3:1-4:1 ratio of fats to carbohydrates + protein by body weight (Bough & Rho, 2007). The CKD is the most effective independent of factors such as age, seizure type and EEG pattern (Kossoff, 2004). However, it is the most restrictive of all the KDs, and therefore usually recommended for children whose meals are prepared for them. Most clinicians consider the MAD to be more appropriate treatment for adolescence and adults (Bergqvist et al., 2009). The Classic ketogenic diet has shown to be remarkably successful with
specific seizure disorders such as Dravet syndrome\textsuperscript{14}, infantile spasms, Lennox- Gastaut Syndrome\textsuperscript{15} myoclonic-astatic epilepsy\textsuperscript{16} and tuberous sclerosis\textsuperscript{17} (Bergqvist et al., 2009). The CKD can be administered in formula form, making it ideal for infants and individuals being tube-fed. The CKD and all KDs should be avoided in individuals with genetic disorders of fat metabolism (Bergqvist et al., 2009).

The CKD is administered with the assistance of medical professionals. There are several preliminary steps prior to the initiation of the diet\textsuperscript{18} (Lee & Kossoff, 2011). Once the preliminary evaluations are completed the patient is admitted to the hospital for a fasting period. During this fasting period patients are given calorie free fluids and a ketogenic formula (Dorward & Kossoff, 2008). During the hospital stay patients ketone and glucose level are monitored.

The CKD is an intensive medical therapy with side effects. These side effects are generally less severe and frequent than those of AEDs. Many of the side effects of the CKD can be treated by changing to a different KD and/or “tweaking” the diet. These side effects include but are not limited to: constipation, low-grade acidosis, kidney stones, weight loss and hypoglycemia (Bailey et al. 2005, Kossoff 2004, Dorward & Kossoff, 2008). Many of these side effects are shared by all of the KDs.

\textit{Medium-chain triglyceride ketogenic diet}

The MCT-KD is a version of the CKD that uses medium-chain triglycerides instead on long-chain triglycerides. In 1971, Huttenlocher et al. discovered that medium-chain triglycerides

\textsuperscript{14}Dravet syndrome, also known as Severe Myoclonic Epilepsy of Infancy (SMEI), is a rare and catastrophic form of intractable epilepsy that begins in infancy. Initial seizures are most often prolonged events and in the second year of life other seizure types begin to emerge (Mayo Clinic, 2013).

\textsuperscript{15}Lennox-Gastaut syndrome is a form of severe epilepsy that begins in childhood. It is characterized by multiple types of seizures and intellectual disability (Johns Hopkins Medicine, 2012).

\textsuperscript{16}MAE is resistant to medication, and is a difficult disorder to manage. Patients experience several seizures daily (Johns Hopkins Medicine, 2012)

\textsuperscript{17}Tuberous sclerosis complex (TSC) is a genetic disorder that causes non-malignant tumors to form in many different organs, primarily in the brain, eyes, heart, kidney, skin and lungs (Johns Hopkins Medicine, 2012)

\textsuperscript{18}Planning required to initiation of CKD (Lee & Kossoff, 2011)
produced more ketone bodies per unit of energy than normal dietary fats (Wheless, 2004). The MCT-KD produced ketone bodies faster than the CKD, therefore requiring less fat and a greater proportion of carbohydrates leading to more food choices and larger portion sizes (Rho et al., 2009). The MCT-KD uses a medium-chain triglyceride oil to provide 60% of the total dietary fat required (Miranda et al., 2012). The MCT oil is usually mixed with skim milk or incorporated into food.

The MCT oil caused gastrointestinal irritation, nausea and vomiting. The MCT-KD ratio was then changed to, 30% MCT oil, 30% long-chain triglycerides, 10% from protein and the remaining calories from carbohydrates (Levy et al., 2012). The alteration of the MCT-KD provided patients with a tolerable alternative to the restrictive CKD. The MCT-KD diet replaced the CKD in many hospitals. The MCT-KD provides larger portion sizes and more carbohydrates, but comes at a greater cost. The average cost of a bottle of MCT oil is $30.00 per/250 ml. A patient using the MCT-KD will usually use 2-3 bottles a day. Most insurance plans to not cover the cost of MCT oil.

The Modified Atkins Diet

The MAD is a recent addition to the ketogenic family. In 2003 Dr. Eric Kossoff, of Johns Hopkins, suggested modifying the induction phase of the Atkins treat epilepsy (Henry et al., 2013). The MAD is based off of the induction phase of the Atkins Diet. The MAD differs from the Atkins Diet in two ways: 1) The MAD encourages more fat consumption; 2) In the tradition Atkins Diet net carbs are calculated as the carbohydrates minus the fiber and sugar alcohols, however, the MAD calculates net carbs as carbohydrates minus fiber. The MAD was created as an attempt to provide a palatable and less restrictive dietary treatment for adolescents and children with behavioral difficulties (Dorward & Kossoff, 2008). The MAD is the most liberal of
the KDs. Unlike the CKD and MCT-KD the MAD has no calorie or protein limits. The MAD does not require fasting, hospitalization or dietitian support. The fat composition of the MAD is similar to a 0.9:1 KD ratio (Dorward & Kossoff, 2008). This ratio is less than the CKD ratio of 4:1 but more than the MCT-KD. The diet composition of the MAD is 64% fat, 30% protein and 6% carbohydrate (Henry et al., 2013).

Unlike the other KDs MAD has not shown efficiency across all epilepsies. The MAD yields the best results in people with idiopathic generalized epilepsies (Henry et al., 2013). Forty-five percent of patients with IGE, including adults, experienced 50-90% seizure reduction with the MAD (Dorward & Kossoff, 2008). Twenty-eight percent experienced >90% seizure reduction when placed on the MAD (Dorward & Kossoff, 2008). In addition to patients with IGE the MAD is also considered for children who must start same day or cannot remain in the hospital during the fasting period (Henry et al., 2013). Patients using the MAD have less side effects than those utilizing MCT-KD or CKD. However, MAD causes a greater increase in total cholesterol. The only other significant abnormality associated with the MAD was blood urea nitrogen (Dorward & Kossoff, 2008).

Clinical use

Despite their proven efficacy, KDs not prescribed often. In 2005, a survey of 88 pediatric neurologists found that only 36% of them regularly prescribed a KD after three or more drugs have failed; 24% occasionally prescribed the diet after surgery had failed; 24% had only prescribed the diet in rare cases; and 16% had never prescribed the diet (Zupec-Kania & Spellman, 2008). The limited use of KDs can be attributed to the lack of adequately trained neurologists and dietitian who can administer the diet. Kossoff et al. (2012:447) point out the reluctance of pediatric neurologists to prescribe KD when they mention:
Despite the high perception of support, the average child was typically treated with 5 anticonvulsants over approximately 3 years prior to beginning the KD. This is in contrast to the 2009 International KD Consensus Statement recommendations that the KD should be considered after 2–3 anticonvulsants have been attempted.

Statements made by the subjects I interviewed support the 2005 findings. The individuals I interviewed expressed a lack of medical support. Interviewee1, the mother of a 9-year-old boy with IGE, stated that she was told the ketogenic diet was “not safe and no longer used” (Personal communication 2014). She saw three different pediatric neurologists, before finding one that would prescribe a KD. Interviewee1, found a pediatric neurologist with the help of the Charlie Foundation. She expressed frustration with the medical establishment and their reluctance to use KDs. Interviewee1, faced additional challenges with her insurance company. The pediatric neurologist prescribed the MCT-KD for her son; however Liquigen was not covered by her health insurance. Interviewee 1 decided to place her son on the MCT-KD and pay for the Liquigen formula out-of-pocket.

The limited use of KDs was evident during an interview with a nurse practitioner at Emory University Hospital’s Neurology clinic. EmoryNP, informed me that in her 20 years as a nurse practitioner at the clinic she has never seen a doctor prescribe a KD. She was familiar with the treatment because two the patients she sees previously used KDs to manage their seizure disorders. EmoryNP was only familiar with the MCT-KD, but she had very little knowledge about the process.

It is unfortunate that such a promising treatment is denied to so many because of lack of trained medical professionals.

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19 Commercially marketed MCT oil emulsion.
Community and Support Groups

Support groups seem to pick up where the medical professionals fall short. Support groups and foundations provide practitioners with resources such as, neurologist listings, recipes and solutions to everyday challenges. There are over 30 “yahoo groups” dedicated to KDs. These groups discuss the everyday challenges that accompany living with a restricted diet, products and new research about KDs. Out of the eight yahoo groups I reviewed I noticed that tree of them had posts from neurologist that were supportive of the KD treatments. These neurologist suggested articles, products, clinics and answered basic medical questions. There are also many blog sites that chronicle the progress of patients using KD treatments. Blog sites often reflect the experience on the writer, and have limited community participation. In addition to support groups foundations play a large role in informing people about KD.

The Charlie foundation has created the videos for parents about the diet, as well as one directed at physicians and another, an instructional video, for dieticians. The foundation has distributed over 50,000 of these videotapes gratis. The Charlie Foundation also funded the initial publication of *The Epilepsy Diet Treatment: An Introduction to the Ketogenic Diet* (Wheless, 2004). Other influential foundations are Mathew’s Friends and the Carson Harris Foundation. Mathew’s Friends promotes dietary awareness, provides support group meetings, clinic listings, recipes and updates on new research in the UK. The Carson Harris Foundation is an American foundation that supports KD research.

With limited support from the medical community individuals have become experts ketogenic diets.
Personal experience with MAD

I have refractory idiopathic generalized epilepsy\textsuperscript{20} (IEG), manifesting as Tonic, Myoclonic, and Myoclonic-absence seizures. I currently take 400 mg of Lamotrigine daily. I have been using AEDs to control my seizures for over twenty years. As part of this research I have decided to participate in using a KD to help control my seizures. After evaluating all of the KDs I decided to participate in the MAD. My decision to use the MAD was effected by three factors: 1) The MAD does not require fasting or hospitalization, 2) I can use the MAD without the assistance of a medical nutritionist, and 3) The MAD allows me to eat a wider variety of food choices.

On February 3\textsuperscript{rd}, 2014 I went to the doctors to have a full blood panel preformed\textsuperscript{21}. I used the results of the blood panel from February as a baseline for my vital statistics. During the course of this project I kept a diary of my weight, seizure activity and ketone levels. In order to safely transition from AEDs to the MAD, on February 27\textsuperscript{th}, I attempted to enlist the help of my neurologist. I informed my neurologist that I was still having myoclonic spasms during the week, and would like to add the MAD to my treatment. My Neurologist had never heard of the diet and suggested that we begin adding Keppra\textsuperscript{22} to my current AED regiment. Despite my insistence she was not in interested in hearing or acknowledging my request for a KD treatment. I began the MAD on March 1\textsuperscript{st}, 2014, without the help of a physician. I continued to take my AED throughout the trail. I decided to reduce my AED dosage once I was in a state of ketosis for a week. By the end of the process I had reduced the dosage by 200 mg/daily. It is important to note

\textsuperscript{20}Idiopathic generalized epilepsy is a group of epileptic disorders that are believed to have a strong underlying genetic basis. Patients with an IGE subtype are typically otherwise normal and have no structural brain abnormalities.

\textsuperscript{21}Blood panel results provided in Error! Reference source not found. Table 4

\textsuperscript{22}Levetiracetam
that reducing, stopping or changing AEDs should not be done without the guidance of a neurologist, what I did is ill-advised and unsafe.

The first three days of the diet was extremely challenging, I experienced headaches, dizziness and weakness in my limbs. After the initial three days I no longer experienced the aforementioned symptoms. My diet consisted mainly of meat and green-leafy vegetables. I found it difficult to achieve my daily fat requirements. The lack of fat in my diet hindered my body’s ability to shift into ketosis. After weeks of not being able to achieve ketosis, I began to cook all of my food in coconut oil. The coconut oil, being a medium-chain-triglyceride, help me achieve ketosis in five days. Once in ketosis I was able to maintain between 15 and 40 mg/dL. Although I was able to maintain a state of ketosis, I was unable to maintain a steady level. In an attempt to maintain stable ketone levels I added unsaturated fats such as, Flax seed oil, avocado oil, and almond oil to my diet. I began to take one table spoon of each oil a day. The additional oil helped me achieve a steady ketone level of 40 mg/dL. On March 27th, I reduced my AED dosage by 100mgs, within a week I reduced the dosages again by another 100mgs.

Obtaining ketosis was more difficult than expected. It took approximately three weeks to enter a state of ketosis, and an additional week to stabilize ketone levels. I began to see a reduction in my myoclonic spasms within the third week. I was no longer experiencing myoclonic spasms during throughout the day; however I was still experiencing them in the morning. By the end of week four all of my myoclonic spasms had stopped. A lot of the studies involving KDs shows, in addition to reduced seizures, patients experience a sense of mental clarity. Unfortunately, I did not experience a sense of mental clarity. I experienced very minimal side effects, which were ameliorated by small changes in food choices. During the first week I experience constipation. I addressed this by adding raw flax seeds to every meal. In addition to
brief constipation the amount of fat in my diet made me nauseous. The nausea persisted for six weeks. There were additional, unexpected, side effects: my hair became soft and shiny, my nails began to grow faster and my skin became softer. KDs usually increase cholesterols and lipids; however, I was not able to return to the doctors for a follow up blood panel, as my health insurance lapsed. In addition to the changes in my seizure activity, I lost 20 lbs in four weeks.

Conclusion

Dietary treatments for epilepsy have provided successful broad spectrum control of seizures since ancient Greece. With the advancement of ketogenic diets people with epilepsy have a more treatment options. Ketogenic diets provide stable seizure control at an affordable price with minimal and manageable side effects. These diets are not home remedies but proven medical treatments for epilepsy. I believe that KDs should be the first line of treatment, not the last. Although there has been a reemergence of the use and research of KDs, many physicians and neurologists are not aware of them. Ketogenic diets are challenging and take dedication, but the alternatives of long term AEDs are detrimental to a person’s health. The medical evidence supports the use of KDs, yet many neurologists are reluctant to prescribe them. I agree with Kossoff et al (2012:447) when he says “…further education of child neurologists regarding the benefits of the KD may be warranted.” In fact I believe further education for all neurologists on the benefits of the KD is warranted.

I personally will continue the MAD and look for a neurologist who is familiar with the KD treatments.
Appendix

**Tables & Figures**

Tables 1 and 2 are extracted from, Drug treatments of epilepsy: Options and limitations by Deiter Schmidt, published in the *Journal of Epilepsy & Behavior*.

Simplified synopsis of drug interaction properties of common AEDs.

<table>
<thead>
<tr>
<th>Antiepileptic drug</th>
<th>Enzyme inducer (CYP)</th>
<th>Enzyme inhibitor (CYP, UGT)</th>
<th>Effect of drug on disposition of other AEDs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clobazam (CLB)</td>
<td>No</td>
<td>No</td>
<td>No relevant change</td>
</tr>
<tr>
<td>Felbamate (FBM)</td>
<td>No</td>
<td>No</td>
<td>No relevant change</td>
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<tr>
<td>Gabapentin (GBP)</td>
<td>No</td>
<td>No</td>
<td>No relevant change</td>
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<tr>
<td>Levetiracetam (LEV)</td>
<td>No</td>
<td>No</td>
<td>No relevant change</td>
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<tr>
<td>Lacosamide (LCM)</td>
<td>No</td>
<td>No</td>
<td>No relevant change</td>
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<tr>
<td>Zonisamide (ZNS)</td>
<td>No</td>
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<td>No relevant change</td>
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<tr>
<td>Topiramate (TPM)</td>
<td>No (&lt;200 mg/day)</td>
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<td>No relevant change</td>
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<tr>
<td>Carbamazepine (CRZ)</td>
<td>Yes</td>
<td>No</td>
<td>LTG, TGB, VPA (▼▼)</td>
</tr>
<tr>
<td>Ethosuximide (ETS)</td>
<td>No</td>
<td>No</td>
<td>PHT, VPA (▲, CRZ/▼)</td>
</tr>
<tr>
<td>Lamotrigine (LTG)</td>
<td>Yes</td>
<td>No</td>
<td>No relevant change</td>
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<tr>
<td>Oxcarbazepine (OXC)</td>
<td>Yes</td>
<td>No</td>
<td>OXC doses &gt;100 mg (▼▼); CRZ, LTG, PHT, TGB, VPA (▼▼)</td>
</tr>
<tr>
<td>Phenytoin (PHT)</td>
<td>Yes</td>
<td>No</td>
<td>CRZ, LTG, OXC, PHT, TGB, VPA (▼▼)</td>
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<td>Pregabalin (PGN)</td>
<td>No</td>
<td>No</td>
<td>No relevant change</td>
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<tr>
<td>Primidone (PRM)</td>
<td>Yes</td>
<td>No</td>
<td>see PHB</td>
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<tr>
<td>Valproate (VPA)</td>
<td>No</td>
<td>Yes</td>
<td>CRZ-E, LTG, PB, free PHT (▲)</td>
</tr>
<tr>
<td>Vigabatrin (VGB)</td>
<td>No</td>
<td>No</td>
<td>PHT (▼), other AEDs (▲)</td>
</tr>
</tbody>
</table>

Note. CYP, Cytochrome p450 System; UGT, Uridine Diphosphate-Glucuronate Transferase System; CRZ-E, Carbamazepine epoxide; ▲▲, no relevant change; ▲, increase in plasma concentration; ▼, decrease in plasma concentration; ▼▼, major decrease in plasma concentration.

Table 1

Overview of adverse effects of AEDs.

<table>
<thead>
<tr>
<th>Early-onset adverse events</th>
<th>CBZ</th>
<th>CLB</th>
<th>ETS</th>
<th>FBM</th>
<th>GBP</th>
<th>LCM</th>
<th>LEV</th>
<th>LTG</th>
<th>OXC</th>
<th>PGN</th>
<th>PHB</th>
<th>PHT</th>
<th>TGB</th>
<th>TPM</th>
<th>VPA</th>
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<td>Somnolence</td>
<td>+++</td>
<td>+</td>
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<tr>
<td>Dizziness</td>
<td>++</td>
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<td>Seizure aggravation</td>
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<tr>
<td>Gastrointestinal</td>
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<td>++</td>
<td>(▲)</td>
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</tbody>
</table>

Note. (*) Minimally increased risk in clinical use; +, risk higher than for AEDs without +; ++, highest risk among AEDs. In general, although exposure to some modern AEDs is still limited, treatment with a number of modern AEDs appears to be advantageous compared with treatment with some of the classic AEDs (see summary of risks). It should, however, be noted that the incidence of many early adverse events shown here can be lowered by using slow titration and avoiding above-average dosages and combination therapy, if possible. For definitions of AEDs see Table 1.

Table 1
Tables 3 is extracted from, Dietary treatments for epilepsy: Management guidelines for the general practitioner by Paul R. Lee and Eric H. Kossoff, published in the Journal of Epilepsy & Behavior.

Initial vitals: Height: 5’ 8”, Weight: 293lbs, BMI: 44.9, BP 133/ 93

**Results of blood panel taken on 2/3/14**

<table>
<thead>
<tr>
<th>Panel</th>
<th>Results</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin D, 25-Hydroxy</td>
<td>19.6 ng/mL</td>
<td>29.9 ng/mL - 74.0 ng/mL</td>
</tr>
<tr>
<td>Vitamin B12 level</td>
<td>1078 pg/mL</td>
<td>211 pg/mL - 911 pg/mL</td>
</tr>
<tr>
<td>Folate level</td>
<td>&gt;20.0 ng/mL</td>
<td>&gt;= 5.5 ng/mL</td>
</tr>
<tr>
<td>Vitamin B1 level whole blood</td>
<td>60 nmo/L</td>
<td>70 – 80 nmo/mL</td>
</tr>
<tr>
<td>Vitamin B6 level</td>
<td>58.5 nmo/L</td>
<td>20.0 – 125.0 nmo/L</td>
</tr>
<tr>
<td>Zinc level</td>
<td>86 mcg/dL</td>
<td>20 – 120 mcg/dL</td>
</tr>
<tr>
<td>Non-HDL Cholesterol</td>
<td>117 mg/dL</td>
<td>&lt;=129 mg/dL</td>
</tr>
<tr>
<td>Cholesterol level total</td>
<td>173 mg/dL</td>
<td>&lt;=199 mg/dL</td>
</tr>
<tr>
<td>HDL Cholesterol</td>
<td>56 mg/dL</td>
<td>&gt;=40 mg/dL</td>
</tr>
<tr>
<td>LDL Cholesterol</td>
<td>95 mg/dL</td>
<td>&lt;=99 mg/dL</td>
</tr>
<tr>
<td>VLDL Cholesterol</td>
<td>22 mg/dL</td>
<td>&lt;=29 mg/dL</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>110 mg/dL</td>
<td>&lt;=149 mg/dL</td>
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<tr>
<td>Insulin</td>
<td>15.7 mIU/L</td>
<td>3.0 – 25 mIU/L</td>
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<tr>
<td>Glucose</td>
<td>92 mg/dL</td>
<td>65 – 110 mg/dL</td>
</tr>
<tr>
<td>Hemoglobin A1C</td>
<td>5.6%</td>
<td>4.3 – 6.1 %</td>
</tr>
<tr>
<td>TSH Reflex order</td>
<td>1.11 mclU/L</td>
<td>0.55 – 4.78 mclU/L</td>
</tr>
</tbody>
</table>
Ketogenic Diet Resources

Mathew’s Friends: http://site.matthewsfriends.org/
Address: Ncype, St Piers Lane, Lingfield, Surrey RH7 6PW, United Kingdom
Phone:+44 1342 836571

The Charlie Foundation: http://www.charliefoundation.org/
515 Ocean Ave., #602N
Santa Monica, CA 90402
Phone: (310) 393-2347

The Carson Harris Foundation: http://www.carsonharrisfoundation.org/
PO Box 310
Glen Arm, Maryland 21057

Modified Atkins for Seizures: http://www.atkinsforseizures.com/
Works Cited


