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Abstract

The modified Atkins diet has been used since 2003 for the treatment of children and adults with refractory epilepsy. This “alternative” ketogenic diet is started in clinic, without fasting, hospitalization, and restriction of protein, calories, or fluid intake. Now after 10 years of continued use, approximately 400 patients have been reported in over 30 studies of the modified Atkins diet as treatment for intractable seizures, with results demonstrating similar efficacy to the ketogenic diet and improved tolerability. The modified Atkins diet is being increasingly used in the adult population. Clinical trials have provided insight into the mechanisms of action of dietary therapies overall. This review will discuss the past decade of experience with the modified Atkins diet as well as predictions for its role in the treatment of epilepsy a decade from now.

Keywords:
- Atkins
- Ketosis
- Ketogenic Diet
- Epilepsy

Key questions
1. How is the modified Atkins diet different from the traditional classic ketogenic diet (or low glycemic index treatment)?
2. Is the modified Atkins diet effective?
3. Which patients should receive the modified Atkins diet instead of the ketogenic diet?
4. What clues does the modified Atkins diet give us into the mechanisms of action of all dietary therapies?
5. What is the future of the modified Atkins diet 10 years from now?

1. Introduction

Patients with refractory epilepsy today have more treatment options than ever before. New anticonvulsant drugs with novel mechanisms of action such as exogabine, lacosamide, perampanel, and rufinamide have been introduced in recent years. Epilepsy surgery is becoming more widely available and safer, with new techniques and earlier resections leading to improved quality of life even for patients with the most medically resistant seizures. Vagus nerve stimulation is beneficial for many patients, and other forms of neurostimulation, such as deep brain stimulation (targeting the anterior nucleus of the thalamus) and responsive neurostimulation (targeting the cortex), are in various stages of development and licensure.

Included in the variety of therapeutic options are dietary therapies, now often being referred to by basic scientists as “metabolism-based therapies” [1]. The classic ketogenic diet (KD) is one of the oldest therapies for epilepsy, having been introduced to the world formally in June 1921 [2], and now popular worldwide for the treatment of children with refractory epilepsy. The KD is an extremely high fat (approximately 90% of calories), low carbohydrate, moderate protein diet that is calorie- and fluid-limited and started typically in the hospital by a dietitian familiar with its use [1]. In the 92 years since its introduction, there have been changes to the initiation period of the KD to make it more tolerable, but the composition itself has not been significantly altered since its original conception.

As KD usage has increased, neurologists, dietitians, and parents alike have sought alternatives with better tolerability and fewer adverse effects and have considered utilizing these treatments for a broad range of neurologic conditions other than epilepsy. In 2003 at Johns Hopkins and in 2005 at Massachusetts General Hospital, two “alternative” diets were created to make dietary therapies easier to implement, and studies demonstrated that the restrictiveness of the KD was not necessary in all circumstances [3,4]. These diets, the modified Atkins diet (MAD) and the low glycemic index treatment (LGIT), have changed the mindset of many neurologists and parents regarding the feasibility of dietary therapies and appear to have opened the door for their wider use. The LGIT has been shown to be effective, but for the purposes of this review, the MAD only will be discussed in detail.

2. History

In 2003, the first case series of 6 patients (three of whom were adults) who were treated with the Atkins diet for seizures was published [3]. The first patient was a 10-year-old boy (Patient 2) whose
parent started the Atkins diet unsupervised, loosely following recipes from a KD regimen he had received 2.5 years prior, and this led to seizure freedom. Seizures remained completely controlled on the Atkins diet. The child, however, that truly raised our awareness and led to further studies was a 7-year-old girl (Patient 1) with intractable epilepsy due to a left parietal cortical dysplasia. In May 2003, her mother placed her on the Atkins diet to restrict carbohydrates a week in advance of the classic KD, primarily in order to acclimate her child prior to starting. She achieved large urinary ketosis, and her 80 seizures per day stopped after 3 days and did not return for 3 years of continued dietary treatment. These children and 4 other patients were presented in a poster at the 2003 American Epilepsy Society Annual Meeting in Boston and received media attention. The article was published that same month (December 2003) in Neurology [3].

In 2006, the diet was first formally referred to as the “modified Atkins diet” to distinguish it from the Atkins diet [5]. There are 3 primary differences between these diets: 1) the “induction phase” of the Atkins diet (20 g of carbohydrates per day) is maintained indefinitely on the modified Atkins diet (MAD) instead of being gradually increased, 2) high-fat foods are mandatory on MAD (not just acceptable as with the Atkins diet), and 3) weight loss is not the primary goal of the MAD as with the Atkins diet. Families that were following the Atkins book often reported insufficient ketosis and efficacy, thus the decision to change the name to emphasize these differences [6].

3. Key questions

3.1. How is the modified Atkins diet different from the traditional classic ketogenic diet (or low glycemic index treatment)?

The guiding principle of the MAD is to be easy to grasp and implement (Table 1). As such, there are no formal guidelines for the composition of the foods eaten, with the exception of carbohydrates [7]. Upon reviewing the food records of patients on the MAD, they receive an approximate 1–2:1 ketogenic ratio (grams of fat:carbohydrate and protein combined), but it is only approximate and may change day by day. Therefore, calculating a ketogenic ratio to provide to patients is not recommended [5]. Fig. 1 demonstrates the differences in composition between diets. The MAD is taught to patients and/or families in an outpatient clinic visit lasting 1 h, during which materials are provided, including recipes, carbohydrate lists, food label explanations, and details regarding monitoring ketones and possible side effects (Table 2).

Net carbohydrates, calculated by subtracting fiber from total carbohydrates, are limited to 10 g/day from age 2 to 12 years. There is no specification on which types of carbohydrates are eaten, unlike the LGIT which tracks the glycemic indices of carbohydrates; however, carbohydrates with lower glycemic indices (e.g., those found in berries and whole grains) typically allow for higher quantities to be eaten [4]. Adolescents are limited to 15 g/day and adults to 20 g/day. A multivitamin supplement and calcium supplement with vitamin D are recommended. Based upon the results from a 2011 prospective clinical trial, we recommend a 400-calorie KetoCal® 4:1 shake during the initial month for children, but recognizing the potential out-of-pocket expense, other high-fat supplements (e.g., whipping cream or oils) may be substituted [8]. If the MAD has led to seizure improvement after 1 month of use, we then suggest arranging a follow-up with a dietitian and neurologist along with obtaining laboratory studies at 3 months after initiation.

The MAD differs from the LGIT primarily in regard to the nature and quantity of carbohydrates provided. The LGIT specifies that only carbohydrates (approximately 40–60 g/day) with glycemic indices <50 can be eaten, whereas the MAD does not [4]. However, most families do eat carbohydrates with lower glycemic indices on the MAD in order to allow for larger quantities (e.g., several strawberries versus a very tiny piece of chocolate). The exact composition of the LGIT, however, is largely similar to the MAD with 60% fat, 30% protein, and 10% carbohydrates and approximates a 1:1 ketogenic ratio [4]. One major reported difference is the lack of urinary ketosis with the LGIT [4]. We use the LGIT at our center for patients who cannot tolerate the MAD (or KD) because of the high fat content, or who have persistent difficulties with hyperlipidemia or are symptomatic from overketosis (e.g., vomiting or fatigue).

3.2. Is the modified Atkins diet effective?

The first formal, prospective, open-label study was designed for children with refractory epilepsy and was funded graciously by the Dr. Robert C. Atkins Foundation [5]. Twenty children were treated from September 2003 to May 2005, with 16 (80%) completing the 6-month trial [5]. Using an intent-to-treat analysis, 13 (65%) had >50% seizure reduction at 6 months, including 7 (35%) with >90% improvement. These results were strikingly similar to most studies of the classic KD, justifying its continued use and study. Interestingly, although the presence of large urinary ketosis at 1 month correlated with success, this did not persist at 3 and 6 months. This suggests that strict adherence to the diet during the first month may improve efficacy, but this would need to be confirmed with subsequent studies.

A second prospective study randomized children to 10 or 20 g of net carbohydrates per day for 3 months, followed by a crossover to the opposite treatment arm [9]. Similar to our 2006 study, it was found that a stricter carbohydrate limit (10 g/day) was more effective, and switching later (in either direction) did not influence seizure control [9]. The most recent study to test this “strict first month” hypothesis used a 400-calorie KetoCal® 4:1 shake as a supplement to a 10-g/day MAD during the first month in an open-label manner [8]. When compared to historical MAD outcomes to date, this approach was more successful (80% with >50% seizure reduction including 37% with >90% after 1 month). Again, loosening the restrictiveness (in this case, by eliminating the shakes at 1 month) did not lead to increased seizures. Contrary to our prediction, KetoCal® did not appear to boost urinary ketosis; rather, it increased the daily fat intake and, thus, the ketogenic ratio to 1.8:1 [8].

Table 1

<table>
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<tr>
<td><strong>Initiation</strong></td>
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<tr>
<td>- Baseline laboratory studies (complete blood count, liver and kidney function tests, electrolytes, fasting lipid profile) and 2-day food record obtained before beginning the diet</td>
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<tr>
<td>- High-fat foods added liberally</td>
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<tr>
<td>- Carbohydrates: 10 g/day for children, 15 g/day for adolescents, 20 g/day for adults (net carbohydrates counted = total carbohydrates minus fiber; no subtraction of sugar alcohols)</td>
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<tr>
<td>- Multivitamin supplement and calcium supplement with vitamin D daily</td>
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<tr>
<td>- If possible, KetoCal® 4:1 = 400-calorie shake daily (2/3 cup powder with 8 oz of water or one “tetrapak” liquid container) for the first month</td>
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<td>- Clear carbohydrate-free fluids encouraged</td>
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<tr>
<td><strong>Monitoring and maintenance (1st month)</strong></td>
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<tr>
<td>- Weight checked weekly</td>
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<tr>
<td>- Ketones checked twice a week for at least the first month</td>
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<tr>
<td>- Carbohydrate counting guides reviewed (from dietitians, books, Internet, apps)</td>
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<tr>
<td>- Avoid anticonvulsant changes</td>
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<tr>
<td>- Avoid low carbohydrate store-bought products</td>
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<tr>
<td><strong>Maintenance (after 1st month)</strong></td>
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<tr>
<td>- May increase net carbohydrates (by 5–10 g/day), change anticonvulsants, use low-carb products (as deemed necessary)</td>
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<tr>
<td>- Follow-up laboratory studies (including lipid profile) after 3 months</td>
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<tr>
<td>- Urine ketones checked weekly</td>
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<tr>
<td>- Food record at 3 months</td>
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<tr>
<td><strong>Discontinuation</strong></td>
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<tr>
<td>- Increase carbohydrate limit by 10 g/day/week until 60 g/day, then substitute regular meals, one per week. A quicker titration off may be acceptable in some circumstances.</td>
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There have been many other studies examining the MAD, which are listed in Table 3 [10–37]. Several of these publications in particular have demonstrated some findings of particular note. Two studies in 2007 and 2012 from Korea confirmed the benefit of the MAD, with the first study suggesting that stable blood ketones over time (beta-hydroxybutyrate of >3 mmol/L) may be important [10,11]. Dr. Sharma and her team in India have studied the MAD twice, initially for children with refractory infantile spasms, using the MAD in children as young as 6 months of age [12]. Most recently, in the only randomized controlled study of the MAD for the treatment of childhood epilepsy, published this year in Epilepsia, it was found that the MAD arm (n = 50) was more likely to have >50% seizure reduction than the control arm of standard medical management (n = 52) (52% vs. 11.5%, p < 0.001) [13]. Similarly, the likelihood of >90% seizure reduction was higher (30% vs. 7.7%, p = 0.005). This trial followed the general study design of a previous trial by Neal et al. of the classic KD, published in 2008 [38].

Other recent pediatric studies include a pilot series of the MAD for Sturge–Weber syndrome and a case series treating childhood absence epilepsy [31,32]. An attempt at a direct comparison was performed in France in 2009 and found retrospectively that the KD led to better seizure reduction than the MAD but only after 3 months (not at 6 months) [21]. In a series comprised of patients from 5 countries, retrospective data suggested that switching from the MAD to the KD led to a 30% chance of additional improvement, in many ways similar to raising the dose of an anticonvulsant [39]. Interestingly, the only children in this series who became seizure-free after changing to the KD had myoclonic-astatic epilepsy (Doose syndrome), indicating that patients with that syndrome may respond preferentially to either higher fat, greater ketosis, or, possibly, calorie limitation.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Differences between the ketogenic and the modified Atkins diet.</th>
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<tr>
<td><strong>Calories (% recommended daily allowance)</strong></td>
<td>Ketogenic diet</td>
</tr>
<tr>
<td>Fluids (L)</td>
<td>Restricted (75%) or matched</td>
</tr>
<tr>
<td>Fat</td>
<td>85%–90%</td>
</tr>
<tr>
<td>Protein</td>
<td>15%</td>
</tr>
<tr>
<td>Carbohydrates</td>
<td>5%</td>
</tr>
<tr>
<td>Fasting period</td>
<td>Occasionally done</td>
</tr>
<tr>
<td>Admission to hospital</td>
<td>Typically done</td>
</tr>
<tr>
<td>Meal plans computer-created</td>
<td>Yes</td>
</tr>
<tr>
<td>Foods weighed and measured</td>
<td>Yes</td>
</tr>
<tr>
<td>Sharing of food at family meals</td>
<td>No</td>
</tr>
<tr>
<td>Ability to eat foods made in restaurants</td>
<td>No</td>
</tr>
<tr>
<td>“Low carbohydrate” store-bought products</td>
<td>Not used</td>
</tr>
<tr>
<td>Intensive education provided</td>
<td>Yes</td>
</tr>
<tr>
<td>Dietitian involvement</td>
<td>Yes</td>
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<tr>
<td>Multiple studies over many years showing benefits</td>
<td>Yes</td>
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</table>

Fig. 1. Differences between the macronutrient compositions among the diets.

Lastly, as the MAD has been used for 10 years, we are starting to obtain long-term outcomes from patients who have received it for longer than the 6 months typically reported in clinical trials to date. At a mean of 20 months, 30 of 54 (55%) children with long-term MAD duration maintained >50% seizure reduction [40]. Side effects continued to be mild and predominantly consisted of gastrointestinal discomfort, fatigue, and constipation [40].

3.3. Which patients should receive the modified Atkins diet instead of the ketogenic diet?

In our opinion, the classic KD is more appropriate than the MAD for infants under age 2 years and for those receiving formula-only nutrition (e.g., gastrostomy tube-fed). However, the decision becomes difficult when determining which diet is best to begin in most children ages 2–12 years. As several of the MAD studies suggest that a strict first month improves efficacy, a reasonable strategy would be to start with the KD and then switch to the MAD later [8,9]. Some families truly require the supervision and detailed meal plans provided with the KD by dietitians, and the MAD is deemed too variable. In this age range, children whom we consider for the MAD before the KD include the following: 1) children in busy, large families in which sharing food is highly important; 2) children with limited tolerance for extremely high-fat foods; 3) children with a need for more protein than allowed on the KD; 4) children in families in which hospital admission or a fasting period would be problematic because of cost, location, or other factors; 5) children with idiopathic generalized epilepsies (e.g., absence epilepsy, juvenile myoclonic epilepsy); and 6) children who must be started on treatment urgently (e.g., same day), which can be done in the outpatient clinic.

Even as far back as 2003 in the original case series on the MAD, 4 of 6 patients were over the age of 12 years, suggesting that the “ideal” population for this alternative diet may be adolescents and adults [3]. Historically, these patients were not typically offered dietary therapy, although this is changing with the MAD. The first formal prospective study was published in 2008 using the same study design and criteria as the first pediatric study except that the patients enrolled were 15 years of age or older [15]. Thirty adults, ages 18–53 years, were started on a 15-g/day net carbohydrate MAD. Most who responded did so within 2 weeks, including 47% by 1 and 3 months and 33% after 6 months [15]. Surprisingly, weight loss correlated with efficacy at 3 months, although we are not aware of any other study reporting this observation. The MAD was very well-tolerated in adults, with only a slight increase in mean total cholesterol (187 to 201 mg/dL) and an associated increase in HDL cholesterol, although the latter was not statistically significant.

Other studies on adults have followed, generally with equivalent outcomes [18,26,33–35]. We now recommend a net carbohydrate limit of 20 g/day, which is well-tolerated and leads to similar levels
of ketosis. Similar to absence epilepsy in children, the MAD may have a unique role for another idiopathic generalized epilepsy with seizures that can persist into adulthood, JME (juvenile myoclonic epilepsy) [34]. As a direct result of the success of the MAD for adults, our hospital opened an Adult Epilepsy Diet Center in August 2010, with now over 100 patients treated to date. Several clinical trials are underway including dietary treatment of status epilepticus and examining if KetoCal® shakes are similarly effective and well-tolerated in adults as in children. Other adult epilepsy diet centers are actively attempting, such as in idiopathic generalized epilepsies. If determined to be effective, animal models of these epilepsies could then be tested metabolically fascinating aspects of research into ketogenic therapies. For decades, and still today, the KD is hypothesized to work by breaking down fats into ketone bodies, which can be used for energy [1]. This was believed to be the primary, if not sole, mechanism of action of the KD. The MAD does not always achieve long-term high levels of urinary ketosis, and many children and adults can lose ketosis over time yet have preserved seizure control [5]. It is possible that an initial period of high ketosis is important or even that low levels of stable ketosis may improve seizures, but the MAD has raised questions about the classic belief that persistent, high ketosis is critical. Further study on this issue is warranted.

The MAD trials also suggest that the first month is the most important to achieve success. Data from KD studies have shown this as well, with improvement tending to occur within 2–4 weeks of onset and 4:1 ketogenic ratios being more effective than lower ratios [42,43]. What occurs during the first month that metabolically “shocks” the system and improves seizures? This change could be attributed to increased utilization of fat, the effect of elevated ketones, or other metabolic changes that have yet to be identified. If better elucidated, then, alternative methods to metabolically alter the brain could be devised, perhaps needing to be used only for short periods of time. Considering the potential adverse effects of long-term dietary therapy use, effective short-term dietary therapy may have profound implications on health and nutrition [44].

In addition, as the MAD becomes more widely used, we suspect that it will continue to be used for epilepsy indications not traditionally attempted, such as in idiopathic generalized epilepsies. If determined to be effective, animal models of these epilepsies could then be tested with dietary therapy, and the potential molecular targets could be identified.

### 3.4. What clues does the modified Atkins diet give us into the mechanisms of action of all dietary therapies?

One of the most scientifically fascinating aspects of research into alternative diets such as the MAD and LGIT relates to the mechanisms of action of ketogenic therapies. For decades, and still today, the KD is hypothesized to work by breaking down fats into ketone bodies, which can be used for energy [1]. This was believed to be the primary, if not sole, mechanism of action of the KD. The MAD does not always achieve long-term high levels of urinary ketosis, and many children and adults can lose ketosis over time yet have preserved seizure control [5]. It is possible that an initial period of high ketosis is important or even that low levels of stable ketosis may improve seizures, but the MAD has raised questions about the classic belief that persistent, high ketosis is critical. Further study on this issue is warranted.

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3.5. What is the future of the modified Atkins diet 10 years from now?

Ten years since its introduction, some trends have occurred that might allow for prediction as to the future of the MAD in 2023. There is little doubt that the MAD will continue to be utilized in adults with refractory epilepsy, and this age group may even be the primary age group for which the MAD is used. Adolescents may also be universally started on this instead of the classic KD.

Early case reports have already suggested that the MAD may have a unique role in regions of the world, including developing countries, where the resources of a full ketogenic diet center (including dietitians) may not be available. A 2-year-old boy in Honduras with Lennox–Gastaut syndrome was successfully treated with the MAD after materials were translated into Spanish, and his mother was educated by a local child neurologist [17]. There was also a cost savings overall for this family; the higher costs of meats and dairy in this country were offset by the reduction in costs for anticonvulsants. Similar projects are underway in India, China, and other countries. A prospective clinical trial for adults with epilepsy using solely email correspondence with a neurologist (no dietitian) demonstrated feasibility and preliminary efficacy [33]. Telemedicine may improve the efficacy of “remote” dietary treatment and provide access to this treatment for patients unable to travel long distances to a KD center.

As further trials of the MAD continue, we suspect that the incidence of adverse effects, especially the long-term ones, may be less than with the KD because of the inherent higher protein content. This has not been shown to date, but there is evidence in one study [40]. If established, children receiving the KD beyond 1–2 years may be counseled to switch to the MAD in cases where prolonged therapy is indicated. For children with GLUT-1 deficiency who need long-term dietary treatment, the MAD could prove very helpful [29].

In addition, if the MAD continues to be demonstrated as similarly effective, yet less restrictive and safer than the KD, it would be a logical choice for trials of new-onset epilepsies. Families and patients are often averse to perceived risks of anticonvulsants, and although the MAD is not free of adverse effects, it may be seen as a more “natural” approach by many. Trials would perhaps specify a time limit (e.g., 1–2 months) after which the MAD would be discontinued and an anticonvulsant started if seizure control is not achieved. In addition, nonepilepsy uses under investigation (e.g., brain tumors, Alzheimer’s disease, ALS, and migraines) where a strict KD may be beneficial, yet a hospital admission and restrictive meal plans would negatively impact quality of life, could be tested with the MAD instead [45–48].

4. Summary

Now, 10 years after its development, it is clear that the MAD is effective and offers a comparable alternative for those considering dietary therapy but reluctant to implement the classic KD. Research provides clues to the underlying mechanisms of dietary therapies and suggests that an intense metabolic alteration through increased fat metabolism during the first month may be critical. Studies also suggest that this less restrictive diet can preserve efficacy and improve tolerability and health of the children and adults receiving dietary treatment. Finally, the MAD provides exciting new applications for dietary therapies, including those in adults, in other neurologic diseases, and in regions with limited resources.

Disclosures

Dr. Kossoff is on the Scientific Advisory Board for Atkins Nutritional, Inc. Dr. Kossoff, Dr. Cervenka, and Ms. Henry receive grant support for a clinical trial involving KetoCal® by Nutricia, Inc.

Key questions (answered)

1. How is the modified Atkins diet different from the traditional classic ketogenic diet?
   The MAD composition is also carbohydrate-restricted but requires slightly less fat. The primary difference is the lack of restrictions on protein, calories, and fluids, as well as the outpatient, nonfasting initiation period.

2. Is the modified Atkins diet effective?
   Yes, with 10 years of use, there are 423 children and adults reported in 31 studies from multiple centers who have undergone MAD. When added together, 187 (47%) patients overall have had a >50% seizure reduction, which is comparable to the results found for the ketogenic diet.

3. Which patients should receive the modified Atkins diet instead of the ketogenic diet?
   For children under the age of 2 years and those receiving formula-only nutrition, the ketogenic diet is preferable. For adolescents and adults, the MAD is probably the better option. In children in other age groups, diet prescription is at the discretion of their neurologist and dietitian, as well as their parents.

4. What clues does the modified Atkins diet give us into the mechanisms of action of all dietary therapies?
   Data suggest that a strict, highly ketogenic, high-fat initiation period (probably 1 month) may be critical. Long-term high levels of ketosis may not be required. Perhaps a short-term, intense, metabolic alteration is one mechanism by which dietary therapy is effective.

5. What is the future of the modified Atkins diet 10 years from now?
   We suspect that the MAD will be used primarily for adults and adolescents and that children receiving long-term KD will be transitioned to it. There may be additional utility in regions of the world with limited resources, as a first-line therapy for some forms of epilepsy, and even for nonepilepsy indications.

References


