KETONIC DIET IN EPILEPSY: TIME FOR NIGERIA AND AFRICA ACCESS.

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Abstract
Ketogenic diet, a high fat, low carbohydrate diet was devised in 1921 to mimic the effects of starvation originally determined to be beneficial for epileptic seizures. The diet fell out of favour with the development of anticonvulsant agents, but has experienced resurgence in use over the past 20 years, particularly in the treatment of refractory children epilepsy. Since the rebirth of ketogenic diet in the United States of America in the mid-1990s, there has been growing global acceptance of its use. The diet is reported to be efficacious and cost effective. It has the potential to improve quality of life of patient and lessen the burden on family and society. The African continent is lagging behind in the use of ketogenic diet in treatment of epilepsy. The diet hardly has been subject of discourse either in Nigeria academic circles or the Press. Nigeria entry into the diet program is important particularly in the area of provision of choice to patients as well as the kindling of research interest in the diet. Local recipes could be identified and, possibly, formulations unique to the country also developed.

Keywords: Ketogenic diet, Epilepsy, epileptic seizures and Nigeria.

INTRODUCTION
Fasting as a therapy for seizure has been known since biblical times (Mark 9:14-29). In the early 20th century the ketogenic diet was used to mimic the biochemical mechanism of fasting. The first scientific reports on the value of fasting and water diets in epilepsy were authored by Guelpa and Marie, both French physicians in 1911 [1]. However, it was not until the 1921 American Medical Association Convention, at which Rawle Geyelin, an eminent American pediatrician declared successful treatment of epilepsy by fasting, that this method gained the attention of medical professionals [2].

In 1921, Rollin Woodyat reported that three water-soluble compounds-β-hydroxybutyrate, acetacetate and acetone (known collectively as ketone bodies) were produced by the liver in otherwise healthy people when starved or if they consumed a very low-carbohydrate, high fat diet [2-6]. Russel Wilder in 1924 built on this research and coined the term Ketogenic diet, a diet that produced a high level of ketones in the blood (ketonemia) through an excess of fats and lack of carbohydrate [7-10].

A ketogenic diet thus is a high-fat, adequate-protein, low carbohydrate diet that is used primarily to treat difficult-to-control illnesses/malfunction of the human body [11-16]. Normally, carbohydrates contained in food are converted into glucose, which is then transported around the body and is particularly important in fuelling brain function. However, when there is very little carbohydrate in the diet, the liver converts fats into fatty acids and ketone bodies. The ketone bodies pass into the brain and replace glucose as an energy source [17-20].

Elevated level of ketone bodies in the blood, a state known as ketosis, has been utilized for various therapeutic purposes [14,21-25]. These include treatment of epilepsy, cancer, obesity/weight loss, diabetes, glomatherapy, sclerosis and also for enhancing physical performance, energy expenditure and respiratory ratio [26-32].

Epilepsy on the other hand is a medical condition of chronic neurological disorders characterized by recurrent and unprovoked seizures; it results from abnormal, excess or hyper-synchronous neuro-activity in the brain [33-38]. Common causes of epilepsy include hypoxic ischemic encephalopathy, central nervous system (CNS) infections, trauma, congenital CNS abnormality, metabolic disorders, CNS lesions, stress, brain tumors, illicit drug use and alcohol withdrawal. Others are cerebrovascular diseases, head trauma and other degenerative diseases such as dementia [39-44].

About 100 million people worldwide suffer from epilepsy and 80% of them live in the developing countries [45-47]. The incidence is greater in African-Americans and in socially disadvantaged populations and, about 200,000 new cases of epilepsy are diagnosed each year [48]. Epilepsy is the most common serious neurological condition in the world and can affect anyone at any time in life; it has no respect for age, sex, race or social class. Seizures tend to develop in childhood or by late adolescence, but the likelihood of developing epilepsy rises again after the age of 65. One in twenty people will have a single seizure sometime in life. Despite the alarming figure, no instant remedy exists to eliminate convulsions. While drugs work for some, others find them ineffective. What seems to work just as well, if not better, especially in children, is the relatively unknown, high-fat diet [49-54].

People with epilepsy face social stigma due to negative and incorrect knowledge of the disease [55]. In many developing countries, the disease is thought to be contagious. Consequently, people tend to keep safe distance from the patients to avoid contact with their saliva especially during seizure attacks [56,57]. The epilepsy patient in Nigeria is without dignity and suffers not only the social effects of stigmatization, but economic challenges as well. The prevalence of the disease in the country is not known as there are no reliable statistics. Any statistic is retrospective and based on hospital data which very often is unreliable. With a population of over 150 million, there is little doubt Nigeria has her share of the burden of epilepsy. This emotion-laden illness is yet to receive the necessary attention as there appears to be no protocol for treatment in place. The mainstay of treatment is use of anticonvulsant medications.

Drug affordability, side effects and persistent seizures are issues patients and relatives have to grapple with. In the past 15 years, the use of ketogenic diet has expanded enormously - to at least 50 countries worldwide, and a huge amount of clinical evidence of its efficacy is documented. Dramatic outcomes have also been reported with KD in the treatment of epilepsy, even when the diet has been discontinued after a period of administration [45,58-61]. The traditional ‘classical diet’ (4:1) is upheld as the most effective among
the formulations. In some centers outside the United States diet in the ratio of 1.5:1 is reported to be equally effective. However, in Tropical Africa there are little or no country-specific research data on diet formulations and efficacy; a void that should aptly be filled by African scientists. There appears to be low awareness or uptake of the diet program in the African Continent; only South Africa and recently Egypt have keyed into it.

This review examines the progress in the use of ketogenic diet in treatment of epilepsy with the aim of sensitizing Nigeria in particular and other African countries generally, to key into the global diet program. It covers such areas as diet evolution/formulations, mechanism of action, approach to diet administration, efficacy of KD, diet challenges, among others.

**DIET EVOLUTION/FORMULATIONS**

Five types/formulations of ketogenic diets have evolved through the years. These are: the original/traditional/classic ketogenic diet, the medium chain triglyceride (MCT) ketogenic diet, the modified medium chain triglyceride ketogenic diet, the modified Atkins diet and the low glycemic index (LGI) ketogenic diet [9-11,38,62-65]. Calorific contributions [66] of different types/formulations of ketogenic diet are shown in Fig. 1.

**Figure 1** Calorific contributions from food components of the ketogenic diets, by weigh [67] (MAD-modified Atkins diet; LGIT-low glycemic index treatment)

The classic ketogenic diet provided just enough protein for body growth and repair, and sufficient calories to maintain the correct weight for age and height [67-72]. It contained a 4:1 ratio by weight of fat to combined protein and carbohydrate. This was achieved by excluding high-carbohydrate foods such as starchy fruits and vegetables, bread, pasta, grains and sugar, while increasing the consumption of foods high in fat such as cream and butter [4,73-77]. The diet restricts daily calories, and is calculated by the patient’s dietitian with a distribution of 85-90% long-chain fatty acid, 6-8% protein, and 2-4% carbohydrates [10,75,78-80]. The classic diet was introduced in the 1920s and actively employed between 1920s and 30s, with subsequent decline in usage thereafter due to introduction of anticonvulsants drugs.

In the 1960s, it was discovered that medium-chain triglycerides (MCT) produce more ketones per unit of energy than normal dietary fats (which are mostly long-chain triglycerides). Peter Huttenlocher in 1971 [8] devised a ketogenic diet where about 60% of the calories came from the MCT oil, and this allowed more protein and up to three times as much carbohydrate as the classic ketogenic diet. The MCT diet is comprised of 71% medium-chain fatty acid, 10% protein, and 19% carbohydrate [81-87].

Between 1970 and the 80s modified MCT diet which combines the traditional KD and MCT diet came on stream [88-92]. The modified MCT diet distributes the calories as 30% MCT oil, 40-50% conventional or long-chain fatty acids, 10-20% protein and 5-10% carbohydrates [38,86,93,94].

The 1990s witnessed the advent of the modified Atkins diet [5,11,95,96]. The diet comprised 60-70% long-chain fatty acid, 25-30% protein and 5% carbohydrate [38,70,97,98].

The low glycemic index diet birthed in 2012; it provided an alternative diet regimen with more liberal total carbohydrate intake, though restricted to foods that produce relatively little increase in blood glucose (glycemic index < 50). The glycemic index scores individual carbohydrate based on its effect on raising blood glucose within two hours of consumption. The diet’s dietary distribution is 60-70% long-chain fatty acid, 20-30% protein and 10% carbohydrate [38,70,99].

**MECHANISM OF ACTION**

Despite nearly 100 years of use, the mechanism of antiepileptogenic action of KD remains unclear [100,101]. Many studies have focused research on the potential mechanism by which the KD exerts its protective effects on epileptic individuals. Unfortunately, none of the proposed mechanisms was sufficient to explain how the KD works and this is obvious through the controversies in the research of explanation [102]. Early theories regarding mechanism of action of KD focused on the role of ketone bodies, but the newer studies are expanding the understanding of molecular changes induced by the diet and their potential role in controlling seizures [14,103,104]. Such studies make up a large percentage of recent publications regarding the KD. It has long been hypothesized that ketosis was the major mechanism of action of the KD. All three ketone bodies are increased in serum and cerebrospinal fluid of individuals on KD. Acetone suppresses seizures in animal models of tonic-clonic, typical and atypical absence, and complex partial seizures [105,106]. Acetoacetate has been shown to be neuro-protective against the excitatory neurotransmitter glutamate, which is believed to have a role in provoking seizures as well as contributing to neuro-degeneration in intractable epilepsies [107,108]. Finally, β-hydroxybutyrate is structurally similar to γ-aminobutyric acid (GABA), a key inhibitory neurotransmitter and potent anticonvulsant [109]. The level of GABA is affected by the ketogenic state in a way that is advantageous for seizure control. In the shift from glycolysis to fatty acid metabolism, ketosis forces the consumption of oxaloacetate (OAA), a tricarboxylic acid cycle substrate. OAA can be used to synthesize the excitatory neurotransmitters glutamate and aspartate. When OAA is consumed in the ketogenic state, however, glutamate is shunted to GABA synthesis, ultimately making more of this inhibitory neurotransmitter and less of the excitatory neuro-transmitters. These processes are summarized in Figs.2a, 2b and 3.

Other mechanisms for anti-epileptic activity of the ketogenic diet include the Phosphoenolpyruvatecarboxykinase (PEPCK) mechanism proposed by Krieger, Steve [113]; acidosis and dehydration [114]; changes in lipid levels [115]; Altered energy state [114,116]; neurosteroids accumulations [117,118] as well as caloric restriction [119,120].


**Fig(2a)** Glucose metabolism and points at which interventions could affect neuronal excitability and seizure control. [110]

Glucose can be diverted to the pentose phosphate shunt (PPP) via fructose-1,6-diphosphate (FDP). 2-Deoxy-2-glucose (2DG) inhibits glycolysis by blocking the phosphoglucoisomerase step. The ketogenic diet (KD), via ketone bodies, bypasses glycolysis by providing acetyl-CoA (AcCoA) to the TCA (tricarboxylic acid cycle) after glycolysis. Anaplerotic compounds “refill” depleted intermediates from the TCA. Other abbreviations: β-OHB, beta-hydroxybutyrate; AcAc, acetocetate; PDH, pyruvate dehydrogenase intermediate from the TCA.

**Figure 2b** Ketosis mechanism for seizure control (111)

**APPRAOCH TO DIET ADMINISTRATION**

There have been researches in the approach to diet administration [115,121-124]. The traditional KD was administered after a 3-5 days or a week of fasting [2,101,106,125,126]. Recent studies by Sinha and Kossoff [115], Lee [123], Cervenka and Kossoff [127] and Mosek et al., [128] have revealed that the diet may not require a fasting initiation, and may even be initiated with full calories. Also, although the traditional administration of the KD required patient’s admission into hospital, these studies have also revealed that KD can be administered with or without admission into a hospital. However, some researchers still favour admission in a hospital as it helps to monitor the patient’s progress, diet administration and to initiate the diet properly [122-135]. The modified Atkins and the LGI diets do not require fasting and are easy to start at home; thus removing the need for hospitalization. The modified Atkins diet however, is more accessible while the LGI diet is more palatable to teens and adults [38,136-140].

**EFFICACY OF KETOGENIC DIET**

Studies have revealed that the ketogenic diet is effective against all types of epilepsy and seizures, and that no significant differences in terms of performance exist among the various diet types (classical, MCT, Modified MCT, Modified Atkins and LGI KDs) with respect to their efficacy on epilepsy [11,14,89,141,142]. Efficacy of ketogenic diet is also reported against infant spasms with up to 53.5-90.0% success [45,143-147]. The diet is effective against Lennox-Gastaut syndrome [27,92,148-151]; Dravet-Syndrome [152,153] with about 50 - >50% success; Myoclonic-astatic epilepsy with up to 54.5% [154,155] as well as status epileptus [156,157]. It is reported as a therapy of choice, when response to anticonvulsant drugs has failed [38,139,145,146,158].

Although the diet has been used successfully in many different seizure types and epilepsy syndromes, certain types of epilepsy may respond better than others to KD. Myoclonic-astatic epilepsy, myoclonic jerks and generalized seizures tend to respond best [136,144,159,160]. It is further reported that when seizure reduction was not dramatic, there was an improvement in patient’s quality of life and a reduction in the level of anticonvulsant drugs used [89,161,162]. The efficacy of ketogenic diet on epilepsy cuts across all age groups [115,123,128]. The diet has been reported to be effective against childhood epilepsy [137,145,163,164], and most research on ketogenic efficacy is concentrated in this area [161,165,166]. There is also substantial evidence on the efficacy of the diet against adolescent epilepsy [89,167,168], as well as epilepsy in adults [87,169-171]. Children however, are reported to show better response to treatment with ketogenic diets than the other age grades [162,169,172]. Interestingly, KD has been reported to be potentially more effective than the new anticonvulsant drugs and is well tolerated when it is effective [23]. The diet also works synergistically/complementarily to some anticonvulsant drugs [173]. Kossoff & Huffman reported that KD achievement in the treatment of childhood epilepsy could be up to 85-89% decrease in seizure [104]. The efficacy of ketogenic diet in the treatment of epilepsy is summarised in Table 1.

**DIET CHALLENGES**

As with many other medical therapies, the KD has its side effects. A summary of possible anticipated side effects of ketogenic diet is given in Table 2. Their recognition is very important for proper monitoring of children for development of complications [101]. The criticism of KD relates to the potential health effects of reducing intake of foods generally recognized as healthy, including fruits,
vegetables, and whole grains. This is a legitimate concern; however, a very-low-carbohydrate diet can include a wide range of vegetables (e.g., tomatoes, cucumbers, and peppers).

**Table 2. Summary of possible anticipated side effects of the ketogenic diet [95]**

<table>
<thead>
<tr>
<th>Metabolic</th>
<th>Gastro Intestinal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acidosis</td>
<td>Nausea/emesis (initiation)</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Constipation (classic KD)</td>
</tr>
<tr>
<td>Inadequate growth</td>
<td>Diarrhea (MCT-KD)</td>
</tr>
<tr>
<td>Rapid ketosis/acidity</td>
<td>Worsening GERD</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>Acute pancreatitis</td>
</tr>
<tr>
<td>Vitamin, trace element deficiency</td>
<td>Hypoproteinemia</td>
</tr>
</tbody>
</table>

**Hematological**
- Low Na, Mg
- Anemia
- Easy bruising
- Leukopenia

**Cardiac**
- Prolonged QT syndrome
- Cardiomyopathy

**Neurological**
- Basal ganglia changes
- Coma, Obnubilation
- Optic neuropathy (thiamine deficiency)

**Gastro Intestinal**
- Nausea/emesis (initiation)
- Constipation (classic KD)

**Infectious disease**
- Susceptibility to infection

**Orthopedic**
- Fractures

**Renal**
- Symptomatic nephrolithiasis (6%)
- Fanconi renal tubular acidosis
- Dehydration

**Unknown**
- Bone
- Muscle
- Liver

and even small amounts of fruit. It would also be prudent to take a multivitamin/mineral supplement to ensure adequate intake of all essential micronutrients [174].

Whereas traditional KD is most time consuming to prepare, the MCT diet is easier but there are issues of side effects which include abdominal cramps, diarrhea, nausea and vomiting; and the diet being relatively more expensive and less affordable for families [8,162,169,175]. The twin issue of nausea and loss of appetite in ketogenic diet [137,176-178] can lead to malnutrition [45,157,162].

There is also a risk of increase cholesterol levels if the diet is not properly managed [161,177,181,182]. Ketogenic diets can also put strain on the kidney [95,181-183] and may lead to kidney damage and dehydration [163,177,181,185].

The diet can be difficult to maintain in many countries like in Asia owing to cultural tendency toward carbohydrate rich foods [45]. Efforts at making KD more palatable have led to the several modifications. Culture-specific diets are now being designed to meet the needs of a wide variety of cultures and nations (Table 3).

**Table 3. Representative ketogenic diet meals from participating countries (ratios and calories calculated based on the best estimates when not specifically provided) [45]**

<table>
<thead>
<tr>
<th>Country</th>
<th>Meal Description</th>
<th>Calories (kcal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Argentina</td>
<td>36% fat heavy whipping cream</td>
<td>35 g</td>
</tr>
<tr>
<td></td>
<td>36% olive oil</td>
<td>2 g</td>
</tr>
<tr>
<td></td>
<td>2% carrot, celery</td>
<td>50 g</td>
</tr>
<tr>
<td></td>
<td>Mayonnaise, heavy cream</td>
<td>26 g</td>
</tr>
<tr>
<td></td>
<td>Canola oil (224 kcal, 3:1)</td>
<td>12 g</td>
</tr>
<tr>
<td>Belgium</td>
<td>Iceberg lettuce</td>
<td>17 g</td>
</tr>
<tr>
<td></td>
<td>Avocado</td>
<td>20 g</td>
</tr>
<tr>
<td></td>
<td>Pecans</td>
<td>33 g</td>
</tr>
<tr>
<td></td>
<td>Pineapple (564 kcal, 2.5:1 ratio)</td>
<td>12 g</td>
</tr>
<tr>
<td>Singapore</td>
<td>Red noodle</td>
<td>89 g</td>
</tr>
<tr>
<td></td>
<td>Pork, lean, boiled</td>
<td>17 g</td>
</tr>
<tr>
<td></td>
<td>Cabbage, green, boiled</td>
<td>20 g</td>
</tr>
<tr>
<td></td>
<td>Sesame oil</td>
<td>37 g</td>
</tr>
<tr>
<td>South Africa</td>
<td>Braaiwurst</td>
<td>162 g</td>
</tr>
<tr>
<td></td>
<td>Grilled sausage/streakers, roast (sage sausage)</td>
<td>125 g</td>
</tr>
<tr>
<td></td>
<td>(442 kcal, 4:1 ratio)</td>
<td></td>
</tr>
<tr>
<td>Germany</td>
<td>Braaiwurst</td>
<td>162 g</td>
</tr>
<tr>
<td></td>
<td>Butter</td>
<td>2 g</td>
</tr>
<tr>
<td>Sweden</td>
<td>Cooled pensquash</td>
<td>160 g</td>
</tr>
<tr>
<td></td>
<td>Octopus</td>
<td>29 g</td>
</tr>
<tr>
<td></td>
<td>Canola oil</td>
<td>11 g</td>
</tr>
<tr>
<td></td>
<td>Creme fraiche (34%)</td>
<td>3 g</td>
</tr>
<tr>
<td></td>
<td>Tomato sauce</td>
<td>3 g</td>
</tr>
<tr>
<td></td>
<td>Leek</td>
<td>34 g</td>
</tr>
<tr>
<td></td>
<td>Cabbage (emulsion based on peanut oil)</td>
<td></td>
</tr>
<tr>
<td>India</td>
<td>Chicken</td>
<td>1 g</td>
</tr>
<tr>
<td></td>
<td>Onion</td>
<td>14 g</td>
</tr>
<tr>
<td></td>
<td>Tomato</td>
<td>10 g</td>
</tr>
<tr>
<td></td>
<td>Ginger (480 kcal, 3:7:1 ratio)</td>
<td>1 g</td>
</tr>
<tr>
<td></td>
<td>Clove</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bay leaf</td>
<td>50 g</td>
</tr>
<tr>
<td></td>
<td>Garlic powder, red chili powder</td>
<td>100 g</td>
</tr>
<tr>
<td></td>
<td>Cheese</td>
<td>70 g</td>
</tr>
<tr>
<td></td>
<td>(564 kcal, 3:8:1 ratio)</td>
<td></td>
</tr>
<tr>
<td>France</td>
<td>Tarte flambée (flambéed pie)</td>
<td>47 g</td>
</tr>
<tr>
<td></td>
<td>Butter</td>
<td>50 g</td>
</tr>
<tr>
<td></td>
<td>Apricot</td>
<td></td>
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</tbody>
</table>

**DISCUSSION**

Epilepsy knows no racial, cultural or national boundaries. It is a major public health issue and the most common non-infectious neurological disease in developing African countries including Nigeria; it remains a serious medical and social problem [186,187].

In Nigeria the disease is usually controlled with medications. Many patients receive treatment with three or more antiepileptic drugs when their seizures are intractable [188]. However, more than 30% of people with epilepsy are known not to have their seizure controlled even with the best available medications. Often, anticonvulsant medication is life-long and may have major effects on quality of life of patients which, usually is marked by energy deficits, memory loss, fear of experiencing the next seizure and work limitations [159,189-192]. Cognitive impairments due either to the recurrent discharges or the multiple antiepileptic drugs contribute to disruption of life quality [193-197]. Patient drug compliance in this situation is bound to be poor. Still a sizeable number of patients either cannot afford or do not even have access to all available anticonvulsant drugs [45].
Faced with a health challenge of the magnitude, many have resorted to the use of traditional medicines and the combined use of traditional and spiritual methods [198].

Both the reports of the International League Against Epilepsy (ILAE) and World Health Organisation (WHO) emphasize the fact that a large percentage of the population with epilepsy in developing countries are not adequately treated [45,199]. This explains the scenario in countries like Nigeria where the person with epilepsy is likely to drop out of school, suffer job loss, finds it impossible to marry, loses the spouse and be tormented to the extent of becoming a vagrant vagabond [187,200,202]. The seemingly helpless state gives cause for concern. The antiepileptic drugs (AEDs) are still to prove the magic pill. But KD appears to be revolutionizing the treatment landscape for epilepsy. The intervention package for epilepsy in Nigeria should be expanded to include use of KD, such that treatment method can be determined by informed choices rather than the availability of options.

Of great interest is the resurgence of KD in the United States and which use has expanded to most major academic medical centres [45,59]. In parallel with its increase use in the United States, physicians in many other countries are now using KD in the treatment of epilepsy. In Korea where the diet was in collision with the dietary culture of the people, the efficacy of the diet has successfully won over doctors and patients families [177]. The diet has the potential advantage of theoretically being available everywhere and at lower costs than the newer AEDs [45]. The work of Kossoff and McGrogan reveals not one of the 70 centres in 41 countries outside the United States involved in the diet program has abandoned the use of KD [45]. Rather, the growing acceptance of the diet was underscored in the first recommendations written by 26 neurologists and dieticians from 9 countries, which were published in Epilepsia in November 2008 [202].

KD has been used to increase the effectiveness of conventional therapies thus helping to reduce their untoward and dose-related effects [31,173,203,204]; and almost exclusively for intractable epilepsy after multiple anticonvulsants have been tried unsuccessfully [45,144,164]. It is recognized that in such patients the probability of successful drug treatment is often extremely low. The diet is highly effective in this category of patients, resulting in greater than 50% improvement in seizure frequency in the two-thirds of those treated, and complete cessation of seizures in 7-23% [101,144,164,205]. The initial challenges posed by issues of tolerability and palatability have been subject of global research and, underlie the modification in the diet protocols [206].

The establishment of a KD program however requires a considerable amount of organization. This usually includes development of protocols for outpatients and inpatients care, investigations to monitor the diet, parents/patients/carer educational materials, as well as a satisfaction and quality of life survey for parents/patients/carers to evaluate the subjective response to therapy [169]. In this regard, academic medical centres may appear best suited as entry point for the diet program, especially in developing African countries.

Internationally, the diet appears to be gaining momentum both academically and with the general public. With the exception of majority of Africa and Central America, centres providing KD can be found everywhere, often with several in each nation [45]. In the United States, nearly 80 centres use the diet. South Africa (recently Egypt), with centres in Pretoria and Cape Town, appears the only country that is established in the diet programme in Africa. And it has such large referral base that the centres are in the process of establishing a support network for interested neurologists and dieticians with the help of the Epilepsy South Africa Organisation [45].

Nations and centres have now progressed to the point of offering the diet in their cultural, religious and financial setting [207]. Meals and recipes could come from foods available and traditional to the countries. Vegetables have been used as recipe in meals in South Africa [45], and Nigeria is home to a wide variety of vegetables with low carbohydrate content [208,209]. The country also boasts of huge human capital in science and medicine, and a good number of academic medical centres. These are but a few of the strengths available in the country for the diet program. The program when started is likely to enjoy International cooperation/collaboration, and support from Government, local charity groups and food companies.

CONCLUSIONS

It is a matter of decision, for Nigerian scientists and academic medical centres to join in the diet program in the African continent. This would not only help to address the issue of choice in treatment for the suffering patients but also, make available a whole lot of local research data in the country. It is a programme that could create a win-win situation for all. KD has established itself as an antiepileptic ‘product’ and is racing to become a universal remedy of sort. Efficacy in epilepsy is not much the issue with KD as its mode of action. Nigeria, and indeed Africa, is presented with an option it can no longer wait to embrace and exploit.

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