THE HISTORY AND EFFICACY OF THE KETOGENIC DIET

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by

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Abstract

The ketogenic diet is a trending diet garnering much attention and interest. But do people know what it is, how it works, and where it came from? The ketogenic diet is a high-fat, low-carbohydrate diet that was originally created to treat childhood epilepsy but has been found useful for many other things. Because of its low carbohydrate allowance, the body is deprived of glucose, its main fuel source, and instead burns stored fat into ketone bodies for energy. This process of ketosis promotes weight loss in a highly controversial way and can implicate unhealthy consequences. Ketosis can quickly worsen into ketoacidosis, a life-threatening condition that dangerously lowers the pH of the blood. In addition, the ketogenic diet's recommended high fat intake can increase LDL cholesterol levels, which can quickly become detrimental to health. Not only this, it may promote rapid weight loss in a short period of time, but does not guarantee that weight loss will be sustained. Several articles are under examination to determine the ketogenic diet's efficacy and if it is trending for all the right or wrong reasons. Low-carbohydrate (ketogenic) diets and low-fat diets are compared in numerous studies and ways, including weight loss results, cholesterol and lipid levels, but somehow inconsistent results prevail. Through the use of randomized control trials, case reports, and several systematic reviews with meta-analyses, the ketogenic diet's history, worldwide prevalence, and uses beyond weight loss, including treatment of certain cancers, are brought to light in a way that reveals the truth and significant bodily effects behind its trending name.

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What is the ketogenic diet?

The ketogenic diet is by nature a high-fat, low-carbohydrate, low-protein diet. The extremely limited consumption of carbohydrates significantly reduces the levels of glucose in the body. Glucose is the primary source of fuel for most tissues in the body so when it becomes unavailable, the body begins to break down its fat stores into ketone bodies to be used as an alternate source of energy. This process is known as ketosis and if prolonged, can result in ketoacidosis, which occurs when the body continues to break down fat into ketone bodies at an even faster rate. During ketoacidosis, the numerous amounts of ketones circulating in the body can lower the pH of blood to a dangerous level. Ketoacidosis is a life-threatening condition that can quickly lead to death if not treated. The biological effects of the ketogenic diet mimic what happens in the body when fasting or even starvation occurs. Individuals who undergo starvation/fasting and individuals who consume a ketogenic diet all enter this state of ketosis.

History of the Ketogenic Diet

The ketogenic diet was developed in the 1920s as a way to treat epilepsy in children (Stafstrom & Rho, 2004). Originally, fasting was used to treat seizures because anecdotal evidence at that point suggested that complete abstinence from food and drink provided a cure. However, this practice was found to be more successful in children than in adults (Conklin, 1922). The makeup of the ketogenic diet was first proposed as one gram of protein per kilogram of body weight (in children), 10-15 grams of carbohydrates per day, and the remainder of calories designated to fat (Stafstrom & Rho, 2004). This distribution is identical to the ketogenic diet that is in practice today. When following this diet,

significant improvements in behavior and cognitive effects were reported along with reduction of seizures (Peterman, 1925). The resulting successes created extensive interest in the ketogenic diet, as there have been many studies done that evaluate changes in mood, behavior, and cognitive skills (in addition to the reduction of seizures) when following the ketogenic diet.

Use of the Ketogenic Diet in Childhood Epilepsy

Because of the early potential exhibited by the ketogenic diet in treating childhood epilepsy, a systematic review was conducted to determine the effectiveness of the ketogenic diet as a treatment for childhood epilepsy (Keene, 2006). The inclusion criteria of studies consisted of the following: written in English or French, completed after 1990, and patients 18 years old or younger with diagnosed refractory epilepsy, which is defined as ineffectual control of seizures despite use of multiple antiepileptic medications. The authors considered the outcomes in a time period of 6 months where the key outcome measured was the change in seizure frequency, where 100% would be complete cessation of seizures and seizure reduction of 50% or greater was considered a "significant decrease." With these criteria, a total of 26 studies were included, which provided a total patient population of 972. All individuals had previously used antiepileptic medication without a significant decrease in seizures. Each study was then categorized as either class one, two, or three evidence. Class one was defined as including a prospective-controlled study with well-defined cohort, adequate sample size, blinded interpretation of outcome, and presence of a description of method analysis. Unfortunately, none of the 26 studies were classified as class one. Class two evidence was defined as either being an

uncontrolled prospective study or a retrospective study that had well-defined cohort, an adequate sample size, blinded interruption of outcome, and a description of method analysis. Fifteen of the studies were considered to be class two. Any study that did not meet these standards was considered to be class three. Only the studies that qualified as class two were included in the analysis of the efficacy of the diet, and are shown below in Table 1 from the article.

Author	Diet Fast	Design	Total Sample	% Sample at 6 Months	% Seizure -Free*	% Greater Than 50% Reduction*
DiMario [2]	y	R	48	50	8	35
Coppola [3]	y	R	56	38	7	20
Maydell [4]	y	R	146	66	16	12
Hassan [5]	y	R	53	39	11	26
Kankirawatana [6]	y	R	35	57	17	40
Kang [7]	y	R	199	61	33	58
Nordli [8]	y	R	32	66	19	22
Vining [9]	y	P	51	69	12	53
Freeman [10]	y	P	150	77	3	51
Kossoff [11]	y	R	23	78	17	55
Kinsman [12]	y	R	58	?	29	38
Ruthenstein [13]	y	R	13	77	6	15
Lion François [14]	y	R	29	?	10	35
Wirrell [15]	n	R	14	86	14	14
Vaisleib [16]	n	R	65	100	32	22

Abbreviations:
N = No fast
P = Prospective -no control group
R = Retrospective
Y = Fast present
* Percentage based on initial sample size (intent to treat).

Table 1. Description of studies using the ketogenic diet as a treatment for childhood epilepsy (Keene, 2006)

Unfortunately, the remaining studies were only used in the analysis of adverse effects of the ketogenic diet, which are not under examination in this case. At the end of the 6-month time period, 15.6% of the total patients had 100% cessation of seizures while 33.0% had a seizure reduction of 50% or more. This study did show successes of the ketogenic diet treating childhood epilepsy but still left many questions unanswered, such as: Which type of patient would the ketogenic diet benefit most? What is the necessary or optimal duration of following the ketogenic diet to yield benefits with regard to epilepsy?

Which patient is at risk of an adverse event? Is there a cost/benefit of the diet? The authors concluded that more research needs to be conducted, but because of the large range of antiepileptic medications available today, the ketogenic diet is uncommonly used as an exclusive treatment for epilepsy and therefore less time and attention is devoted to answering these questions.

Worldwide Use of the Ketogenic Diet

Since epilepsy is a somewhat prevalent condition (affecting about 100 million people worldwide) in all races and cultures, The Johns Hopkins Medical Institutions conducted a worldwide survey-based study in 2005 to evaluate the use of the ketogenic diet worldwide (Kossoff & Mcgrogan, 2005). Unfortunately, many countries cannot afford or do not have access to anticonvulsant drugs as a treatment for epilepsy so the ketogenic diet is their best option. The investigators contacted international ketogenic diet centers via email for information regarding patient enrollment, year the diet was first offered, unique cultural and religious issues in the country, community opinion, and research interests. After receiving responses, they had collected full historical information about the ketogenic diet in 73 centers in 41 different countries (outside of the United States). While the types of foods consumed in each country varied, they all successfully followed the proper ketogenic diet ratio. Although the exact number of people using the ketogenic diet as a treatment for epilepsy is unknown, it is considered to be a highly utilized diet worldwide. The map below, Figure 1, taken from this article, shows which countries have individuals following the ketogenic diet.

Figure 1. Countries providing the ketogenic diet highlighted in gray (Kossoff & Mcgrogan, 2005)

Ketogenic Diet as a Treatment for Brain Cancer

For the past 30 years, the ketogenic diet has been under investigation to determine if it is a possible treatment for brain cancer – specifically for glioblastomas, which are extremely aggressive tumors that develop in the brain. Because many tumors depend on glucose for development and survival, it has been proposed that the lack of glucose consumed during the ketogenic diet can inhibit the growth of tumors (Schwartz, Noel, Nikolai, & Chang, 2018). Several studies have been conducted that assess the effects of the ketogenic diet on tumor cell growth and survival, both in humans and in animals. A randomized control trial tested both *in vitro* and *in vivo* models to determine if ketone bodies decrease tumor cell viability and prolong survival time of mice with metastatic cancer (Poff, Ari, Arnol, Seyfried, & Dagostino, 2014). The *in vitro* model used VM-M3/Fluc cells, which are known to progress and metastasize in an extremely similar

manner to that of metastatic cancers. These cells were extracted from a spontaneous brain tumor that developed in VM/Dk inbred mice. These cells were plated into one of four groups: high or low glucose media with or without ketone supplementation. The authors found that low glucose, ketone supplemented cells had the slowest cell proliferation rate and the greatest percent of cell viability (close to 100%) in contrast to the high glucose, non-supplemented group that had the fastest cell proliferation rate and the smallest percent of cell viability (close to 75%). The data results in Figure 2 are shown below, taken from the article.

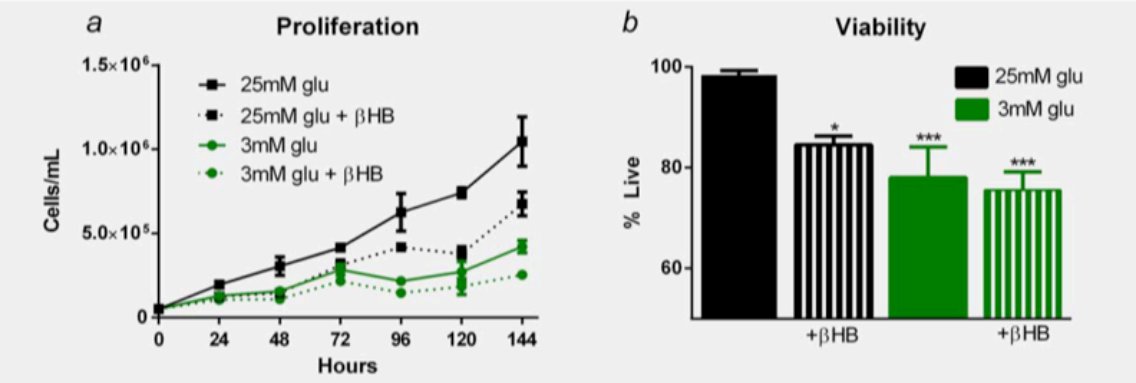

Figure 2. *In vitro* cell proliferation and viability data (Poff, Ari, Arnol, Seyfried, & Dagostino, 2014)

The *in vivo* model included 36 adult male VM/Dk mice that were all implanted with VM-M3/Fluc (cancer) cells and were assigned to 4 different groups: standard diet, calorie restricted, 1,3-butanediol (a type of ketone body), or ketone ester. They found that the bioluminescence scan of all mice did not show significantly different tumor changes. However, the mice supplemented with ketones demonstrated a significantly prolonged survival time compared to the control mice; the mice supplemented with ketone esters showed the greatest increase in mean survival time. The data results in Table 2 are shown below, taken from the article.

Treatment	Cohort Size (N)	Mean Survival Time (days)	Increase in Mean Survival Time
Control (SD)	13	31.2	---
CR	8	36.9	18.7%
BD	7	47.0	50.6%*
KE	8	52.8	69.2%***

Table 2. In vivo survival time data*; *p < 0.05; ***p < 0.001* (Poff, Ari, Arnol, Seyfried, & Dagostino, 2014)

There have been several similar studies conducted which led to the creation of a systematic review that was able to include 13 studies (Miraghajani, et al., 2017). The sole inclusion criterion was that a study had to be an experimental study on animals that reported effects of the ketogenic diet (as the only treatment) on tumor cells growth rate and survival time. Table 3 below was taken from this systematic review describing all 13 studies that were used including the first author, country, year, animal model, cancer type, study design, age, sex, study duration, type of intervention, sample size, main result, and P-values.

Author/year/country	Animal model/ sample size	Tumor type	Age (week)	Study design	Sex	Duration (day)	Type of diet/hypo, hyper/iso	Result
Shukla/2014/USA	Mice/9, 9	Pancreatic	Not mentioned	Experimental	Female	21	ND (*ad libitum*), KD/hyper	Tumor growth: KD < ND
Poff/2013/USA	Mice/13, 8	Not mentioned	10-18	Randomized Experimental	Male	90	SD, KD/hyper	Tumor growth: KD < SD Survival time: KD > SD
Allen/2013/USA	Mice/6, 7	Lung	4-6	Experimental	Female	80	SD, KD/not mention	Tumor growth: KD < SD
Caso/2012/USA	Mice/37, 37, 38, 37	Prostate	4-6	Randomized Experimental	Male	100	WD (WD, NCKD, 10%, 20% CKD)/ hyper	Tumor growth: 20% CKD < W Survival time: 20% CKD > WI
Abdelwahab/2012/ USA	Mice/19, 19	Glio ma	10	Randomized Experimental	Male	150	SD[a] KC[b]/not mention	Survival time: KC > SD Tumor growth: KC < SD
Kim/2012/USA	Mice/60	Prostate	6-8	Randomized Experimental	Male	54	WD[c] + Veh NCKD/hyper+veh	Tumor growth: NCKD < WD
Masko/2010/USA	Mice/50, 50, 50	Prostate	6	Randomized Experimental	Male	100,60	NCKD[e]/hyper CHO 10%f/hyper CHO 20%h/hyper	Survival time: NSg Tumor growth: NS
Mavropoulos/2009/ UK	Mice/48, 41, 41	Prostate	8	Randomized Experimental	Male	90,70	NCKD[i] MCD[j]/hyper LFD[k]/hyper	Tumor growth: NCKD < MCD Survival time: NCKD < MCD
Otto/2008/UK	Mice/24	Gastric	6-8	Randomized Experimental	Female	20	KD[l]/hyper SD	Tumor growth: KD < SD Survival time: KD > SD
Zhou/2007/USA	Mice/12-14 (per group)	Brain	10-12	Experimental	Male	7	SD or KD[m]/hyper Ror (UN)	Tumor growth: SD-UR, KD-UI Survival time: KC-R > SD-UR KC-UR
Seyfried/2003/UK	Mice/33	Brain	10-12	Experimental	Male	13	SDUR KD-UR/hyper SD-R KD-R/hyper	Tumor growth: UR > R
Hao/2015/China	Mice/12, 12, 12	Colon	4-6	Randomized Experimental	Male	40	MKD[n] LKD[o] Hyper SD	Tumor growth: MKD/LKD < S Survival time: MKD (35.1) > LKD (33.8) > SD (24.8)
Morscher/2015/ Austria	Mice/11, 11, 11, 11	Neuroblastoma	5-6	Randomized Experimental	Female	35	SD CR-SD[p] KD/not mention CR-KD[q]	Tumor growth: CR-KD < KD · CR-SD < SD Tumor volume: SD: 3541±219 mm^3>CR-SD: 1884±256 mm^3 KD: 1721±78 mm^3 > CR-KD: 1199±158 mm^3 Survival time: CR-KD (100%) > KD (73%) > CR-SD (83%) > SD (36%)

[a]Standard rodent chow, [b]72% fat, 3% carbohydrate, 15% protein, [c]35% fat, 49% carbohydrate, 16% protein, [d]84% fat, 0% carbohydrate, 16% protein, [e]84% fat, 0% [f]74% fat, 10% carbohydrate, 16% protein, [g]Not significant, [h]64% fat, 0% carbohydrate, 16% protein, [i]83% fat, 0% carbohydrate, 17% protein, [j]40% fat, 43% carb fat, 71% carbohydrate, 17% protein, [k]35.5% fat, 0.2% carbohydrate, 13% protein, [m]80% fat, 3.3% carbohydrate, 16.7% protein, [n]36.2% MCT, 21.8% Omega 3, 20% protein, [o]69% fat, 3% carbohydrate, 20% protein, [p]Calorie restriction SD, [q]Calorie restriction KD. R=Restricted, UR=Unrestricted, SDUR=Standard diet un KD=KetoCal diet, CR=Calorie-restricted, ND=Normal diet, WD=Western diet, CKD=Carbohydrate ketogenic diet, NCKD=Noncarbohydrate ketogenic diet, M diet, LFD=Low carbohydrate diet, CHO=Carbohydrate

Table 3. Summary of studies included in the systematic Review (Miraghajani, et al., 2017)

Of the 13 studies included, all concluded that the ketogenic diet had an inhibitory effect on tumor growth and 9 included evidence indicating that the ketogenic diet inhibited tumor growth and even prolonged survival time of the animals. Because this is a systematic review, it was not limited solely to glioblastomas but also to pancreatic, prostate, gastric, colon, and lung cancers. These studies vary greatly in animal physiology, tumor make-up, ketogenic diet components/ratio/administration, and living

conditions. However, these studies exclusively utilize animal models and therefore it cannot be definitively concluded that these results would transfer to humans.

In this context, a study was performed to see whether humans, particularly children in this case, would have the same successes with the ketogenic diet (Nebeling & Lerner, 1995). A sample of pediatric patients with advanced-staged cancer were given a ketogenic diet that consisted of 60% medium-chain triglyceride oil, 20% protein, 10% carbohydrate, and 10% other dietary fats to test the effects on tumor glucose metabolism in an 8-week time period. They assessed changes in tumors through PET scans and found an average 21.8% decline in glucose uptake at the tumor sites.

Following this study, a systematic review was written that investigated the role of ketogenic diets on adult and pediatric gliomas (Martin-Mcgill, et al., 2018). However, they were unable to find any randomized control trials and instead used six case reports, totaling 39 patients total. They found that all studies reported overall "progression-free survival", but because they were case studies, the effectiveness of the ketogenic diet as a treatment for gliomas in humans could not be established. They stated that the acceptability of the ketogenic diet is unknown and that randomized control trials need to be conducted.

What is the ketogenic diet used for now?

Although developed as a therapy for epilepsy, the ketogenic diet is now widely used as a weight loss and weight maintenance diet. However, because of its discordance with standard dietary guidelines that state an adult should consume 130 grams of carbohydrates a day and consume only 20-35% of daily caloric intake from fats (U.S.

Department of Health and Human Services and U.S. Department of Agriculture, 2015) it leaves many people skeptical about its safety and effectiveness. Its prevalence has triggered the exploration of the diet's mechanisms and efficacy in promoting weight loss. However, there are still many gaps in the evidence, as some studies strongly support the diet and some do not find it any more beneficial in promoting weight loss than a standard low-fat diet. It is proposed that while following the ketogenic diet, weight loss is achieved through a combination of factors including the satiating effect of protein, increased energy expenditure, appetite suppression from ketosis, increased bound-water loss, and food choice restriction (Malik & Hu, 2007). However, many argue that the weight loss during the ketogenic diet primarily results from water loss and food choice restriction, not just from ketosis. One of the biggest criticisms of the ketogenic diet is that it can be used as a way to initially lose weight, but is not an effective way to promote sustained weight loss. Numerous studies have been conducted comparing the weight loss results of low-carbohydrate diets (ketogenic) versus low-fat diets.

After evaluating several randomized control trials, the results remain inconsistent. However, there are many study samples and design factors to consider that may cause discordant results among studies, such as such as: BMI, age group, sex, duration of diet, chronic disease prevalence, physical activity level, and adherence to diet. One randomized control trial assessed the differences in a low-carbohydrate ketogenic versus a low-fat diet in severe obesity and found that in a 6-month period, the low-carbohydrate ketogenic diet group achieved greater weight loss (Samaha, et al., 2003). However, after one year the authors found that there were no longer significant differences in weight loss

between the two groups (Stockman, 2006). The graph below, figure 3, is taken from the article.

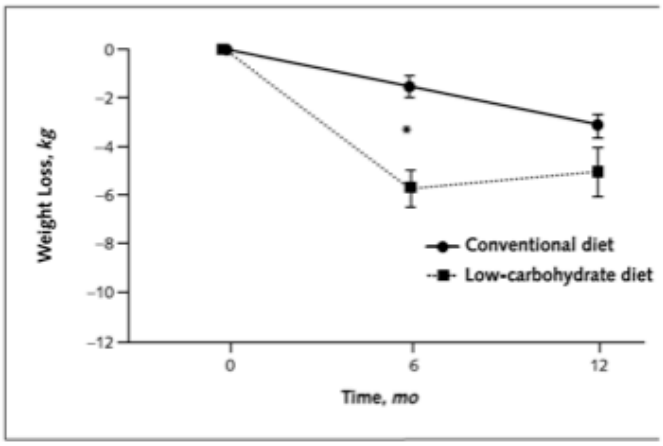

Figure 3. Data showing weight loss differences between low-carbohydrate ketogenic diet and low-fat diet in severe obesity (Stockman, 2006).

A systematic review with meta-analysis, examined 13 studies, which included a total of 1577 individuals, that compared the differences in weight loss between a very-low-carbohydrate ketogenic diet and a low-fat diet (Bueno, Melo, Oliveira, & Ataide, 2013). Each individual was randomly assigned to a group, very-low-carbohydrate diet (790 individuals) or low-fat diet (787 individuals). A very-low-carbohydrate-diet was defined as the consumption of less than 50 grams of carbohydrates per day or 10% of daily energy from carbohydrates and a low-fat diet was defined as a restricted-energy diet with less than 30% of daily energy from fat. The participants in these studies were assigned to the respective diets to assess potential changes in body weight and key risk factors. Table 4 below, taken from the article, includes key information of all 13 studies including duration of diet, amount of dietary counseling, number of dropouts, percentage of females, country, risk factor, mean age, mean BMI, and carbohydrate intake.

Source	Duration (months)	Dietary counselling	Dropouts (*n*/*N*)	Females (%)	Country	Risk factor	Mean age (years)	Mean BMI (kg/m^2)	CHO intake/d (VLCKD)*
Brinkworth *et al.*[28]	12	Adequate	38/107	70	Australia	CV risk factor	50·6	33·6	36 g
Dansinger *et al.*[50]	12	Inadequate	41/80	47	USA	CV risk factor	47	35	190 g
Davis *et al.*[51]	12	Adequate	14/105	78	USA	T2D	53·5	35·9	33 %
Dyson *et al.*[52]	24	Inadequate	4/26	73	UK	T2D	52	35·1	Unreported
Foster *et al.*[53]	12	Inadequate	37/63	68	USA	None	44·9	34·1	Unreported
Foster *et al.*[27]	24	Adequate	113/307	68	USA	None	45·5	36·1	Unreported
Gardner *et al.*[30]	12	Inadequate	26/153	100	USA	None	42	32	34 %
Iqbal *et al.*[29]	24	Adequate	76/144	10	USA	T2D	60	37·4	47 %
Lim *et al.*[54]	15	Inadequate	25/60	80	Australia	CV risk factor	48·4	31·4	36 %
McAuley *et al.*[55]	12	Inadequate	15/63	100	NZ	None	45	36·1	33 %
Shai *et al.*[49]	24	Adequate	44/213	16	Israel	CV risk factor	51·5	30·7	40 %
Stern *et al.*[26]	12	Adequate	45/132	17	USA	None	53·5	42·9	120 g
Truby *et al.*[56]	12†	Inadequate	98/116	72	UK	None	39·8	32	Unreported

CHO, carbohydrate; VLCKD, very-low-carbohydrate ketogenic diet; CV, cardiovascular; T2D, type 2 diabetes mellitus; NZ, New Zealand.
* Mean carbohydrate intake in the VLCKD group at the end of the follow-up, measured by dietary assessment, shown as g/d or percentage of energy from carbohydrates per d.
† Truby *et al.*[56] assessed only the body weight at 12 months.

Table 4. Systematic review study descriptions comparing very-low-carbohydrate ketogenic diets and low-fat diets (Bueno, Melo, Oliveira, & Ataide, 2013).

This systematic review analyzed several aspects of the studies including body weight, LDL and HDL cholesterol, and blood triglyceride levels. They determined that the individuals assigned to the very-low-carbohydrate (ketogenic) diet achieved greater weight loss and reduction in triglyceride levels compared to the individuals assigned to the low-fat diet. The mean differences in weight loss are shown below in Figure 4, taken from the article.

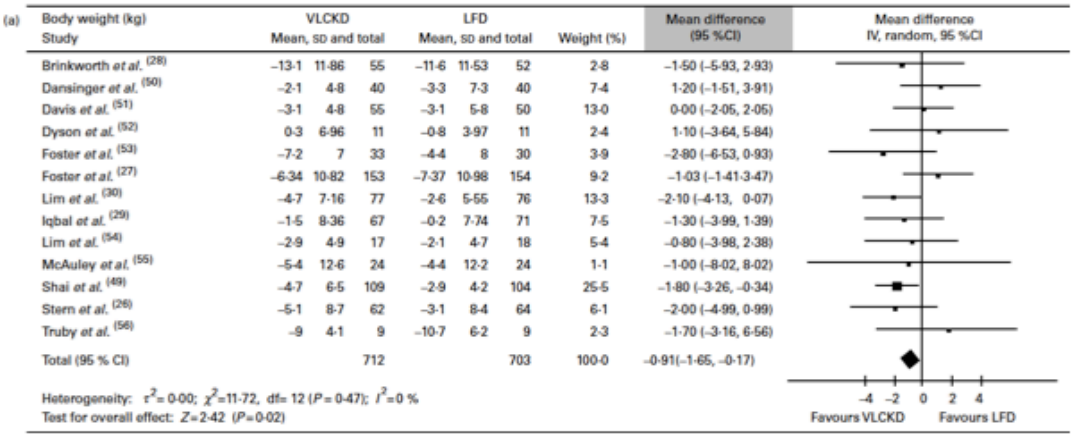

Figure 4. Data comparing weight loss between very-low-carbohydrate ketogenic diet and low-fat diet (Bueno, Melo, Oliveira, & Ataide, 2013).

However, the individuals in the very-low-carbohydrate ketogenic diet group also exhibited increases in HDL and LDL. They also found that the loss of body weight from a very-low-carbohydrate ketogenic diet can be the result of the increase in resting energy expenditure. This article concluded that very-low-carbohydrate ketogenic diets promote greater weight loss than low-fat diets and that ketogenic diets like these are an effective way to reduce obesity (and the risk of chronic diseases associated with obesity). However, these apparent benefits of a very-low-carbohydrate ketogenic diet should be weighed against evidence from this study suggesting a rise in LDL cholesterol, as it could be detrimental to one's health.

A second meta-analysis investigated several similar studies that compared weight loss results between low-carbohydrate and low-fat diets, but arrived at a different conclusion (Nordmann, et al., 2006). This study was comprised of five randomized control trails that included a total of 447 individuals. Like the previous study, each individual was randomly assigned to a group: very-low-carbohydrate diet (222 individuals) or low-fat diet (225 individuals). There are some discrepancies with the way low-fat diets were defined in each study; one study considered low-fat to include a maximum of 10% of daily energy to come from fat intake whereas the other 4 allowed a maximum of 30%. While this does not necessarily make a great difference in the data, it should be considered when evaluating the results. The data obtained indicated that greater weight loss was achieved in the low-carbohydrate diet groups in a 6-month period. However, at the 12-month mark, there was no significant difference in weight loss between the low-carbohydrate and low-fat diet groups.

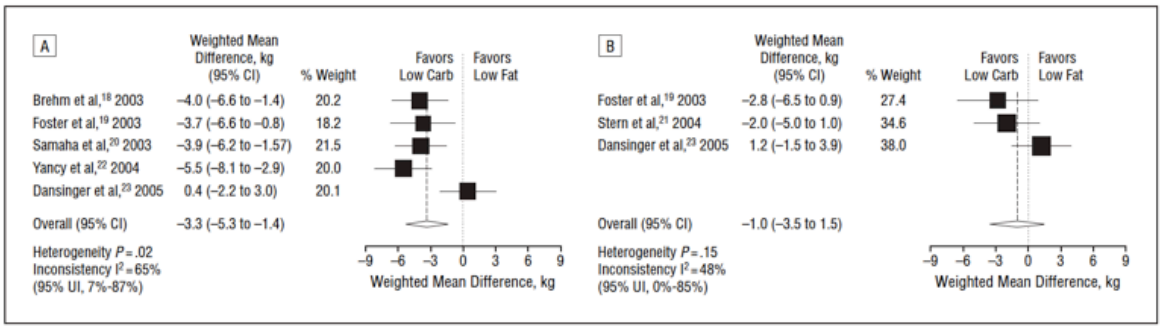

Figure 5. Forest plot showing weighted mean differences between low carb and low-fat diets (Nordmann, et al., 2006)

Though this was different than the previous systematic review, the results were similar pertaining to triglyceride, HDL and LDL cholesterol levels (meaning the low-carbohydrate groups had a decrease in triglyceride levels but an increase in both HDL and LDL cholesterol levels). Because there were no differences in weight loss between the low-fat and low-carbohydrate groups, along with the increase in LDL cholesterol levels in the low-carbohydrate group, this study did not find strong evidence to recommend a low-carbohydrate diet as a means for weight loss or risk reduction. They concluded that each diet is as effective as the other, but the changes in total cholesterol need to be taken into consideration when choosing a diet to follow.

When considering the different findings from these two systematic reviews, it is important to consider the differences in each study and what potentially led to these discordant findings. The first systematic review with meta-analysis concluded that a very-low-carbohydrate ketogenic diet yielded greater weight loss than the low-fat diet, however some things come to light when looking closer. Their study had greater dropout rates than the second systematic review with meta-analysis and they stated in the conclusion that very-low-carbohydrate ketogenic diets produce greater weight loss results was consistent with all studies except for the studies that had 24-month follow ups. They go further to mention that the weight-loss differences they observed long-term appear to

be of little clinical significance, although statistically significant. This appears to be more in line with what the second systematic review with meta-analysis concluded and agrees with the criticism that the ketogenic diet can be used as a way to initially lose weight, but is not an effective way to promote sustained weight loss.

Discussion

The ketogenic diet is a relevant and impactful diet in nutritional sciences today. Many people are unaware of where this trending diet came from, how it was created, and how it became so popular. Evidence shows that it has been successful in treating childhood epilepsy, but whether or not it is a viable treatment for certain tumors and for weight loss is still in question. Clinical trials are currently being conducted to investigate the ketogenic diet as a treatment for brain cancers (recurrent/refractory brain tumors) and are scheduled to be completed around November 2023. Hopefully such investigations will be able to give answers to some of the unanswered questions many have about the ketogenic diet's efficacy when treating cancer. With regard to the ketogenic diet as a means for weight loss, long-term studies (5-10 years) need to be conducted to discover if weight loss is continued/maintained and if cholesterol levels continue to increase. Many people do not consider the long-term effects that certain exposures have on the body, but just immediate results. It is imperative to understand exactly what a diet could do to the body, helpful or harmful, in a long-term perspective. There are many other ideas and myths about the ketogenic diet and what positive effects it can have on the body, but because of the negatives that are unable to be ignored, I would not recommend the ketogenic diet as a weight loss solution.

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