Céto Minceur

L'alimentation Céto/LCHF est une alimentation qui se prête bien à la perte de poids. (1) (2)

Le corps ne fonctionne plus en brûlant les sucres (via le glucose) mais en brûlant les graisses (via les cétones), y compris les graisses stockées dans le corps.

L'alimentation Céto/LCHF entraîne une baisse de la faim et un signal plus marqué de la satiété, au point que de nombreuses personnes qui l'adoptent réduisent naturellement leurs quantités d'aliments. (3)

Enfin, l'alimentation Céto/LCHF rétablit le fonctionnement hormonal optimal : les personnes souffrant de résistance à l'insuline (état de pré-diabète) peuvent à nouveau rétablir un niveau d'insuline bas. C'est aussi cette hormone qui gère le stock des gras corporels. Quand elle fonctionne bien, on retrouve une ligne normale. (4)

Comment faire pour manger Céto/LCHF afin de perdre du poids ?

- Bien repérer les aliments qui entraînent une production d'insuline et les éviter (sucres, amidons).
- Choisir tous les jours de bonnes sources de lipides.
- Ecouter sa faim, et ne manger que lorsqu'on a faim.
- S'arrêter quand on n'a plus faim.
- Modérer les protéines à une portion par repas (soit 30 gr de protéines, ce qui correspond à la taille d'une paume de viande ou de poisson).
- Trouver des recettes Céto/LCHF qui permettent de se faire plaisir tout en restant dans les conditions d'une alimentation faible en glucides et riche en lipides.
- S'assurer qu'on choisit des aliments contenant suffisamment de micro-nutriments (il n'existe aucun micro-nutriments que l'alimentation Céto ne puisse apporter. Toutes les vitamines et les polyphénols des fruits se retrouvent aussi dans les légumes).

Cette alimentation Céto/LCHF est-elle bonne pour la santé ?

Cette alimentation a fait l'objet de nombreuses études, et notamment à des fins thérapeutiques (pour soigner l'épilepsie, le diabète, le cancer, la maladie de Parkinson, la dépression, l'autisme, la maladie d'Alzheimer, les maladies auto-immunes inflammatoires). Pour ces patients, elle n'entraîne pratiquement aucune contre-indication ni effets secondaires.

Pour les personnes qui souhaitent perdre du poids, il n'y a pas d'effet secondaire, et il semble par ailleurs que cette approche soit plus efficace et plus agréable à adopter qu'un régime basses calories classique. (5)

Par ailleurs, outre le poids qu'elles perdent plus ou moins vite selon les personnes, les métabolismes de chacun et l'état hormonal de chacun, elles bénéficient aussi d'amélioration de leur santé :

- Une humeur stable et positive (6)
- Plus d'énergie pendant la journée (grâce à un approvisionnement constant en cétones)
- Une baisse d'inflammation, entraînant de meilleures défenses immunitaires (7)
- Une plus belle peau (les peaux acnéïques peuvent redevenir normales) et de plus beaux cheveux (8)
- De meilleures capacités cognitives (le cerveau est un grand consommateur de gras) (9)
- Une meilleure absorption des vitamines lipo-solubles
- De meilleurs bilans sanguins (abaissement des triglycérides, du cholestérol LDL) (10)

La perte de poids est-elle garantie ?

Il est courant quand on adopte l'alimentation Céto/LCHF, de perdre quelques kilos les premiers jours, qui sont en général des kilos de fluides corporels. Le corps se déleste d'une partie de ses stocks d'eau, dont il n'a plus besoin lorsqu'il s'adapte à la cétose.

Par la suite, les résultats sont assez variables. Les personnes qui ont beaucoup de poids à perdre y parviendront en général plus rapidement, de même que celles qui consommaient de grandes quantités de glucides.

L'alimentation Céto/LCHF participe à un meilleur fonctionnement hormonal (notamment via les hormones insuline, ghréline et leptine). Mais d'autres hormones et glandes exercent aussi une fonction sur le poids : la thyroïde, les surrénales, ainsi que les hormones sexuelles (oestrogènes notamment). Certaines personnes souffrant d'un dérèglement hormonal pourront avoir moins de facilité à perdre du poids.

Il arrive aussi que des intolérances à des produits particuliers empêchent de perdre du poids : le plus souvent, les produits laitiers et les oléagineux. Les supprimer aide alors à perdre le poids superflu.

Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomized controlled trials.

Abstract

BACKGROUND:

Low-carbohydrate diets have become increasingly popular for weight loss. However, evidence from individual trials about benefits and risks of these diets to achieve weight loss and modify cardiovascular risk factors is preliminary.

METHODS:

We used the Cochrane Collaboration search strategy to identify trials comparing the effects of low-carbohydrate diets without restriction of energy intake vs low-fat diets in individuals with a body mass index (calculated as weight in kilograms divided by the square of height in meters) of at least 25. Included trials had to report changes in body weight in intention-to-treat analysis and to have a follow-up of at least 6 months. Two reviewers independently assessed trial eligibility and quality of randomized controlled trials.

RESULTS:

Five trials including a total of 447 individuals fulfilled our inclusion criteria. After 6 months, individuals assigned to low-carbohydrate diets had lost more weight than individuals randomized to low-fat diets (weighted mean difference, -3.3 kg; 95% confidence interval [CI], -5.3 to -1.4 kg). This difference was no longer obvious after 12 months (weighted mean difference, -1.0 kg; 95% CI, -3.5 to 1.5 kg). There were no differences in blood pressure. Triglyceride and high-density lipoprotein cholesterol values changed more favorably in individuals assigned to low-carbohydrate diets (after 6 months, for triglycerides, weighted mean difference, -22.1 mg/dL [-0.25 mmol/L]; 95% CI, -38.1 to -5.3 mg/dL [-0.43 to -0.06 mmol/L]; and for high-density lipoprotein cholesterol, weighted mean difference, 4.6 mg/dL [0.12 mmol/L]; 95% CI, 1.5-8.1 mg/dL [0.04-0.21 mmol/L]), but total cholesterol and low-density lipoprotein cholesterol values changed more favorably in individuals assigned to low-fat diets (weighted mean difference in low-density lipoprotein cholesterol after 6 months, 5.4 mg/dL [0.14 mmol/L]; 95% CI, 1.2-10.1 mg/dL [0.03-0.26 mmol/L]).

CONCLUSIONS:

Low-carbohydrate, non-energy-restricted diets appear to be at least as effective as low-fat, energy-restricted diets in inducing weight loss for up to 1 year. However, potential favorable changes in triglyceride and high-density lipoprotein cholesterol values should be weighed against potential unfavorable changes in low-density lipoprotein cholesterol values when low-carbohydrate diets to induce weight loss are considered.

A Randomized Trial of a Low-Carbohydrate Diet for Obesity

Abstract

BACKGROUND

Despite the popularity of the low-carbohydrate, high-protein, high-fat (Atkins) diet, no randomized, controlled trials have evaluated its efficacy.

METHODS

We conducted a one-year, multicenter, controlled trial involving 63 obese men and women who were randomly assigned to either a low-carbohydrate, high-protein, high-fat diet or a low-calorie, high-carbohydrate, low-fat (conventional) diet. Professional contact was minimal to replicate the approach used by most dieters.

RESULTS

Subjects on the low-carbohydrate diet had lost more weight than subjects on the conventional diet at 3 months (mean [±SD], –6.8±5.0 vs. –2.7±3.7 percent of body weight; P=0.001) and 6 months (–7.0±6.5 vs. –3.2±5.6 percent of body weight, P=0.02), but the difference at 12 months was not significant (–4.4±6.7 vs. –2.5±6.3 percent of body weight, P=0.26). After three months, no significant differences were found between the groups in total or low-density lipoprotein cholesterol concentrations. The increase in high-density lipoprotein cholesterol concentrations and the decrease in triglyceride concentrations were greater among subjects on the low-carbohydrate diet than among those on the conventional diet throughout most of the study. Both diets significantly decreased diastolic blood pressure and the insulin response to an oral glucose load.

CONCLUSIONS

The low-carbohydrate diet produced a greater weight loss (absolute difference, approximately 4 percent) than did the conventional diet for the first six months, but the differences were not significant at one year. The low-carbohydrate diet was associated with a greater improvement in some risk factors for coronary heart disease. Adherence was poor and attrition was high in both groups. Longer and larger studies are required to determine the long-term safety and efficacy of low-carbohydrate, high-protein, high-fat diets.

Perceived Hunger Is Lower and Weight Loss Is Greater in Overweight Premenopausal Women Consuming a Low-Carbohydrate/High-Protein vs High-Carbohydrate/Low-Fat Diet

Abstract

The impact of a low-carbohydrate/high-protein diet compared with a high-carbohydrate/low-fat diet on ratings of hunger and cognitive eating restraint were examined. Overweight premenopausal women consumed a low-carbohydrate/high-protein (n=13) or high-carbohydrate/low-fat diet (n=15) for 6 weeks. Fasting body weight (BW) was measured and the Eating Inventory was completed at baseline, weeks 1 to 4, and week 6. All women experienced a reduction in BW (P<.01), although relative BW loss was greater in the low-carbohydrate/high-protein vs high-carbohydrate/low-fat group at week 6 (P<.05). Based on Eating Inventory scores, self-rated hunger decreased (P<.03) in women in the low-carbohydrate/high-protein but not in the high-carbohydrate/low-fat group from baseline to week 6. In both groups, self-rated cognitive eating restraint increased (P<.01) from baseline to week 1 and remained constant to week 6. Both diet groups reported increased cognitive eating restraint, facilitating short-term weight loss; however, the decrease in hunger perception in the low-carbohydrate/high-protein group may have contributed to a greater percentage of BW loss.

Carbohydrate Restriction has a More Favorable Impact on the Metabolic Syndrome than a Low Fat Diet

Abstract

We recently proposed that the biological markers improved by carbohydrate restriction were precisely those that define the metabolic syndrome (MetS), and that the common thread was regulation of insulin as a control element. We specifically tested the idea with a 12-week

study comparing two hypocaloric diets (~1,500 kcal): a carbohydrate-restricted diet (CRD) (%carbohydrate:fat:protein = 12:59:28) and a low-fat diet (LFD) (56:24:20) in 40 subjects with atherogenic dyslipidemia. Both interventions led to improvements in several metabolic markers, but subjects following the CRD had consistently reduced glucose (−12%) and insulin (−50%) concentrations, insulin sensitivity (−55%), weight loss (−10%), decreased adiposity (−14%), and more favorable triacylglycerol (TAG) (−51%), HDL-C (13%) and total cholesterol/HDL-C ratio (−14%) responses. In addition to these markers for MetS, the CRD subjects showed more favorable responses to alternative indicators of cardiovascular risk: postprandial lipemia (−47%), the Apo B/Apo A-1 ratio (−16%), and LDL particle distribution. Despite a threefold higher intake of dietary saturated fat during the CRD, saturated fatty acids in TAG and cholesteryl ester were significantly decreased, as was palmitoleic acid (16:1n-7), an endogenous marker of lipogenesis, compared to subjects consuming the LFD. Serum retinol binding protein 4 has been linked to insulin-resistant states, and only the CRD decreased this marker (−20%). The findings provide support for unifying the disparate markers of MetS and for the proposed intimate connection with dietary carbohydrate. The results support the use of dietary carbohydrate restriction as an effective approach to improve features of MetS and cardiovascular risk.

A Low-Carbohydrate as Compared with a Low-Fat Diet in Severe Obesity

BACKGROUND

The effects of a carbohydrate-restricted diet on weight loss and risk factors for atherosclerosis have been incompletely assessed.

METHODS

We randomly assigned 132 severely obese subjects (including 77 blacks and 23 women) with a mean body-mass index of 43 and a high prevalence of diabetes (39 percent) or the metabolic syndrome (43 percent) to a carbohydrate-restricted (low-carbohydrate) diet or a calorie- and fat-restricted (low-fat) diet.

RESULTS

Seventy-nine subjects completed the six-month study. An analysis including all subjects, with the last observation carried forward for those who dropped out, showed that subjects on the low-carbohydrate diet lost more weight than those on the low-fat diet (mean [±SD], –5.8±8.6 kg vs. –1.9±4.2 kg; P=0.002) and had greater decreases in triglyceride levels (mean, –20±43 percent vs. –4±31 percent; P=0.001), irrespective of the use or nonuse of hypoglycemic or lipid-lowering medications. Insulin sensitivity, measured only in subjects without diabetes, also improved more among subjects on the low-carbohydrate diet (6±9 percent vs. –3±8 percent, P=0.01). The amount of weight lost (P<0.001) and assignment to the low-carbohydrate diet (P=0.01) were independent predictors of improvement in triglyceride levels and insulin sensitivity.

CONCLUSIONS

Severely obese subjects with a high prevalence of diabetes or the metabolic syndrome lost more weight during six months on a carbohydrate-restricted diet than on a calorie- and fat-restricted diet, with a relative improvement in insulin sensitivity and triglyceride levels, even after adjustment for the amount of weight lost. This finding should be interpreted with caution, given the small magnitude of overall and between-group differences in weight loss in these markedly obese subjects and the short duration of the study. Future studies evaluating long-term cardiovascular outcomes are needed before a carbohydrate-restricted diet can be endorsed.

The effects of a low-carbohydrate ketogenic diet and a low-fat diet on mood, hunger, and other self-reported symptoms.

Abstract

OBJECTIVE:

To investigate the effects of weight loss diets on mood, food cravings, and other self-reported symptoms.

RESEARCH METHODS AND PROCEDURES:

Mood and other symptoms were evaluated by participant self-report using the Atkins Health Indicator Test (AHIT) in individuals undergoing weight loss following either a low-carbohydrate, ketogenic diet (LCKD) or a low-fat diet (LFD). Participants were 119 overweight community volunteers randomized to an LCKD or an LFD. An additional 51 participants who had completed an earlier trial contributed data for the psychometric analyses but were not included in the prospective analyses. Self-reported symptom levels on seven scales factor-analytically derived from the AHIT (negative affect, fatigue, somatic symptoms, physical effects of hunger, insomnia, hunger, and stomach problems) were acquired during 12 visits.

RESULTS:

After adjusting for the change in BMI over the course of the trial, participants experienced significant improvements in most symptoms regardless of diet. Diet group x visit interactions were observed for negative affect [$F(9,803) = 2.30$, $p = 0.015$] and hunger [$F(9,803) = 3.62$, $p < 0.0002$]. Examination of means indicated that the LCKD group reported less negative affect and hunger, compared with the LFD group.

DISCUSSION:

Regardless of diet, participants experienced significant improvement in a broad range of symptoms. Symptoms of negative affect and hunger improved to a greater degree in patients following an LCKD compared with those following an LFD. Whether these symptom changes explain the greater short-term weight loss generally experienced by LCKD followers deserves further research.

The ketone metabolite β-hydroxybutyrate blocks NLRP3 inflammasome-mediated inflammatory disease.

Abstract

The ketone bodies β-hydroxybutyrate (BHB) and acetoacetate (AcAc) support mammalian survival during states of energy deficit by serving as alternative sources of ATP. BHB levels are elevated by starvation, caloric

restriction, high-intensity exercise, or the low-carbohydrate ketogenic diet. Prolonged fasting reduces inflammation; however, the impact that ketones and other alternative metabolic fuels produced during energy deficits have on the innate immune response is unknown. We report that BHB, but neither AcAc nor the structurally related short-chain fatty acids butyrate and acetate, suppresses activation of the NLRP3 inflammasome in response to urate crystals, ATP and lipotoxic fatty acids. BHB did not inhibit caspase-1 activation in response to pathogens that activate the NLR family, CARD domain containing 4 (NLRC4) or absent in melanoma 2 (AIM2) inflammasome and did not affect non-canonical caspase-11, inflammasome activation. Mechanistically, BHB inhibits the NLRP3 inflammasome by preventing K(+) efflux and reducing ASC oligomerization and speck formation. The inhibitory effects of BHB on NLRP3 are not dependent on chirality or starvation-regulated mechanisms like AMP-activated protein kinase (AMPK), reactive oxygen species (ROS), autophagy or glycolytic inhibition. BHB blocks the NLRP3 inflammasome without undergoing oxidation in the TCA cycle, and independently of uncoupling protein-2 (UCP2), sirtuin-2 (SIRT2), the G protein-coupled receptor GPR109A or hydrocaboxylic acid receptor 2 (HCAR2). BHB reduces NLRP3 inflammasome-mediated interleukin (IL)-1β and IL-18 production in human monocytes. In vivo, BHB or a ketogenic diet attenuates caspase-1 activation and IL-1β secretion in mouse models of NLRP3-mediated diseases such as Muckle-Wells syndrome, familial cold autoinflammatory syndrome and urate crystal-induced peritonitis. Our findings suggest that the anti-inflammatory effects of caloric restriction or ketogenic diets may be linked to BHB-mediated inhibition of the NLRP3 inflammasome.

Beyond weight loss: a review of the therapeutic uses of very-low-carbohydrate (ketogenic) diets.

Abstract

Very-low-carbohydrate diets or ketogenic diets have been in use since the 1920s as a therapy for epilepsy and can, in some cases, completely remove the need for medication. From the 1960s onwards they have become widely known as one of the most common methods for obesity

treatment. Recent work over the last decade or so has provided evidence of the therapeutic potential of ketogenic diets in many pathological conditions, such as diabetes, polycystic ovary syndrome, acne, neurological diseases, cancer and the amelioration of respiratory and cardiovascular disease risk factors. The possibility that modifying food intake can be useful for reducing or eliminating pharmaceutical methods of treatment, which are often lifelong with significant side effects, calls for serious investigation. This review revisits the meaning of physiological ketosis in the light of this evidence and considers possible mechanisms for the therapeutic actions of the ketogenic diet on different diseases. The present review also questions whether there are still some preconceived ideas about ketogenic diets, which may be presenting unnecessary barriers to their use as therapeutic tools in the physician's hand.

Diet-induced ketosis improves cognitive performance in aged rats.

Abstract

Aging is associated with increased susceptibility to hypoxic/ischemic insult and declines in behavioral function which may be due to attenuated adaptive/defense responses. We investigated if diet-induced ketosis would improve behavioral performance in the aged rats. Fischer 344 rats (3- and 22-month-old) were fed standard (STD) or ketogenic (KG) diet for 3 weeks and then exposed to hypobaric hypoxia. Cognitive function was measured using the T-maze and object recognition tests. Motor function was measured using the inclined-screen test. Results showed that KG diet significantly increased blood ketone levels in both young and old rats. In the aged rats, the KG diet improved cognitive performance under normoxic and hypoxic conditions; while motor performance remained unchanged. Capillary density and HIF-1alpha levels were elevated in the aged ketotic group independent of hypoxic challenge. These data suggest that diet-induced ketosis may be beneficial in the treatment of neurodegenerative conditions.

Effect of a low-carbohydrate, ketogenic diet program compared to a low-fat diet on fasting lipoprotein subclasses.

Abstract

BACKGROUND:

Low-carbohydrate, ketogenic diets (LCKD) are effective for weight loss, but concerns remain regarding cardiovascular risk. The purpose of this study was to determine the effect of an LCKD program on serum lipoprotein subclasses.

METHODS:

This was a randomized, two-arm clinical trial in an outpatient research clinic involving overweight, hyperlipidemic community volunteers motivated to lose weight. Subjects were randomized to either an LCKD (n = 59) and nutritional supplementation (including fish, borage and flaxseed oil), or a low-fat, reduced-calorie diet (LFD, n = 60). The main outcomes were fasting serum lipoprotein subclasses determined by nuclear magnetic resonance analysis.

RESULTS:

The mean age of subjects was 44.9 years, the mean BMI was 34.4 kg/m(2), and 76% were women. Comparing baseline to 6 months, the LCKD group had significant changes in large VLDL (-78%), medium VLDL (-60%), small VLDL (-57%), LDL particle size (+2%), large LDL (+54%), medium LDL (-42%), small LDL (-78%), HDL particle size (+5%), large HDL (+21%), and LDL particle concentration (-11%). Compared with the LFD group, the LCKD group had greater reductions in medium VLDL (p = 0.01), small VLDL (p = 0.01) and medium LDL (p = 0.02), and greater increases in VLDL particle size (p = 0.01), large LDL (p = 0.004), and HDL particle size (p = 0.05).

CONCLUSIONS:

The LCKD with nutritional supplementation led to beneficial changes in serum lipid subclasses during weight loss. While the LCKD did not lower

total LDL cholesterol, it did result in a shift from small, dense LDL to large, buoyant LDL, which could lower cardiovascular disease risk.

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