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Original

Evidence-based nutritional recommendations for the prevention and treatment of overweight and obesity in adults (FESNAD-SEEDO consensus document). The role of diet in obesity prevention (II/III)

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Abstract

This study is a consensus document of two Spanish scientific associations, FESNAD (Spanish Federation of Nutrition, Food and Dietetetic Associations) and SEEDO (Spanish Association for the Study of Obesity), about the role of the diet in the prevention and of overweight and obesity in adults. It is the result of a careful and systematic review of the data published in the medical literature from January 1st 1996 to January 31st 2011 concerning the role of the diet on obesity prevention.

The conclusions obtained have been classified according several evidence levels. Subsequently, in agreement with these evidence levels, different degree recommendations are established. These recommendations could be potentially useful to design food guides as part of strategies to prevent overweight and obesity.

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Key words: *Obesity. Overweight. Prevention. Diet. Nutrition.*

RECOMENDACIONES NUTRICIONALES BASADAS EN LA EVIDENCIA PARA LA PREVENCION Y EL TRATAMIENTO DEL SOBREPESO Y LA OBESIDAD EN ADULTOS (CONSENSO FESNAD-SEEDO). LA DIETA EN LA PREVENCIÓN DE LA OBESIDAD (II/III)

Resumen

Se presenta un consenso de la Federación Española de Sociedades de Nutrición, Alimentación y Dietética (FESNAD) y la Sociedad Española para el Estudio de la Obesidad (SEEDO) sobre la dieta en la prevención del sobrepeso y la obesidad, tras efectuar una revisión sistemática de los datos de la literatura médica desde el 1 de enero de 1996 al 31 de enero de 2011 .

Las conclusiones obtenidas se han catalogado según niveles de evidencia.

Se establecen unas recomendaciones clasificadas según grados que pueden servir de guía y orientación en el diseño de pautas alimentarias dirigidas a la prevención de la obesidad o el sobrepeso.

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Palabras clave: *Obesidad. Sobrepeso. Prevención. Dieta. Nutrición.*

Abbreviations

ALA: Alpha-linolenic acid.
BMI: Body mass index.
CI: Confidence interval.
DF: Dietary fibre.
DHA: Docosahexaenoic acid.
EFSA: European Food Safety Authority.
EPA: Eicosapentaenoic acid.
GI: Glycaemic index.
GL: Glycaemic load.
IOM: Institute of Medicine of the USA.
MedDiet: Mediterranean diet.
MUFA: Monounsaturated fatty acids.
OR: Odds Ratio.

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PUFA: Polyunsaturated fatty acids.
RCT: Randomised controlled trial.
RR: Relative risk.
SD: Standard deviation.
SFA: Saturated fatty acids.
TFA: Trans fatty acids.
WHO: World Health Organisation.

Introduction

In light of the high prevalence of obesity and overweight in our country[1] and the multitude of nutritional approaches proposed to combat them, the Spanish Federation of Nutrition, Food and Dietetetic Associations (FESNAD) and the Spanish Association for the Study of Obesity (SEEDO) have jointly proposed to clarify the role of the various nutritional factors for both the prevention and treatment of Obesity and Overweight. For this purpose a FESNAD-SEEDO consensus has been prepared, containing nutritional recommendations based on evidence which will serve as a tool to health professionals when designing prevention strategies or treatment guidelines for obesity or overweight.

It must be noted that the opinions expressed in this document have been agreed upon between the representatives of the different associations listed in the authorship and, as such, they represent the position of all of them.

The consensus is organised into 3 documents published separately. This work covers the review of the dietary aspects of the prevention of obesity and overweight.

Methodology levels of evidence

The methodology and working system of this consensus have already been described.[2] Briefly, we can say that for the design of the following recommendations we reviewed the scientific literature which covers the general areas of interest for the consensus, published between 1st January 1996 and 31st January 2011. On the basis of the conclusions obtained from that review, the evidence was classified and recommendations were formulated according to the method proposed in 2008 by the European Association for the Study of Obesity[3] and which consists of a simplified version of the system proposed by the Scottish Intercollegiate Guidelines Network (SING)[4] (tables I and II).

On the basis of the criteria for its preparation, the resulting document is applicable to the adult population (excluding pregnancy and breastfeeding) which, apart from obesity, presents no malnutrition or chronic diseases.

Preliminary analysis of the reviews and recommendations published

In order to obtain an overall perspective and define the key areas associated with the prevention of obesity in adults through diet, there has been a review of guidelines, consensuses, strategies, publications and relevant documentary sources on the issue before deciding on the questions to address in this document (see methodology section). Below there is a brief summary of the documents assessed in order to address the prevention of obesity in adulthood.

National documents

In the year 2005, the NAOS Strategy (Strategy for Nutrition, Physical Activity and the Prevention of Obesity) was started at the Ministry for Health and Consumer Affairs, through the Spanish Food Safety and Nutrition Agency (AESAN). Its objective was to raise awareness among the population of the problem that obesity represents to health and to promote an initiative to help promoting healthy life habits, chiefly through a healthy diet and regular physical activity.[5]

Table I
Levels of evidence[19]

Levels of evidence		
1	1++	High quality meta-analysis, systematic reviews of RCT's or RCT's with a very low risk of bias.
	1+	Meta-analysis well executed, systematic reviews of RCT's or RCT's with a low risk of bias.
	1-	Meta-analysis, systematic reviews of RCT's or RCT's with a high risk of bias.
2	2++	High quality systematic reviews of case-control or cohort studies.
	2+	High quality case-control or cohort studies with a very low risk of confusion or bias and a high probability that the relationship is causal.
	2-	Well executed case-control or cohort studies with a low risk of confusion or bias and a moderate probability that the relationship is causal.
3		Non-analytical studies (e.g. clinical cases, case series).
4		Opinion of expert(s).

Table II
Levels of recommendation[19]

	Levels of recommendation
A	At a minimum a meta-analysis, systematic review or RCT with a classification of 1++ and directly applicable to the target population, or a systematic review or RCT with a body of evidence consisting mainly of studies graded at 1+, directly applicable to the target population, and demonstrating overall consistency in its outcomes.
B	A body of evidence which includes studies graded at 2++, directly applicable to the target population and which demonstrates overall consistency in its outcomes, or evidence extrapolated from studies graded at 1++ or 1+.
C	A body of evidence which includes studies graded at 2+, directly applicable to the target population and which demonstrates overall consistency in its outcomes, or evidence extrapolated from studies graded at 2++.
D	Evidence of level 3 or 4, or evidence extrapolated from studies graded at 2+.

Studies classified as 1- and 2- must not be used in the process of preparing recommendations because of their high bias potential.

In 2007 the Spanish Society of Primary Care Physicians (SEMERGEN), the Spanish Association for the Study of Obesity (SEEDO) and the Spanish Society for Endocrinology and Nutrition (SEEN) published a strategy, in leaflet form, designed with the objective of preventing (but also diagnosing and treating) overweight and obesity in the general population.[6]

In 2007, Aranceta et al.[7] published a review in the magazine Public Health Nutrition with the objective of proposing the way to address the development of strategies for preventing obesity in Spain.

In 2008, Quiles et al.[8] described the leading strategic documents developed in different Spanish autonomous communities which contain courses of action (health policies) for the prevention of obesity.

International documents

In 2003, the World Health Organisation (WHO) published the book "Diet, nutrition and the prevention of chronic diseases", which contains ample information about the evidence available to date about the role of the diet in preventing unintentional weight gain.[9]

In 2004, the WHO approved the Global Strategy on Diet and Physical Activity, by which it urged the Member States to implement national action plans,[10] and which includes specific dietary-nutritional recommendations to prevent obesity, aimed both at populations and individual people.

In 2006, the National Institute for Health and Clinical Excellence (NICE) in Britain published an extensive document whose objectives included increasing the effectiveness of interventions to prevent overweight and obesity in the population.[11]

In 2007, the WHO published a document entitled "The challenge of obesity in the WHO European region and the strategies for response", which also detailed dietary-nutritional factors associated with the prevention of obesity.[12]

In 2008, the American Heart Association[13] published a comprehensive document which, among other factors, reviewed the diet related health patterns associated with the unintentional gain of body weight.

In 2009, the American Dietetic Association, in conjunction with the American College of Sports Medicine, published a position paper which included advice on the composition of the diet for the prevention of weight gain.[14]

In 2010, the Scottish Intercollegiate Guidelines Network published evidence-based guidelines which included recommendations for the prevention of obesity in children, young people and adults.[15]

Finally, in 2010, the Dietary Guidelines Advisory Committee of the United States, with the participation of the Cochrane Collaboration, published comprehensive information in relation to preventive dietary-nutritional aspects of obesity.[16]

Dietary factors associated with the prevention of obesity

1. Energy balance and body weight

1.1. Energy density

It appears that the application of thermodynamic principles to human physiology is beyond doubt, although the metabolic pathways and routes involved are more complex factors, we are still far from fully understanding. Even though we accept that under the conditions of a specific genotype, the excess or unused energy intake is stored in the form of fat, we would like to review the evidence which informs us of whether energy density is associated with weight changes. Energy density is defined as the quantity of energy available in food or drink, per unit weight.[17] In this sense, Cucó et al. observed in the Mediterranean population that the energy density of the diet is positively associated with a greater intake of energy, total fats and saturated fats, although it did not assess its effect on body weight.[18] Furthermore, a systematic review by Alinia et al.[19] showed that most of the available evidence indicates a possible inverse association between the consumption of fruit (which generally has low energy density) and overweigh, although this review did not focus on the effect of energy density on body weight.

Four cohort studies with a follow-up time ranging from six to eight months demonstrate a positive association between energy density and changes in weight.

Bes-Rastrollo et al.,[20] carried out a cohort study of 50,026 women between 1991 and 1999, concluding that the increase in dietary energy density was associated with greater weight gain in middle-aged women. The women in whom the greatest increase in energy density was observed during the follow-up period (highest quintile) gained significantly more weight than those whose diets had the lowest energy density (lowest quintile) (6.4 kg *vs.* 4.6 kg; P value for trend < 0.001).

Through a prospective cohort study of 168 non-institutionalised women, Savage et al.[21] observed, after a follow-up period of six years, that the women who had diets with the greatest energy density gained an average of 6.4 kg, while the women with diets with the lowest energy density (lowest tertile) had only gained 2.5 kg, this difference had been statistically significant.

In a Randomised Controlled Trial (RCT) Westerterp-Plantenga el al.[22] assessed the effect of energy density on 220 healthy volunteers who were monitored for 6 months. The individuals were divided into two groups according to whether or not they presented the profile of "dietary restriction" (a tendency to consciously limit the type and quantity of food consumed in order to lose or maintain weight). Additionally, each of the groups was urged to (randomly) consume commercial products with or without fat. The group of individuals with a "dietary restriction" profile gained an average of 0.2 kg if they consumed products with fat, but they lost 1.5 kg if they consumed products without fat. On the contrary, the group of individuals without a "dietary restriction" profile lost an average of 0.2 kg if they consumed products without fat, but they gained 1.8 kg if they consumed products with fat. In spite of these differences only showed statistical significance in the group of individuals with the "dietary restriction" profile who consumed products without fat. It was concluded that less energy density from fat can help to maintain body weight, irrespective of the "dietary restriction" profile of the individuals.

Through an RCT carried out with 810 hypertensive or pre-hypertensive adults from the PREMIER study, Ledikwe et al.[23] showed that large or small changes in dietary energy density for six months are associated with weight loss. The analyses were carried out according to tertiles of energy density reduction. Those who were in the highest tertile for the reduction of energy density lost an average of 5.9 kg, those in the middle tertile lost 4 kg and those in the lowest tertile for the reduction of density energy lost 2.4 kg.

Evidence

1. Dietary patterns of high energy density may lead to body weight increase in adults (Evidence Level 1+).

Recommendations

1. Body weight increase may be prevented through the use of diets containing lower energy density food (Recommendation Degree A).

1.2. Energy balance and obesogenic environment

Reviewing the environmental aspects of obesity is clearly relevant, especially because environments which induce and stimulate obesity (obesogenic environments) have been detected.

Food environments refer to the availability of food and they are associated with dietary intake, more specifically with a lower consumption of fruit and vegetables. The presence of supermarkets and other places which make fruit and vegetables available in the neighbourhood is associated with a lower average BMI in the population in comparison with those lacking them or if they are located at great distances, above all for disadvantaged socioeconomic groups. The increase in the number of "fast food" restaurants and convenience stores in a geographical unit has also been associated with a higher average BMI in the population.

These claims are made on the basis of 9 systematic reviews which have studied the relationship between the environment and body weight, the energy intake and the consumption of fruit and vegetables; although in their conclusions they establish that it is necessary to perform further research to have a greater knowledge and understanding of these relationships. Six studies 24-29 found that the neighbourhoods with socioeconomic problems (unemployment, low incomes and education standards) were associated with obesity and with a poorer dietary intake. Eight studies found that the availability of healthy food, either directly or through the absence of supermarkets or the distance at which they are located, is associated with body weight and dietary intake (fruit and vegetables).[26,27,30-35] Two studies found that a high density of fast food restaurants and convenience stores was associated with a high prevalence of obesity.

Evidence

2. The absence of supermarkets with fruit and vegetables availability, or their sitting at great distances –in particular from human settlements with low socioeconomic levels– are conditioning factors for a higher population mean Body Mass Index (BMI) (Evidence Level 1+).

Recommendations

2. Strategies should be implemented which render possible food availability and access to healthy food, particularly fruit and vegetables, so as to generate favourable environments for maintaining a healthy population mean BMI (Recommendation Degree A).

1.3. Energy balance: eating outside home

People's current lifestyles have led to an increase in the number of times in which food is consumed outside of the home, and in the variety of food and snacks consumed on those occasions. This trend shows no sign of slowing in the future. That, combined with the possibility of this energy intake not showing a healthy eating pattern, could have implications for controlling body weight. The EPIC prospective study (European Prospective Investigation Into Cancer and Nutrition)[36] observed that the percentage of daily calories taken in outside of the home in the Spanish autonomous communities being studied (Granada, Murcia, Navarra, San Sebastian and Asturias) ranged from 20% to 23.9%. Women's consumption of fat outside the home was greater, and an increase in the consumption of sugar and a decrease in the intake of fibre were observed in both sexes. Nevertheless, this study did not assess changes in body weight associated with food intake outside of the home.

A recent systematic review carried out by Rosenheck[37] examined the association between eating in fast food restaurants and weight gain and obesity. The review, which included 16 studies (six transversal, seven prospective cohort and three experimental), enabled the author to conclude that, on the one hand, there is consistent evidence which demonstrates that this type of restaurant plays a separate role which leads to an increase in energy intake, thus accelerating the rates of weight gain and obesity. On the other hand, there is sufficient evidence for the public health authorities to make a recommendation to limit the consumption of fast food to reduce weight gain.

Furthermore, six prospective cohort studies[38-43] found a positive and significant association between the consumption of fast food and body weight in adults, although in one of them the positive association was only observed in women.[40] According to the study by Pereira et al.,[43] the consumption of fast food more than once a week is associated with increases in BMI. Duffey et al. did not find evidence of changes to the BMI in relation to the consumption of food in other types of restaurants during a follow-up of three years.[38] However, Bes-Rastrollo et al.[44] concluded, after monitoring a cohort of 9,182 Spanish graduates for an average of 4.4 years, who declared in an initial survey that they ate outside of the home twice or more every week, that after the follow-up they presented a moderate increase in body weight (+129 g/year, p value < 0.001) and a greater risk of gaining more than 2 kg per year (OR = 1.36; CI 95% 1.13; 1.63). Eating outside of the home is significantly associated with a higher risk of ending up suffering from overweight or obesity (RR = 1.33; CI 95% 1.13, 1.57). Even the fact that the survey was not repeated together with the evaluation of the changes in body weight makes it possible that the habits of the volunteers had changed over the years.

Evidence

3. The habitual intake of "fast food" (over once a week) might contribute to increased energy intake and to weight increase and obesity (Evidence Level 1+).

Recommendations

3. Restricting the habitual (more than once a week) intake of "fast food" might prevent weight increase due to this factor (Recommendation Degree A).

1.4. Energy balance: size of rations

There is data indicating that larger rations can make it difficult to self-regulate intake.[45] In this sense, controlled trials have been published in which it is observed that a larger size in the ration offered is associated with a significantly higher intake of food, without the feeling of fullness being affected any more than when a smaller ration is offered.[46,47] A review of experiments carried out both inside and outside of laboratories, illustrated that they unanimously demonstrated that an increase in the size of the ration was associated with the subjects having a higher energy intake.

In 2005, the Dietary Guidelines Advisory Committee of the United States[49] reviewed the evidence of the effect of the size of the ration on energy intake, concluding that it influenced the amount that people consume. Generally speaking, there was a higher energy intake when larger rations were served than when smaller rations were served.

Gilhooly C et al.[50] carried out an RCT in which they examined the characteristics of snack food in relation to dietary energy restriction and weight. The trial was carried out with 32 women for a period of 6 months. The results showed that there was a statistically significant positive relationship between the size of the rations and the habitual BMI ($r = 0.49$, $p = 0.005$). The regression analyses showed that the subjects reporting the highest percentage of weight loss were those who snacked least (adjusted $R2 = 0.31$, $p = 0.009$).

Evidence

4. Offering larger portions conditions an increase of the individual's caloric intake (Evidence Level 2++).

Recommendations

4. The use of smaller portions limits the energy intake (Recommendation Degree B).

1.5. Energy balance: breakfast

The role of breakfast in the risk of obesity in adults is disputed and cause for debate.[51] Two transversal analyses of energy intake showed that skipping breakfast[68] or consuming fewer calories during breakfast[51] is associated with a substantially lower total energy intake over the course of the day. However, the analyses of intra-individual eating habits have demonstrated that an increase in the calorie intake of breakfast to the total energy intake is associated with a lower energy intake over the course of the day.[53,54] An analysis of 2,959 subjects after maintaining an average weight loss of 32 kg over 6 years, showed that most of them (78%) normally had breakfast, although it was also observed that, on the one hand, the remaining 22% (who regularly skipped breakfast) also maintained weight loss, and on the other hand, that the subjects who regularly had breakfast reported carrying out more physical exercise, facts which limit the causality of the relationship between breakfast and controlling body weight.[55]

It would be risky to draw conclusions from these studies, due to the difference in their methodological approaches, and because they were not designed to assess changes in body weight in relation to breakfast.

Six prospective cohort studies which have evaluated the relationship between breakfast and body weight have been identified. Three studies found an inverse relationship between eating breakfast and adult weight gain.[42,56,57] Niemeier et al. and Merten et al.[42,56] observed an inverse relationship between breakfast in adolescents (12-19 years of age) and the risk of obesity years later (18-26 years). In turn, Purslow et al.[57] monitored 6,764 men and women aged between 43 and 75 between the years 1993-1997 and 1998-2000. They observed that the individuals in the lowest quintile of breakfast intake (lowest percentage of daily energy provided by breakfast) gained 1.23 kg (SD: 0.12) while those in the highest quintile gained 0.79 kg (SD: 0.11). This relationship remained significant after making adjustments for sex, age and other confounding factors. However, despite evaluating the dietary intake at the start of the research, the study did not do so during follow-up. Therefore, the lower weight gain in the volunteers who initially had a higher percentage of daily energy intake from breakfast could be due to changes in habits (e.g.: a reduction in the total energy intake). The study by Nooyens[58] initially found an inverse relationship, but after making adjustments for potential confounding factors, the association was not statistically significant. Another study observed this inverse relationship between the consumption of breakfast during adolescence (average age: 15.28) and the gain in body weight six years later among men, but it did not find an association among women.[75] Van der Heijden A et al.[60] carried out a cohort study on 20,064 men aged between 46 and 81 (3,386 did not eat breakfast and 16,678 regularly ate breakfast) to research the association between consuming breakfast and gaining weight in the long term (10 years). A slightly lower weight gain was observed in men who had breakfast than those that didn't, but without statistical significance (1.55 ± 0.05 *vs.* 1.67 ± 0.11 kg, $p = 0.35$). However, they showed that the consumption of breakfast was inversely associated with the risk of a 5 kg weight gain after making adjustments for age, lifestyles and the initial BMI. The association was more pronounced among men with a BMI ≤ 25 kg/m^2 than with those who were overweight, although this association was weakened when adjustments were made for potential confounding factors. The authors concluded that having breakfast could have a moderate impact on preventing weight gain among middle-aged men.

Evidence

5. Research results on the relationship between the omission of breakfast and the risk of overweight and obesity in adults are both controversial and inconsistent.

1.6. Energy balance: snacks

Eating less "refreshments" or snacks is a practice which appears to be becoming increasingly common.[61] It is therefore important to assess their role in weight gain, particularly given the current debate in this respect in the scientific community. However, it is a practice which is difficult to evaluate by analysing relevant publications because of the variety of approaches taken by different authors towards eating snacks. The lack of a globally accepted definition of the term "snack" in scientific literature complicates the interpretation of the studies.[61] Therefore, for the purposes of this analysis, the term "snack" and the words originating from it (snacks, snacking, snacker, etc.) have been accepted as search criteria, in line with the proposal of the Dietary Guidelines Advisory Committee of the United States.[17]

A transversal analysis carried out with 2,437 European volunteers between the ages of 28 and 70 observed that those who snacked between meals were more likely to be obese (OR = 1.24).[62] Other similar studies, such as that carried out by Sánchez Villegas et al.[63] or by Marín-Guerrero et al.[64] have observed this association in the Spanish population. However, the transversal design of all of these studies makes it impossible to conclude if there is causality.

Three studies have been found which have assessed the relationship between snacks and weight gain. A study carried out in Denmark[65] on a population aged between 50 and 64 observed that diets with high snack content were associated with an increase in waist circumference after five years. It is important to stress that the authors of that study only considered the following food to be snacks: chocolates, sweets, liquorice, fruit flavoured chewing gum, toffees, pork scratchings and crisps. Research carried out in Hong Kong[66] observed that a greater variety in the consumption of snacks was associated with an increase in the risk of reaching a BMI of 23 kg/m^2 after a follow-up of between five and nine years, but not with a risk of reaching a BMI greater than 25 kg/m^2 (overweight). Finally, Bes-Rastrollo et al.67 specifically assessed the relationship between snacks and weight gain in a cohort of 10,162 university graduates from Spain (average age: 39) monitored for an average of 4.6 years. Those subjects who regularly snacked were identified (those who replied in the affirmative when asked if they regularly ate between meals). The subjects who snacked regularly gained more weight than those that did not (188 grammes/year compared with 131 grammes per year, $p < 0.01$) after adjustments were made for confounding factors, although these differences in weight are too small to be clinically important in the medium term. Despite the fact that the survey was not repeated when assessing the changes in weight after those years had passed (to assess whether the habits in this respect had also changed) and the observational design of the study mean that it is possible that there are residual confounding factors, thus limiting the extrapolation of causal inferences.

EVIDENCE

6. Research results on the relationship between snack intake and the risk of weight gain are both controversial and inconsistent.

1.7. Energy balance: frequency

The effect of intake frequency on the metabolism has been matter of active study for over 40 years.[68] It is habitual to find claims, by health organisations or reference books, that regularly eating small quantities of food avoids weight gain. Despite this, there are conflicting positions in this respect.[69] The intake frequency could play a role in regulating energy intake and controlling body weight, but it may also result in an increased energy intake. This eating pattern has been associated with benefits to controlling the appetite[70] or increases in the thermogenic effect of food,[71] but it has also been associated with a smaller[72,73] and greater[74-76] risk of obesity. One study found a significantly lower risk of obesity (45%) in individuals with 4 or more intakes per day in comparison with those with 3 or less.[77] Conversely, another study based on a representative sample of the population of the United States found that the BMI increased when the intake frequency increased.[75] We have even found studies which have concluded that intake frequency has no effect on BMI.[78]

Although all of these studies are transversal (some with methodological limitations) and therefore make it impossible to establish causal relationships, they raise doubts about promoting frequent food intake to control body weight.

The cohort study by van der Heijden et al.,[60] to which we referred in the section about breakfast, researched the association between eating patterns and long-term weight gain in men (10 years) in the USA. It was observed that an increase of at least two eating occasions, in addition to the three standard meals, was associated with a greater risk of gaining 5 kg in weight after 10 years (RR: 1.15 (CI 95%, 1.06 to 1.25, for ≥ 2 *vs.* 0 additional eating occasions).

EVIDENCE

7. Research results on the relationship between food intake frequency (number of meals per day) and body weight variation are inconsistent.

2. *Eating patterns and body weight*

2.1. Mediterranean diet

The Mediterranean diet (MedDiet) is characterised by an abundance of food of plant origin, hardly processed and seasonal, preferably fresh; fresh fruit as a typical daily dessert; the consumption of occasional sweets; olive oil as the main source of fat; a low or moderate consumption of dairy products (mainly cheese and yoghurt), and of fish and poultry; weekly consumption of eggs; red meat in small quantities; and a low or moderate consumption of wine, normally during meals.[79-81] The term MedDiet reflects the characteristic eating patterns of several countries of the Mediterranean Basin at the beginning of the 1960's, and it originates from the research coordinated by Dr Ancel Keys.[80,81] Varela-Moreiras et al.[82] have recently concluded that the current diet of Spaniards is markedly different to that of 40 years ago, although Spain is paradoxically a leading producer and exporter of staple foods in the MedDiet, it is markedly deviating from following the MedDiet pattern because of considerable social and economic changes.

The MedDiet can reduce the risk of mortality and provide significant protection against the incidence of the main chronic diseases,[83-85] although, as detailed by Martínez-González et al.,[85-87] and Ballisle,[88] the epidemiological studies available do not make it possible to conclude with certainty that all components of the MedDiet are protectors or if they show the same level of protection, and it is plausible that the overall intake pattern, or other factors associated with the MedDiet in relation to lifestyle, are responsible for some of the benefits observed.

There are doubts about promoting the MedDiet because of concerns that it can result in increases in the BMI because of its high fat content (mainly monounsaturated).[89] However, numerous studies show an inverse association between adherence to the MedDiet and both the BMI and obesity in adults,[90-99] although the transversal nature of the design of these studies makes it impossible to infer causality.

Various non-transversal studies have reviewed the role of the MedDiet in relation to body weight in healthy adults between 1996 and 2011.

In 2004, a randomised crossover trial with 22 healthy adults assessed the effect of a dietary pattern inspired by the MedDiet or the typical Swiss diet on the lipid profile for 4 weeks. Although its aim was to keep the weight of the volunteers constant, a small but significant decrease in their BMI was observed.[100] Nevertheless, this is a short-term study with a very small sample of the population. Furthermore, both the MedDiet considered (which in many ways did not coincide with the description at the beginning of this section) and the difference in energy between this diet and the typical Swiss diet (the Swiss diet provided a further 221 kcal per day) limit the validity of the relationship observed between the diet and body weight.

One of the first prospective cohort studies found, focusing on the effect of adhering to the MedDiet on body weight or changes to the BMI, is that of Sánchez-Villegas et al., published in 2006.[101] A follow-up was carried out with 6,319 Spanish university graduates (University of Navarra Follow-up-SUN-) for 28 months, who were stratified according to their adherence to the MedDiet at the start of the study. Diet changes were also assessed during the follow-up. Although the average weight of the participants increased during the follow-up period, a lower adherence to the MedDiet at the start of the study was associated with a greater weight gain (0.73 kg) in comparison with a greater adherence to the MedDiet (0.45 kg). The results indicate an inverse dose-dependent relationship (p-trend = 0.016). A similar inverse association was observed when assessing the changes which have taken place in the diet during the follow-up period. However, none association was statistically significant once adjustments had been made for important confounding factors.

In the same year, Méndez et al.[102] published a study based on data from the Spanish cohort of the EPIC study (European Prospective Investigation into Cancer and Nutrition). There was an assessment of whether the MedDiet was associated with the incidence of obesity after 3 years of monitoring a sample of 17,238 women and 10,589 men without obesity, between the ages of 29 and 65. The data relating to food intake were compiled by dieticians-nutritionists at the beginning of the study. High adherence to the MedDiet was not associated with an increased incidence of overweight or obesity in subjects with a normal weight at the beginning of the study, a fact which remained unchanged after adjustments were made for potential confounding factors. Even though the fact that the initial dietary survey was not repeated together with the assessment of changes in body weight makes it possible that the habits of the volunteers had changed over the years.

A year later, Tortosa et al.[103] published a follow-up of 5,360 volunteers included in the SUN cohort (University of Navarra Follow-Up). After 6 years of follow-up, it was observed that the abdominal circumference of the volunteers with the greatest adherence to the MedDiet (82 ± 12 cm) was smaller than that of those with less adherence to the MedDiet (82.5 ± 12 cm) (p = 0.038, after adjustments for age and gender). These outcomes were unchanged after adjustments were made for lifestyle and other variables. The authors pointed out that it was unlikely that this effect could be explained by residual confounding factors.

In 2009, Yannakoulia et al.[104] did not find a significant association between the MedDiet and the incidence of overweight or obesity after evaluating 1,528 women and 1,514 men in Greece (the ATTICA study) via a follow-up lasting for 5 years, after performing a multivariate analysis.

In 2009, Rumawas et al.[105] examined the longitudinal association between the MedDiet and the abdominal perimeter in 2,720 volunteers of the Framingham Heart Study Offspring cohort, monitored for an average of 7 years. Greater adherence was associated with a smaller abdominal perimeter ($p < 0.001$), after adjustments were made for potential confounding factors. Despite the fact that the initial dietary survey was not repeated together with the assessment of changes in body weight makes it possible that the habits of the volunteers had changed over the years.

In 2010, Romaguera et al.,[106] published the result of a 5 year follow-up of the EPIC-PANACEA cohort (European Prospective Investigation into Cancer and Nutrition–Physical Activity, Nutrition, Alcohol Consumption, Cessation of Smoking, Eating Out of Home, and Obesity) which included 270,384 women and 373,803 men aged between 25 and 70. The individuals with the greatest adherence to the MedDiet presented a weight change at 5 years of -0.16 kg (CI 95%: -0.24, -0.07 kg) and they were 10% (CI 95%: 4%, 18%) less likely to develop overweight or obesity than those individuals with the less adherence to the MedDiet. The authors took into account various potential confounding factors. The low meat content of the MedDiet appears to be mainly responsible for these beneficial effects in relation to weight gain. It must once again be taken into account that the fact that the initial dietary survey was not repeated together with the assessment of changes in body weight makes it possible that the habits of the volunteers had changed over the years.

As detailed by Romaguera et al.[106] the differences observed in the aforementioned studies could be attributed to the use of different markers to define the MedDiet, to the use of different confounding factors in the statistical models, to the use of underestimations, the size of the sample, or the lack of homogenisation in the dietary pattern of the volunteers.

A systematic review published in 2008 by Buckland et al.[106] concluded that the studies assessing the relationship between the MedDiet and body weight showed inconsistent results, but that they pointed towards a possible role for the MedDiet in the prevention of overweight and obesity. Another, more recent, systematic review of the literature108, concluded that, despite not all of the studies showing a protective effect, the evidence as a whole suggested a possible beneficial effect of the MedDiet for the BMI and obesity.

Evidence

8. Even though inconsistent results do exist, the studies so far performed suggest a possible role of the "Mediterranean" diet in the prevention of overweight and obesity (Evidence Level 2–).

9. The existing evidence suggests that greater adherence to the "Mediterranean" diet might prevent abdominal perimeter increase (Evidence Level 2+).

Recommendations

5. A greater adherence to the "Mediterranean" diet might prevent overweight and obesity and also the increase of the abdominal perimeter (Recommendation Degree C).

2.2. Vegetarian diets

Both the American Dietetic Association[109] and the Canadian Dietetic Association[110] indicate that vegetarians tend to present a lower BMI than omnivores. A review by Berkow et al.[111] pointed out that the observational studies indicated that the weight and BMI of vegetarians is approximately 3-20% lower than those of non-vegetarians, and that while the prevalence figures of obesity range from 0 to 6% in vegetarians, for non-vegetarians they range from 5 to 45%. In turn, the Dietary Guidelines Advisory Committee of the United States[17] indicates that the vegetarian group presents less prevalence of obesity, and suggests that it is possible that one cause of this is the different nutritional profile of their diet, which usually has a lower energy intake, with a lower proportional energy intake from fats, and a higher dietary fibre content in the diet. However, it is possible that the different lifestyle associated with the vegetarian diet is partly responsible for a lower average BMI in those who follow this eating pattern.

In 1998, Appleby et al.[112] carried out a transversal study, with 3,378 women and 1914 men, non-smokers, aged between 20 and 89, to examine the association between vegetarian and omnivore diets and the BMI, using data from the Oxford Vegetarian Study cohort. It was observed that the BMI of vegetarians was lower than that of non-vegetarians (0.99 kg/m^2 in women and 1.13 kg/m^2 in men). After adjustments were made for various confounding factors, these differences were reduced, but they continued to be statistically significant.

In the year 2011, Kennedy et al.[113] examined the effect of the vegetarian diet on the BMI of 10,014 healthy volunteers above the age of 19. After dividing the participants into vegetarians and non-vegetarians, it was observed that the BMI of the vegetarians was significantly lower than that of the non-vegetarians. However, this study did not assess the potential confounding effects (e.g.: regular physical exercise).

Spencer et al.,[114] performed a transversal study to establish the differences in the BMI of the participants of the Oxford cohort of the European Prospective Investigation into Cancer and Nutrition (EPIC-Oxford). The analysis included 37,875 participants, aged between 20 and 97, split into four groups according to their dietary characteristics: Meat eaters, fish eaters (but not meat), lacto-ovo-vegetarians and vegans (who did not consume animal products). The meat eaters presented the highest intake of energy, proteins, total fats, saturated fats and monounsaturated fats. In contrast, the vegans presented the highest intake of fibre and polyunsaturated fats. The average age-adjusted BMI was statistically different for each of the four groups: higher levels for meat eaters (24.41 kg/m^2 in men, 23.52 kg/m^2 in women) and lower average levels in vegan groups (22.5 kg/m^2 in men, 21.98 kg/m^2 in women). The other two groups (fish eaters and lacto-ovo-vegetarians) had intermediate levels which were similar to one another. The prevalence of obesity was significantly lower in vegans, and between vegetarians and the group of fish eaters it was lower than observed in meat eaters. These differences remained unchanged after adjustments were made for various factors. The dietary factors with the greatest importance in relation to BMI included a high percentage of protein intake and a low fibre intake, both between the dietary groups and within each group. The authors conclude that vegan diets and, to a lesser extent, lacto-ovo-vegetarian diets and the diets of people whose only source of animal protein is fish, are associated with a lower BMI and a lower frequency of obesity than diets which include meat.

Similar outcomes were observed in the Swedish Mammography Cohort, studied by Newby et al.[115] This transversal study was designed to assess the association between BMI and the risk of overweight and obesity of the different self-declared eating patterns of 55,459 women: omnivores (they consumed all foods); semi-vegetarians (mostly lacto vegetarians with some fish and eggs); lacto vegetarians (without meat, poultry, fish or eggs) and vegans (no meat, poultry, fish, eggs or dairy products). The group of "omnivores" had a significantly higher intake of protein, with more saturated and monounsaturated fats and significantly lower intake in carbohydrates and fibre than the other "vegetarian" groups. The group of "omnivores" presented the highest intake of refined grains and animal products and the lowest of fruit and vegetable. The prevalence of overweight (BMI ≥ 25 kg/m^2) was 40% among omnivores, 29% among semi-vegetarians and vegans and 25% among lacto vegetarians. In the multivariate linear regression analysis, the women who were not "omnivores" had a significantly lower BMI than the "omnivores". In the logistic regression analysis, the three "vegetarian" groups presented a lower risk of overweight and obesity than the "omnivore" group, which was statistically significant.

Rosell et al.[116] designed a prospective cohort study to assess weight changes at five years among the participants of the EPIC-Oxford. The 21,996 healthy adults were divided into six groups, according to their intake: meat eaters (at the beginning and end of the study); fish eaters (they did not eat meat at the neither at the beginning nor the end of the study); vegetarians (they did not eat meat or fish but they did eat dairy products or eggs at the beginning and end of the study); vegans (they did not eat animal products at either of the two moments); "reverted" (they changed their diet in the direction from vegans to vegetarians to fish eaters to meat eaters) and "converted" (those who changed their diet in the opposite direction). The following differences were observed between the dietary groups. A (significantly) lower weight gain was observed among men and women who were vegans and fish eaters than among meat eaters. The highest weight gain observed was in meat eaters who had not altered their eating pattern. Of those who modified their diet in the 5 year period, the smallest increase in weight was observed in the "converted" group, and the largest in the "reverted" group; however, the average weight gain was not significantly higher in this group than that observed among meat eaters.

Evidence

10. Vegetarian diets are associated, in healthy adults, to a lower Body Mass Index (Evidence Level 2+).

Recommendations

6. Vegetarian diets intake might lead to a smaller weight gain over time in healthy adults (Recommendation Degree C).

3. Nutrients and body weight

The role of the composition of the diet for controlling weight and obesity is debatable. All macronutrients are capable of providing energy and, therefore, of contributing towards the total daily calorie intake, potentially producing a positive energy balance. However, several factors (e.g.: their metabolic utilisation) affect their capacity to produce that positive balance.[117] So, one of the main questions we can ask ourselves is, do the different relative contributions of macronutrients to the total intake have an effect on weight gain?

The importance of this question lies in the fact that, if the energy intake provided by one macronutrient produces a different positive energy balance to that of another macronutrient, this could lead to a recommendation for a specific nutritional composition to prevent weight gain in a person.

3.1. Carbohydrates and body weight

The Dietary Guidelines Advisory Committee of the United States, with the assistance of the Cochrane Collaboration, has recently indicated that healthy diets are rich in carbohydrates and it urges the population to change its current eating patterns towards a diet which based more on food of plant origin, with emphasis on the consumption of vegetables, pulses, whole grains, nuts and seeds.[16] The current intake of carbohydrates in Spain is around 41%82,[118] of the diet's energy, in other words, below the recommendations established by the European Food Safety Authority (45-60%)[119] and the World Health Organisation (55-75%).[9]

However, the role of carbohydrates in controlling body weight is currently a clear cause for scientific debate.[120-122] Carbohydrates are macronutrients which provide energy and which theoretically contribute to excessive weight gain. Despite this, there is no clear evidence showing that the total proportion of carbohydrates in a diet is an important determinant of energy intake.[123]

Carbohydrates contained in diets (with the exception of total sugars) tend to have a modest inverse association with energy density. However, the fat content is generally directly associated with diets with high energy density.[20,124,125]

In 2006, an RCT which assessed the effects of diet composition and the energy balance on predicting changes in the body composition, estimated that subjects who consume a high percentage of carbohydrates (55%) gained less fat mass, percentage of body fat and weight when compared with isocaloric diets with a high fat content (50%). However, on making adjustments for insulin sensitivity, only the predictive factors of changes in fat mass and percentage of body fat remained.

The majority of epidemiological studies show an inverse association between the consumption of carbohydrates and BMI.

In 2005, Ma et al.[127] researched the relationship between the BMI and the dietary intake of carbohydrates in 572 healthy adults who were monitored for 1 year. After making adjustments for possible confounding factors, it was observed that the BMI was not related to the intake of carbohydrates.

A review carried out in 2007[128] analysed 4 studies with the participation of men and women[129-132] and 3 only with women.[133-135] In all of them, the average BMI of the groups which consumed the most carbohydrates was greater than those which consumed less. The methodological problems which limit the capacity to establish causality in the transversal studies reviewed must be noted. Furthermore, given that the high consumption of carbohydrates tends to be associated with a high intake of dietary fibre, it is difficult to attribute this effect solely to its intake.[133,136,137]

In 2009, Ahluwalia et al.[152] observed, in a sample of 966 French middle-aged men, that the intake of carbohydrates was consistently inversely associated with the BMI and the waist circumference, after adjustments were made for numerous possible confounding factors.

In 2009, Merchant et al.[139] assessed the dietary habits of healthy Canadian adults with an optimal BMI belonging to a community where the prevalence of obesity is high. The study included 4,451 volunteers participating in The Canadian Community Health Survey. After adjustments were made for numerous possible confounding factors, it was observed that the risk of obesity was inversely associated with the intake of carbohydrates. The lowest risk was observed for intakes of 290-310 grammes of carbohydrate/day. An intake of carbohydrates below 47% of the total energy intake was associated with a greater risk of suffering from overweight or obesity, and a lower risk for intakes between 47-64%.

In 2010, the European Food Safety Authority indicated, after analysing several studies of long-term intervention, that dietary changes which promote a higher intake of carbohydrates (> 50% of energy) "ad libitum" have been associated with a lower risk of weight gain in several population groups, including subjects with normal weight, overweight and obesity.[119]

Evidence

11. Diets with higher content of complex carbohydrates (approximately ≥ 50% of the total energy intake) are associated to a lower Body Mass Index in healthy adults (Evidence Level 2+).

Recommendations

7. Diets for healthy adults aiming to prevent weight gain should contain a considerable proportion (approximately ≥ 50% of the total energy intake) of complex carbohydrates (Recommendation Degree C).

Liquid carbohydrates vs. solid carbohydrates

No associations have been observed between the form of a food, energy intake and body weight. The 2010 review of the DGAC included 12 studies which did not present consistent experimental designs.[16] One study (PREMIER trial) compared the energy provided by liquids with that of solids140, where a reduction of 100 kcal per day in energy intake from liquids was associated with a weight loss of 250 g at six and eighteen months. In comparison, a 100 kcal reduction in energy intake per day with solid foods was only 100 g for the same periods of time. The difference was only statistically significant at six months. A dose-response trend between changes in body weight and energy intake with drinks was observed at 6 and 8 months.

Six transversal studies researched the impact of an energy supplement with replacement products before breakfast, dinner or before the "ad libitum" consumption of a meal.

The study by Almiron-Roig et al.,[141] compared the impact on energy intake of cola replacement products or fat-free biscuits consumed two hours or twenty minutes prior to the eating occasion. The food format (liquid or solid) had no significantly different impact on energy intake.

Tsuchiya et al.,[142] compared the satiating power of liquid and semi-solid yoghurt with fruit drinks and dairy fruit drinks. The authors concluded that neither the hungriest nor the most satisfied subjects presented energy compensation during the following meal after consuming yoghurt.

Mourao et al. researched the independent effect of the form of food and energy intake in obese and slim adults with food rich in carbohydrates (melon and melon juice), fat (coconut and coconut milk) and proteins (cheese and milk). The inclusion of energy drinks in a meal led to a greater energy intake than consuming the solid version of the same food.

Stull et al.[144] concluded that the response to food replacement products in liquid or solid form has no comparable influence on the appetite or on the eating behaviour response. The participants of their study consumed more calories from farinaceous products after ingesting liquid replacement products than after consuming solids.

Flood-Obbagy and Rolls[145] carried out a randomised transversal trial in the USA, in which they examined how different physical forms of apple (solid pieces or juice) affected the appetite, satiety and the energy intake of a meal. The authors concluded that consuming fruit before a meal can achieve satiety and reduce the subsequent intake of foods, leading to a substantial reduction in the total energy intake of the meal. Furthermore, the energy content of the apple juice, with or without fibre, was compensated by a reduction in the subsequent intake. Furthermore, the apple juice as a supplement did not increase the total energy intake of the meal either.

Through a transversal trial in the USA, Mattes and Campbell,[146] assessed the form in which the food was presented (solid —apple—, semi-solid —apple puree— or liquid —apple juice—) and the eating occasion (as a dessert after meals, or between meals) on the appetite and the daily energy intake in 40 individuals (20 adults with normal weight and 20 with obesity). Although the appetite responded in different ways to the different forms of presentation of the food, these effects did not result in differences in the daily energy intake.

Furthermore, Anne Moorhead et al.,[147] performed a randomised transversal trial in the United Kingdom which assessed the effects of the fibre content and of the structure of the fibre in carrots (whole, blended or its nutrients in a sauce) on postprandial satiety and the subsequent food intake. Significant differences were observed in energy intake for the three forms of presentation. The energy intake was lower when consuming whole and blended carrots than the carrot nutrients. When it was consumed as part of a mixed meal it significantly increase satiety and decreased subsequent intake.

During their study (8 week crossover trial) carried out with 15 volunteers, DiMeglio and Mattes[148] concluded that carbohydrate drinks promote a positive energy balance, while the solid form of the food produces precise dietary compensation.

In a 5 week crossover trial in 2007, Flood and Rolls[149] examined the effects of consuming different forms of soups with low energy density on the total energy intake of the meal for 60 healthy volunteers. Those who consumed soup significantly reduced the total energy intake for the meal, in comparison with those who did not consume soup. There was a reduction of approximately 20% in the meal's energy intake for those who consumed soup.

Evidence

12. The existing evidence regarding the relationship between the physical characteristics of carbohydrates (liquid or solid), the energy intake and the body weight are controversial.

Glycaemic index (GI) or glycaemic load (GL)

The glycaemic index is a system for quantifying the glycaemic response of a food which contains the same amount of carbohydrates as a reference food.[150] The glycaemic load is a product of the GI and the quantity of carbohydrates consumed, and it provides an indication of the quantity of glucose available to metabolise or store after consuming a food which contains carbohydrates.[151]

Both the GI and the GL of the Spanish diet are at the lowest levels in Europe. The average GI of the Spanish diet ranges from 52.2 to 54.8 in women and 53.6 and 56.6 in men. While the GL was estimated to be between 96.7 and 108.5 in women and 117 and 144.1 in men.[152]

Although it has been suggested to use this for the selection of foods which help to improve the nutritional profile of the diet, within the framework of the scientific update sponsored by the FAO-WHO, Venn and Green concluded in 2007 that one must maintain a cautious attitude when choosing food solely on the basis of the GI or GL, as those foods could also present high energy density or contain substantial quantities of sugars or saturated fatty acids.[151]

Currently, there is a debate as to the role of the GI and the GL in the control of body weight.[123]

In a transversal study carried out in Denmark, Lau et al.[153] examined the associations between the glycaemic index, glycaemic load and the BMI in 6,334 adults (average BMI: 26.2 kg/m^2) from the Inter99 study. After making adjustments for energy intake, both the index and load were positively associated with the BMI ($p = 0.017$ and $p < 0.001$, respectively).

A transversal study carried out in the United Kingdom which was published by Milton et al.,[154] studied whether a low glycaemic index was associated with a lower body weight or BMI in 1,152 adults above the age of 65 from the National Diet and Nutrition Survey. No significant associations were found for the GI and body weight or the BMI. The authors concluded that this study did not support the advice of consuming food with a low GI to prevent weight gain in old age.

A transversal study of young Japanese women (aged 18-20)[155] showed an independent positive correlation between the glycaemic index and load and the BMI after adjustments for various confounding factors.

A transversal study carried out in Spain by Méndez et al.,[156] examined the association between the glycaemic index and load and the BMI in a Mediterranean population (7,670 adults aged between 35 and 74). The authors concluded that their study did not support the hypothesis that there is a positive relationship between the GI, GL and obesity, but rather that, in a Mediterranean food culture, a diet characterised by a high GL can be associated with a low BMI.

Hare-Bruun et al.,[157] published a prospective cohort study in Denmark, to research the relationship between the glycaemic index and the resulting changes in body weight in 185 men and 191 women from the Danish arm of the Monitoring Trends and Determinants in Cardiovascular Disease study (MONICA). No significant association was found between the glycaemic load and change in the body weight of men and women. Neither between the glycaemic index and changes in the body weight of men. Among women, the glycaemic index was positively associated with weight changes in adjusted analyses ($p < 0.04$). In six years the values per 10-unit increase in baseline GI increased by 2% (CI 95%: 0.1; 4) for body weight.

Du et al.[158] carried out a prospective cohort study with 89,432 Europeans aged between 20 and 78, who were monitored for an average of 6.5 years to assess the effect of the GI and the GL on body weight and the abdominal circumference. The study does not support its effect on the change in body weight. The GI (but not the GL) was moderately associated with a larger abdominal circumference.

An RCT carried out in Denmark with 45 women aged between 20 and 40, assessed the effects on body weight of a diet low in fat and high in carbohydrates with a low GI or a high GI and was published by Sloth et al.,[159] estimated that body weight decreased significantly in both groups, but that the differences between them were not significant. The authors concluded that the study did not support the hypothesis that diets low in fat and with a low glycaemic index are more beneficial than those with a high GI in terms of body weight, at ten weeks.

An RCT carried out in Brazil,[160] studied the long term effect of a diet with a low glycaemic index compared with a diet with a high GI on the change in body weight in 203 women aged between 25 and 45. After 18 months the weight change was not significantly different between both groups.

De Rougemont et al.,[161] carried out an RCT in France which examined the effects of a diet with a high or low GI on body weight, the BMI and other parameters in adults. The participants were randomly subjected to different diets. After 5 weeks of intervention, the body weight and BMI decreased significantly in the group with the low GI, while the changes in the group with the high GI were not statistically significant. The differences between the groups according to body weight and BMI were significant ($p = 0.04$ and $p = 0.03$, respectively). The authors concluded that the groups with a low glycaemic index may benefit from these diets to regulate body weight. However, this study was carried out with overweight people.

In 2008 a meta-analysis indicated, after reviewing studies published up to 2005, that a reduction in the glycaemic load equivalent to 17 g of glucose per day was associated with a reduction in body weight and vice versa.[162]

Within the framework of the scientific update sponsored by the FAO-WHO, van Damm concluded in 2007 that the studies which assess the effect of the GI on body weight have not been consistent.[123] Finally, the European Food Safety Authority indicated in 2010 that there is no evidence demonstrating that the glycaemic index or the glycaemic load are involved in controlling body weight.[119]

Evidence

13. There is not sufficient evidence to assert that the glycaemic index and glycaemic load of the diet are associated to increased body weight in healthy adults.

3.2. Lipids and body weight

Dietary fats, or lipids, are macronutrients which include fatty acids, triglycerides, and cholesterol. Both the quantity and quality of fatty acids vary according to the food source, and it is possible to observe differences between meat, fish, vegetables and food obtained from industrial processes, among others.

As fatty acids are a heterogeneous group of substances, the biological effects vary significantly. There is evidence which shows that human oxidation and storage of SFA's, MUFA's, PUFA's and TFA's are different,[163] a fact which supports the hypothesis that the different types of fatty acids contribute differently to weight gain.[164,165] For this reason the effect on preventing body weight gain will be analysed separately for total fats, saturated fats (SFA), monounsaturated fats (MUFA), polyunsaturated fats (PUFA), omega-3 and trans fatty acids (TFA).

Total fat

Fat intake in Spain covers approximately 40% of the energy of the diet,[82,118] a figure which is above the upper limit (35%) established by the European Food Safety Authority.[166]

Fat is the macronutrient with the most energy and it exerts a weak effect on satiety. Bray et al.[167] have suggested that a high proportion of fat in the diet may lead to weight gain because it stimulates excessive energy intake, as it satisfies hunger less than the same quantity of energy from carbohydrates. The Dietary Guidelines Advisory Committee of the United States has recently indicated that fat plays a key role in maintaining the energy balance and maintaining weight.[16]

Despite this, currently there is an intense scientific debate about its role as a predictor of obesity and in unintentional weight gain.[121,167-170] In general, diets with a higher percentage of fat-based energy are associated with a higher energy intake,[171-173] although it is not clear if the fat content affects weight gain after adjustments are made for total energy intake.

An analysis from the Nurses' Health Study including 41,518 nurses reported a weak positive association between the consumption of total fat and weight gain at 8 years.[165]

In 2009, Forouhi et al.[174] published a prospective follow-up study of 89,432 European adults in 6 cohorts from the EPIC study in which the association between the total quantity of fat and weight change was assessed, without a significant relationship between both parameters being observed.

Donnelly y cols.[175] carried out a randomised trial to assess the effect of diets with different percentage of fat for preventing weight gain in 305 healthy adults who were monitored for 12 weeks, with normal weight and overweight. While there was an association between the energy intake and weight gain, no relationship was observed with the percentage of fat-based energy.

EVIDENCE

14. Fat intake, after adjusting for the total energy intake, is not associated to weight gain in healthy adults (Evidence Level 2+).

RECOMMENDATIONS

8. In order to prevent weight gain in healthy adults, control of the total energy intake is more important than control of total fat (Recommendation Degree C).

SATURATED FATTY ACIDS (SFA'S)

The main types of SFA in the diet are lauric, myristic, palmitic and stearic acid. It is estimated that the two types of food which contribute most to the intake of SFA's in the European and Spanish diets are full-fat dairy products and meat.[176-178]

The intake of SFA in Spain covers 12.1% of the diet's energy.[4] This figure is above the maximum which is recommended by the World Health Organisation (10%) and the Dietary Guidelines Advisory Committee of the United States (7%) to prevent chronic diseases associated with its excessive consumption.[9,16] The European Food Safety Authority[166] has not established a reference intake, advising that "the smallest possible quantity" be consumed.

SFA intake has been associated with reductions in energy expenditure after comparing its intake with unsaturated fatty acids,[179] although more studies are required to confirm these observations.[166]

In a study in the Nurses' Health Study, Field et al.[165] observed a strong positive association between saturated fat intake and weight gain at 8 years. To the contrary, Forouhi et al.[174] did not observe a significant relationship between SFA intake and weight gain in the EPIC prospective study. They observed a weak association in women, but without statistical significance.

EVIDENCE

15. Investigations addressing the relationship between saturated fatty acids intake in healthy adults and risk of obesity have yielded contradictory results.

MONOUNSATURATED FATTY ACIDS (MUFA'S)

MUFA intake has been associated with various health benefits, in particular a possible role as a cardioprotector.[180]

In Spain it is estimated to cover 17.6% of the diet's energy,[118] with olive oil being the food which most contributes to its intake,[177,178] which is analysed in section 4.5. The European Food Safety Authority has no specific recommendations relating to MUFA's.[166]

The analysis of the Nurses' Health Study did not observe that greater consumption of MUFA's was associated with weight gain at 8 years.[165] Nor in the analysis of 6 cohorts from the EPIC study was any significant association observed between the quantity of MUFA and weight change.[174]

EVIDENCE

16. Monounsaturated fatty acids intake has shown no association to weight gain in healthy adults (Evidence Level 2+).

POLYUNSATURATED FATTY ACIDS (PUFA'S)

Polyunsaturated fatty acids (PUFA's) present two or more points of unsaturation in their chain. Their intake has been associated with various health benefits, in particular linolenic and alpha-linolenic acid, as humans cannot synthesise them from other substrates.[166]

PUFA intake in Spain covers 6.7% of the diet's energy,[4] a figure which is within the established range recommended by the World Health Organisation in 2003 (6-10%). The European Food Safety Authority has no specific recommendations relating to PUFA's.[166]

Neither the observations in the study by Field et al.[165] nor those published by Forouhi et al.[174] associate the consumption of PUFA's with weight gain or change. Although in the EPIC study it was concluded that there was a positive association for women when considering the PUFA/SFA ratio, this was weak and without statistical significance.

Omega-3 fatty acids are an essential type of PUFA whose first double bond is located in the third carbon atom, starting from the end of the chain (methyl group). Alpha-linolenic acid (ALA) is an essential omega-3 fatty acid of plant origin, which enables the human body to synthesise long chain omega-3 fatty acids (EPA and DHA).[181] The intake of omega-3 fatty acids has been associated with various health benefits.[166]

The European Food Safety Authority (EFSA) recommends a suitable intake of ALA of 0.5% of energy and a suitable intake of EPA+DHA of 250 mg/day in adults.[166] Oily fish and, to a lesser extent, white fish are the main sources of omega 3 fatty acids. Nevertheless, large differences are observed in the different regions of Spain.[177]

The EFSA indicates that studies on humans do not provide evidence which indicates that omega-3 fatty acids affect the energy balance.[166] Furthermore, no cause-effect relationship has been established between the consumption of DHA and maintaining a normal body weight.[182]

No evidence has been found in randomised trials or longitudinal studies about the role of omega-3 fatty acids in the prevention of weight gain in healthy adults. In a review by Mousavi et al.[183] it is concluded that the evidence regarding the relationship between the consumption of omega-3 fatty acids and weight gain is inconsistent.

EVIDENCE

17. Polyunsaturated fatty acids intake has shown no association to weight gain in healthy adults (Evidence Level 2+).

18. The evidence regarding the intake of omega-3 fatty acids and its effects on body weight variability or prevention of weight excess in adults is insufficient for establishing any definite recommendation.

TRANS FATTY ACIDS (TFA'S)

Trans fatty acids (TFA) are monounsaturated and polyunsaturated fatty acids which contain at least one double bond in the trans configuration. This configuration can be the result of a microbial fermentation process in the rumen of ruminants (which leads to the presence of TFA in dairy products and in their meat) and through certain hydrogenation processes carried out by some segments of the food industry.[184] Anyway, in the last decade there has been a significant reduction in the TFA content of many foods and it is therefore important that the database of the composition of foods which are used to assess the impact of TFA's on health be updated.[185] In Spain, TFA's provide 0.7% of the energy intake184, a figure which is below the upper intake limit (1%) recommended by the World Health Organisation in 2003.[9]

Regarding the association between TFA intake and weight gain, there is very little available evidence. A review carried out in 2009 by Mozaffarian et al.[186] using observational studies and clinical trials argues that the long term effects have not been assessed through RCT's on humans, due to ethical restrictions, so controlled trials have been carried out on primates.

In a cohort study[187] with the participation of 16,587 men whose abdominal circumferences were measured twice in 9 years, it was observed that every 2% increase in the consumption of TFA (in comparison with its energy equivalent in polyunsaturated fatty acids) was associated with a 2.7 cm increase in the waist circumference after adjustments were made for measuring errors and other confounding factors. A second study[165] with 41,518 women whose weight was measured twice in 8 years showed an association between an increased intake of TFA and an increase in body weight, in both transversal and longitudinal analyses.

In both studies, the changes in adiposity or weight associated with the consumption of total fats, saturated fatty acids, monounsaturated fatty acids and polyunsaturated fatty acids were less consistent.

The two prospective observational studies, with study periods of 8 and 9 years, suggest that the consumption of TFA stimulates weight gain and, in particular, the accumulation of abdominal fat.

EVIDENCE

19. The limited epidemiological studies available show a consistent relationship between the role of trans fatty acids in weight gain and in the increase of abdominal fat (Evidence Level 2–).

3.3. Proteins and body weight

It is important to review the role of proteins in the prevention of obesity, both because of the current popularity of diets with a high protein content for controlling obesity,[188] and to assess the risk-benefit relationship of increasing the current consumption of proteins, as Spain is one of the countries where more proteins are consumed.[189] Among the risks of increasing the consumption of proteins it is worth highlighting data which exists which associates high intakes of protein with a greater risk of suffering from osteoporosis, kidney stones, renal failure, cancer or cardiovascular disease.[190] Two population-based cohort studies with large samples monitored for between 10 and 26 years have recently showed that the prolonged consumption of diets low in carbohydrates and rich in proteins is associated with a higher risk of mortality,[191,192] although it is probable that this effect can be attributed to meat more than protein as a whole, as observed by Sinha et al., in a prospective study with more than half a million people.[193]

The role of proteins in body weight is however debatable. Although it has been suggested that a higher intake of proteins could increase satiety in the short term and therefore decrease energy intake,[194] there are studies which do not support this association.[195-198] The European Authority of Food Safety also considers the evidence in this respect to be insufficient.[199]

Moreover, it is possible that the role of proteins in weight control has different effects according to whether it is predominantly plant-based or animal-based,[198,200] and it is for this reason that they are analysed separately below. A section has been devoted to soy protein, given the extensive literature found in reference to its possible role in body weight.

TOTAL PROTEIN

Only one prospective study has been found which has assessed the effects of consuming total protein (irrespective of whether it is of plant or animal origin) on the body weight of healthy adult humans. After a follow-up of 182 women and 168 men during 23 years, Koppes et al.[201] concluded that protein intake was significantly associated with a higher BMI.

EVIDENCE

20. The evidence regarding the total protein intake and its effects on body weight variability or prevention of weight excess in adults is insufficient for establishing any definite recommendation.

PROTEINS OF ANIMAL ORIGIN

Protein of animal origin includes protein from meat, eggs, milk and the products deriving from those foods.

Five articles have been selected to examine the relationship between proteins of animal origin and body weight in healthy adults.

Kahn et al.[202] assessed the changes in BMI and in the waist circumference in a cohort of 79,236 adults monitored over 10 years. An increase in BMI was directly associated with the consumption of meat and inversely associated with the consumption of plant-based foods, after adjustments were made for numerous possible confounding factors.

In 2006, Rosell et al.[116] evaluated the weight gain in 5 years of 21,966 adults belonging to the European Prospective Investigation Into Cancer cohort (EPIC-Oxford). After adjustments were made for potential confounding factors, it was observed that weight gain was significantly lower in volunteers who had changed their diet to one containing less food of animal origin during the follow-up.

In 2008, after a follow-up of 8,401 volunteers of the Adventist Health Study, Vang et al.[203] observed a significantly higher risk of weight gain associated with the intake of red meat, poultry and processed meat.

There were inconsistent findings in 2009 in a cohort of English adults[204] in which the consumption of red meat and processed meat and their relationship with body weight and abdominal circumference were studied with a follow-up of 10 years. Body weight increased by more than 5 kg for men and women between 1989 and 1999. Men with the highest consumption of red meat and processed meat combined in 1989 had a statistically significantly higher BMI and abdominal circumference. In women, the consumption of red and processed meat was statistically significantly associated with a higher BMI in 1999. Nevertheless, this is a study which is subject to methodological biases, according to the Nutrition Evidence Library of the United States Department of Agriculture.[205]

Finally, in 2010, Vergnaud et al.[198] assessed the association between the consumption of meat (red meat and processed meat) and weight gain in adults. The authors carried out a 5 year follow-up of 270,348 women and 103,455 men participating in the EPIC-PANACEA project (European Prospective Investigation into Cancer and Nutrition-Physical Activity, Nutrition, Alcohol, Cessation of Smoking, Eating Out of Home and Obesity). After adjustments for numerous potential confounding factors, a positive association was observed between the consumption of red meat, poultry and processed meat and a higher BMI.

EVIDENCE

21. The evidence regarding the intake of animal protein and its effects on body weight variability or prevention of weight excess in adults is insufficient for establishing any definite recommendation.

PROTEINS OF PLANT ORIGIN

Protein of plant origin refers to protein which is present in cereals, fruits, vegetables, pulses, nuts, seeds or products deriving from those foods.

No study has been found which specifically assesses the effect of vegetable based proteins (excluding the effect of soy protein, which is analysed separately) on controlling body weight.

EVIDENCE

22. The evidence regarding the intake of vegetable protein and its effects on body weight variability or prevention of weight excess in adults is insufficient for establishing any definite recommendation.

PROTEINS OF PLANT ORIGIN (SOY)

In 2008 a systematic review was published[206] which included 91 works, with results from *in vitro* data, studies of animals, epidemiological and clinical studies which assessed the relationship between soy foods, including soy protein, and the prevention of weight gain. The authors concluded that the consumption of soy or its derivatives, including soy protein, was not associated with a lower BMI or with a reduction in weight gain over time in the available epidemiological studies. Furthermore, it is not clear that its consumption leads to weight loss in "ad libitum" diets.

McVeigh et al.[207] examined the effects of soy proteins with different isoflavone content on serum lipids in young healthy men (27.9 ± 5.7 years) in a transversal randomised trial in Canada. Body composition was measured by bioelectric impedance at the beginning of treatment and after 57 days. During the study, no significant differences in treatment were observed in anthropometric measurements, including body weight, BMI and the percentage of body fat.

A transversal analysis with 2,811 adults in the Nutrition and Health of Aging Population study in China[208] evaluated the association between soy protein intake and the risk of metabolic syndrome and its components. In that study, the authors observed no statistically significant association between soy protein intake and central obesity in men (p-trend = 0.655), in women (p-trend = 0.827), or in the total sample as a whole (p-trend = 0.757).

EVIDENCE

23. No evidence has been found regarding the intake of vegetable (soybean) protein and its effects on body weight variation that might allow definite recommendations concerning weight gain prevention in adults.

3.4. Vitamins and minerals

Research published in 1999 assessed the consumption of vitamin supplements and minerals in 39,833 Spaniards aged between 29 and 69. Intake during the week prior to the interview was 5.2% in women and 1.7% in men.[209] More recent data, published in 2009, based on a sample of 3,220 Spaniards, observes a prevalence of consumption of dietary supplements of 12.1% in women and 5.9% in men, of which 70% in women and 66% in men are solely vitamins, minerals or vitamin and/or mineral supplements, revealing an increase in the consumption of this kind of supplement. Even though, the consumption percentage of dietary supplements is lower than that observed in other European countries such as the United Kingdom, Denmark, Switzerland or Norway, where in some cases it equals a third of the population.[210] The American Dietetic Association (ADA) indicates that in the United States one in every three adults regularly takes them.

Despite the growing belief that vitamin-mineral supplements may prevent certain chronic diseases, in December 2009 the ADA indicated that evidence has not demonstrated that they are effective in doing so.[211] In fact, there is data which indicates that taking a high quantity of supplements could increase the risk of suffering from certain chronic diseases.[212,213]

Some studies have explored the association between the BMI and some micronutrients. Kimmonds et al.[214] have analysed this relationship through the blood levels in a representative sample from the USA (National Health and Nutrition Examination Survey III) of adults above the age of 19. In this study the increase in BMI was related to low levels of certain nutrients (alpha-carotenes, beta-carotenes, beta-cryptoxanthin, lutein/zeaxanthin, total carotenoids, vitamin C, selenium and folate) in comparison with subjects with normal weight. The authors concluded by indicating that it is necessary to study these relationships in greater depth.

In Spain, Zulet et al.[215] assessed the possible association between the intake of vitamin A with the BMI in 61 healthy adults aged between 18 and 22. After adjustments were made for total energy intake, the intake of vitamin A showed a negative correlation with various measurements of adiposity.

Sneve et al.[216] carried out a double-blind RCT aimed at studying whether a supplement of 20,000 UI of vitamin D once or twice a week for 12 months led to weight change in 445 subjects with overweight or obesity. During the study no significant changes were observed in weight, the waist-hip ratio or the percentage of body fat in any of the groups or between some groups and others.

Furthermore, Jorde et al.[217] examined the transversal and longitudinal relationship between the BMI and 25(OH)D serum levels through the Tromsø Study with an intervention (RCT) lasting for a period of one year, with 93 subjects who received 40,000 UI of cholecalciferol per week. A strong negative association was observed between 25(OH)D serum levels and the BMI.

Calcium is worthy of a special mention because the hypothesis that this nutrient could be beneficial for the prevention of weight gain (or its loss) has generated a lot of scientific literature. A systematic review[218] with the subsequent meta-analysis of 13 RCT's, which used supplements with calcium and which reported body weight as the final outcome, found no association between an increase in the consumption of each calcium supplement or dairy products and a loss of weight after adjustments for differences in initial weights between the control and intervention groups.

Teegarden et al.[219] carried out a study to research the impact of dietary calcium or the intake of dairy products on total energy expenditure. No differences were observed between the groups in terms of total energy expenditure.

In 2009, in order to test the hypothesis that supplementing diets with calcium can prevent weight gain in persons with overweight or obesity, an RCT was carried out in which the diet of 340 volunteers was supplemented with 1,500 mg of calcium/day for 2 years, and no statistically significant clinical effects on weight were observed.[220]

Caan et al.[221] carried out a double-blind randomised trial with 36,282 postmenopausal women aged between 50 and 79, belonging to the Women's Health Initiative clinical trial study, to assess the combined effect of vitamin D and calcium. The volunteers received 1,000 mg of calcium and 400 UI of cholecalciferol (vitamin D), or a placebo, every day and their weight changes were evaluated for an average of 7 years. The women who received the supplements presented a minimal, but favourable, difference in body weight (average difference -0.13 kg; CI 95%, -0.21 to -0.05; p = 0.001). The authors concluded that supplementing calcium and cholecalciferol had little effect on the prevention of weight gain in postmenopausal women.

EVIDENCE

24. The existing evidence shows that calcium supplementation is not associated to a lower weight gain (Evidence Level 1+).

25. The existing evidence shows that combined supplementation of calcium with vitamin D does not achieve clinically relevant improvements in body weight control in postmenopausal women (Evidence Level 1+).

26. The available evidence regarding the role of vitamin D alone for preventing weight gain in healthy adults is controversial and does not allow any conclusions to be drawn.

3.5. Dietary fibre

According to the latest scientific update sponsored by the FAO-WHO, dietary fibre refers to the intrinsic polysaccharides of the plant cell walls.[222] Although it has traditionally been categorised according to its solubility in water, it has been recommended that the terms "soluble" and "insoluble" be gradually replaced by terms which refer to fermentability and viscosity.[222,223]

A high intake of dietary fibre is associated with numerous health benefits.[224] However, the average total consumption of DF in Spanish homes[225] has been found to be far below the dietary reference intakes.[226]

One of the benefits attributed to dietary fibre is that it modulates body weight through various mechanisms,[223] one of which is its contribution of low energy density to the diet.[136] Despite this, it is a matter for dispute whether the DF association with body weight is due to fibre intake or other possible confounding factors, including dietary factors.[227]

Consuming dietary fibre from foods has been associated with smaller weight gain in transversal studies.[155,228-230] A transversal study which assessed 16 cohort studies in 7 countries showed that BMI was inversely associated with fibre intake from foods.[231]

The association between food intake and changes in body weight has been examined in various prospective cohort studies whose results have been adjusted for potential confounding factors. Koh-Banerjee P et al.,[232] showed that an increase in fibre intake from fruit and whole grains was inversely associated with weight gain in the long term. The dose-response relationship was stronger for fibre from fruit. For each 20 g/d increase, the weight gain was reduced by 2.51 kg (p value for trend < 0.001), in a cohort of 27,082 men (aged between 40 and 75) with a follow-up of 8 years. Similar results were observed among women,[137,233] young adult women,[234] in a Mediterranean population[235] and in a sample comprising 89,432 European adults.[236]

Finally, the RCT's which have assessed the effect of increasing the fibre content of the diet through dietary supplements have provided inconsistent results.[227]

EVIDENCE

27. A high dietary fibre intake in the context of a diet rich in food of vegetable origin is associated to a better control of body weight in healthy adults (Evidence Level 2++).

RECOMMENDATIONS

9. Increasing the intake of dietary fibre from vegetable origin food might prevent weight gain in healthy adults (Recommendation Degree B).

3.6. Water

There is a deeply-rooted belief that water intake facilitates weight maintenance. A review carried out in 2009[237] attempted to study this matter, concluding that although the limited epidemiological data available suggested a beneficial effect from consuming water to reduce energy intake and facilitate weight control (in particular when used as a substitute for drinks high in calories), intervention studies were required to make intake recommendations for the consumption of water based on evidence.

In 2005, epidemiological data showed that; in the USA energy intake among water drinkers is approximately 9% lower than among non-drinkers of water[238] but it is a relationship which does not prove causality.

Furthermore, an observational analysis published in 2009 on the basis of data from 16,395 American adults, concluded that water intake was not associated with BMI.[239]

Water consumed before or together with a meal was associated with a reduction in the feeling of hunger and an increase in satiety in a small comparative study carried out with 21 middle-aged subjects who were not obese (aged 60-80), but not when this effect was assessed in a younger population (aged 21-35, n = 29).[240]

A transversal observational study carried out in Japan[241] with 1,136 young female students (aged between 18 and 22), after adjustments for potential confounding factors, estimated that the consumption of water from drinks was not associated with the BMI (p trend = 0.25) or with the abdominal circumference (p trend = 0.43). However, water intake from food showed an inverse and separate association with the BMI (p trend = 0.03) and with the abdominal circumference (p trend = 0.0003).

The Beverage Guidance Panel of the USA made recommendations about the benefits and risks of different categories of drinks, taking into consideration weight and health status.[242] These recommendations were brought into question as they were not supported by scientific evidence.[243,244]

In 2004, The American group responsible for Dietary Reference Intakes for electrolytes and water recommended that individuals pay attention to feelings of thirst and that they consume drinks to maintain their hydration status, but it did not make any recommendations regarding the consumption of drinks and weight control. In turn, the Dietary Guidelines Advisory Committee of the United States[246] has recently indicated that there is insufficient evidence to establish relationships between water intake and body weight.

Evidence

28. The available evidence regarding water intake and its effects on body weight variation and/or prevention of weight excess in healthy adults is insufficient for establishing any definite recommendation.

3.7. Ethanol and body weight

Ethanol is an alcohol which constitutes the main product of alcoholic drinks such as wine, beer and spirits. Although its consumption through alcoholic drinks is associated with the diets of practically all populations, in terms of metabolism it must be considered a substance which is capable of providing energy but which is not recognised as performing any essential function for living beings and, unlike macronutrients, it is not transported by proteins, it freely diffuses, it is impossible to regulate and it cannot be stored as a macromolecule. In this text we will use the words alcohol and ethanol as exact synonyms.

Because of its effects on the central nervous system and other target organs, and because of its addictive and toxic capacity, it is considered a psychoactive drug.[247] From a public health perspective, its high consumption (in Spain it has been calculated to represent approximately 5% of the daily energy intake, in other words 247 grammes of alcoholic drink/day),[82,248] it is associated with high morbidity, mortality and social problems. The consumption of alcohol (even when moderate) has been associated both with positive and negative relationships for certain health problems, so advice on its consumption must be assessed individually.[85,247-254]

As a substance associated with nutrition, it presents considerable energy density, so its consumption on a regular basis could theoretically cause imbalances in the energy balance of individuals. In this sense, the consumption of alcoholic drinks has been associated with an increase in the feeling of hunger,[255] with less control of the satiety mechanism[256,257] and with weak dietary compensation in response to its consumption in the short term.[258] Despite this, evidence regarding its effect on body weight provides contradictory results. Below there is a review of the prospective studies which have assessed the relationship between the regular consumption of alcoholic drinks and body weight or the abdominal circumference.

Between 1996 and 2011 ten observational prospective studies have been identified which are aimed at establishing associations between the consumption of alcohol and weight gain or increases in the abdominal circumference.

The study by Sherwood et al.[259] indicates that low or moderate consumption of alcohol is not associated with a substantial weight gain, in a study with volunteers from the community. In this study, 826 women and 218 men participating in the Pound of Prevention Study were monitored over 3 years. In this period, the average weight gain was 1.69 kg (SD ± 5.4 kg) in men and 1.76 kg (SD ± 6.7kg) among women, while the average energy consumption fell by 211 kcal per day in men and 168 kcal/day in women, with an increase corresponding to the total energy intake of alcohol of 0.88% and 0.30% respectively. In the prospective analysis, the change in the energy intake from alcohol was not associated with the weight change in the men or women. However, as this was a study of volunteers who were part of a project for the prevention of weight gain, it is probable that their alcohol intake was lower than that observed in the rest of the population.

In 2003, Sammel et al.,[260] carried out a prospective study with a 4 year follow-up, with 336 women (Afro-Americans and Caucasians), to establish factors associated with weight gain in the final reproductive years. They compared alcohol consumption among those (25%) who had gained more than 10 pounds (4.5 kg) and the rest of the women. The average alcohol consumption was 7.3 (SD ± 15.2) and 8.5 (SD ± 19.0) drinks per week, respectively. These differences were not statistically significant after adjustments were made in the multivariate analysis with other predictive factors of weight in this cohort.

The two studies carried out by the Wannamethee team showed an association between the ethanol intake of the heavy drinkers and weight gain at 5 and 8 years of being monitored, both in men and women. In the study by Wannamethee et al.,[261] in the United Kingdom the association between alcohol intake and body weight was examined for a follow-up period of five years in 6,832 men aged between 45 and 64 who were participating in the British Regional Heart Study. After adjustments were made for possible confounding factors (age, social class, physical activity, number of cigarettes, baseline weight), the average BMI and the prevalence of subjects with a BMI above 28 kg/m^2 was not statistically different between teetotal men and light drinkers (< 30 g/day of alcohol) and moderate consumers. However, heavy drinkers (> 30 g per day) showed an Odds Ratio of 1.29 (CI 95%: 1.10; 1.51) with the heavy consumption of alcohol directly contributing to weight gain and obesity in men.

Another study by Wannamethee et al.,[262] carried out with women in the USA, examined the relationship between ethanol and weight gain at eight years of the follow-up (1991-1999) in 49,324 nurses aged between 27 and 44 from the Nurses' Health Study II. Data suggests that the light to moderate consumption of ethanol (less than 30 g/day) is not associated with weight gain, with the possible exception of Afro-American women. Those who drink a quantity of ethanol above 30 g/day may stimulate an increase in weight gain. The most pronounced OR for the association between weight gain and heavy drinkers (> 30 g/day) was observed for women aged below 35 (OR = 1.64; CI 95%: 1.03; 2.61).

Sayon-Orea et al.[263] have recently published a prospective study carried out in Spain, within the SUN cohort, which included 9,318 adults with an average age of 37.9, and after a period of 6.1 years it was observed that high consumption (7 drinks/week) of beer or spirits (but not wine) is associated with a higher risk of overweight/obesity and a greater weight gain. The consumption of alcohol was only measured at the start of the study, and not at the subsequent follow-up, so it is impossible to exclude the possibility of the volunteers changing their alcohol consumption habits. Regarding the apparent benefit of wine in comparison to other drinks, the authors recognise that it could partly be due to confounding factors such as dietary habits or others associated with a healthy lifestyle.

Three bodies of research have only studied changes through the abdominal circumference. Koh-Banerjee et al.[187] did not find a significant association between the total consumption of alcohol and the gain in waist circumference during a nine year follow-up of participants of the Health Professionals Follow-up Study (1986). After a period of 9 years, they examined the association between the change in dietary intake, physical activity, alcohol consumption and smoking and weight gain and gains in the abdominal circumference in a cohort of 16,587 men aged between 40 and 75. Although the average abdominal circumference increased by 3.3 cm (SD + 6.2) during the study period, the data obtained did not achieve statistical significance.

Vadstrup et al.[264] carried out a prospective study with a 10 year follow-up in which they analysed the abdominal circumference, with a sample of 2,916 men and 3,970 women from Denmark aged between 20 and 83 who were participating in the Copenhagen City Heart Study. They concluded that moderate to high consumption of beer and spirits was associated with larger abdominal circumferences.

The study by Tolstrup et al., in 2008,[265] analysed the frequency of alcohol consumption and its relationship with changes in the abdominal circumference and the development of abdominal obesity. The analysis included a total of 43,543 individuals from the Danish Diet, Cancer and Health Study. This study showed that the frequency of consuming alcoholic drinks was inversely associated with a greater increase in the abdominal circumference.

Two studies have evaluated the relationship between alcohol consumption and an increase in the BMI and the abdominal circumference.

A longitudinal population-based study carried out by Pajari et al.[266] evaluated both weight gain and the increase in abdominal circumference associated with alcohol consumption. The alcohol intake, BMI and waist circumference were quantified for 5,563 Finns aged between 16 and 27. After adjustments had been made for confounding factors (smoking, diet, physical activity, place of residence, socioeconomic status and BMI of the parents) no relationships were observed between alcohol intake and weight gain or the development of abdominal obesity. However, in this study it was observed that very few subjects drank frequently, so the volunteers who drank "daily" were grouped into the same category as those who drank "weekly", a fact which limits the evaluation of the effect of high alcohol consumption on weight gain. Furthermore, the consumption of alcohol by adolescents in Finland is much lower than the average for European consumption, so it is possible, as explained by the authors, that the effect of alcohol on body weight observed in this study differs from high consumption in other European countries.

Finally, Bergmann et al.[267] assessed the relationship between lifetime consumption of alcohol and the measurement of abdominal and general adiposity in the EPIC cohort. After adjustments were made for confounding factors, it was observed that the consumption of alcoholic drinks was positively associated with abdominal and general obesity in men and with abdominal obesity in women. It was observed that the increase in the risk of abdominal obesity continually rose in both sexes for consumption above 6 g of alcohol/day (approximately half the alcohol provided by a glass of alcoholic drink).

Evidence

29. The available studies yield contradictory and inconsistent observations, although some evidence does suggest some level of association between high ethanol intake and weight gain (Evidence Level 2–).

Recommendations

10. A restriction of high ethanol intake might prevent weight gain associated to this factor (Recommendation Degree D).

4. Foods and body weight

4.1. Fruit and vegetables

The regular consumption of fruit and vegetables is clearly associated with a better state of health, a lower prevalence of chronic diseases and less risk of mortality.[254] Despite this, 57% of the Spanish population does not consume vegetables on a daily basis, and 62.2% do not consume fruit every day.[118]

It has been reported that an increase in the consumption of fruit and vegetables has a modest association with a smaller weight gain at five or more years for middle aged subjects. The studies reviewed showed an inverse but weak relationship between the consumption of fruit and vegetables and weight gain.

A transversal analysis of the University of Navarra Follow-up study (SUN)[235] determined the association between fibre intake and the consumption of fruit and vegetables with the probability of weight gain in five years. It found a significant inverse association between the consumption of fruit and vegetables and weight gain, but only in men.

The study by Goss and Grubbs[268] compared the consumption of fruit and vegetables in the 7 counties with the highest average BMI with the 7 counties with the lowest average BMI. In the counties with the highest average BMI, 40.5% consumed 3 or less portions of fruit and vegetables per day, compared with 30.3% in the counties with the lowest average BMI. Similarly, 59.6% in the counties with a high average BMI ate 3 or more portions of fruit and vegetables per day, compared with 69.6% in the counties with the lowest average BMI.

In a study carried out in the south of India, Radhika et al.,[269] evaluated the association between the consumption of fruit and vegetables with different cardiovascular risk factors in 983 adults. After adjustments were made for possible confounding factors, the quartile with the highest intake of fruit and vegetables showed a significant inverse association with the BMI and abdominal circumference in comparison with the lowest quartile.

In another study carried out in China[270] which examined the association between the consumption of red meat and vegetables with excess body weight, observed that excess body weight was not significantly associated with the consumption of vegetables.

A matched case-control study carried out in the USA[271] analysed the difference in dietary intake between normal subjects (control) and subjects with overweight/obesity (cases). On average, the subjects with overweight/obesity consumed one ration less than the control group ($p < 0.01$), and it was also found that the rations of fruit per day were inversely associated with the percentage of body fat.

In a prospective cohort study, Buijsse et al.[271] analysed whether there was an association between the consumption of fruit and vegetables with subsequent changes in body weight within the scope of the European Prospective Investigation into Cancer and Nutrition (EPIC). A total of 89,432 adults from Denmark, Germany, the United Kingdom, Italy and the Netherlands were included, with a 6.5 year follow-up. All cohorts gained an average weight of 330 g. The intake of fruit and vegetables was found to be inversely associated with the change in weight; for every 100 g of fruit and vegetables consumed, the change in weight was -14 g per year.

A prospective cohort study[273] carried out in the USA with 74,063 healthy nurses aged between 38 and 63 (Nurses' Health Study) analysed the changes in the consumption of fruit and vegetables in relation to the risk of obesity and weight gain. After a 12 year follow-up the patients with the highest intake of fruit and vegetables had a 24% lower risk of becoming obese and a 28% lower risk of gaining 25 kg or more.

In Spain, Vioque et al.[274] assessed the association between the intake of fruit and vegetables and weight gain in a period of 10 years. The weight gain was significantly lower with each increase in the consumption of fruit and vegetables per quartiles ($p = 0.0001$). Regarding the consumption of vegetables, the risk of weight gain was 82% lower in the quartile with the highest consumption (more than 333 g per day). When considering fruit and vegetable together, the risk of weight gain decreased by quartiles, with the upper quartile having the biggest decrease (OR = 0.22; CI 95%: 0.06; 0.81; $p = 0.022$).

EVIDENCE

30. A high intake of fruit and vegetables is associated with a lower long-term body weight increase in adults (Evidence Level 2+).

RECOMMENDATIONS

11. The dietary prevention of body weight gain may be modulated through the use of diets with a high fruit and vegetable content (Recommendation Degree C).

4.2. Whole grains

In the search for the effects of food groups on body weight and on the prevention of weight gain in adults, whole foods and, more specifically, whole grains have been the subject of various studies. The National Survey on Dietary Intake in Spain reveals that the average consumption of whole-grain bread in Spain is 6 grammes/day. The Spanish Food Safety and Nutrition Agency indicates that "the consumption of grains, preferably whole grains, should increase".[118]

Four transversal studies[228,275-277] consistently observed that the intake of whole grains was associated with a lower BMI and adiposity.

Two prospective studies with a large population sample have shown statistically significant associations between the consumption of whole grains and better control of body weight.[234,278]

Two systematic reviews published in 2008[279,280] have been found (one associated with a meta-analysis) in which it is concluded that there is an association between the consumption of whole grains and a lower BMI and protection against gains in weight and adiposity.

That which was published by Williams PG et al.[279] assessed whether there was evidence when observing the role of whole grains and pulses in the prevention and control of overweight and obesity. For the purpose of our analysis of the 53 studies considered, 20 examined whole grain intake and, of them, 10 out of 11 studies found that a high intake of whole grains was associated with a lower prevalence and measurement of obesity. The remaining studies addressed weight reduction to control obesity. The authors concluded that there was robust evidence that a diet with high whole grain content was associated with a lower BMI, with smaller abdominal circumferences and with a lower risk of being overweight.

Harland JI and Garton LE,[280] carried out a systematic review to examine the relationship between the consumption of whole grains and body weight in which they included 15 transversal trials published between 1990 and 2006. A total of 119,829 subjects aged above 13 were included in a pooled analysis. The authors concluded that a high intake of whole grains (approximately 3 rations per day) was associated with a lower BMI and central adiposity. They also indicate that people who consume whole grains have healthier lifestyles (less smokers, more frequent physical activity, less fat in the diet and higher fibre content).

EVIDENCE

31. A high intake of whole grains is associated with a lower Body Mass Index (Evidence Level 2+).

RECOMMENDATIONS

12. It is recommended that, in order for body weight gain prevention, the diet contain a considerable proportion of whole grains (Recommendation Degree C).

4.3. Sugars

In 2003 the World Health Organisation (WHO) defined "free sugars" as the monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and fruit juices.[9] However, in 2007, within the framework of the scientific update sponsored by the FAO-WHO, Cummings and Stephen indicated that such a term creates difficulties and suggested replacing it with "total sugars" (all of the monosaccharides or disaccharides present in a food, with the exception of polyols) or specific monosaccharides and disaccharides.[220] They also consider the term "added sugars" to be unsuitable, a term which is used by the Institute of Medicine (IOM) in the United States to refer to sugars and syrups added to food and drink while it is being processed or prepared (this includes sugar, brown sugar, corn-based sweeteners, dextrose, fructose, honey, invert sugar, etc.), with the exception of those naturally present in milk and fruit.[32]

Moreover, in 2003 the WHO recommended not exceeding 10% of energy intake from "free sugars" to prevent weight gain, recognising that this recommendation was open to debate. Its recommendation was based on studies in which it was observed that limiting the free sugar content of a diet resulted in improvements in body weight control9. Furthermore, in 2005, the IOM suggested not exceeding 25% of the energy intake from "added sugars".[190] This cut-off point was established on the basis of data from dietary surveys which showed that above this level it was more likely to have a low intake of important essential nutrients.[32] The European Food Safety Authority (EFSA) considers the available evidence to be insufficient to establish an upper limit for the consumption of added sugars based on their effects on body weight.[119]

Data has not been found on the consumption of "free sugars" (as defined by the WHO) or "added sugars" (as defined by the IOM) for the Spanish population. The ESFA indicates that the energy intake from sugars in the European population ranges from 16 to 36%.[119]

Sugars are carbohydrates with low nutritional density whose intake must be reduced, according to the Dietary Guidelines Advisory Committee of the United States, because they contribute towards excess calorie intake.[16] The World Health Organisation has recently indicated that there is convincing evidence regarding the relationship between the consumption of food high in sugars and obesity when those foods replace the consumption of foods with low energy density, such as fruit and vegetables.[254]

Despite this, the role of sugars in stimulating weight gain is disputed, as the studies in that respect provide inconsistent outcomes.[170,281]

A review carried out by Saris indicated that the consumption of sucrose in solid foods was not clearly associated with the prevalence of obesity, although it recognised that there was a lack of evidence (particularly regarding the liquid or solid form in which sucrose is consumed) in that respect.[170] No cohort studies or RCT's published since 1996 have been found regarding the specific role of sugars (total, added and free) in body weight, except for those focused on sugary drinks (see section 4.4).

In 2010, the EFSA indicated that the evidence about the repercussions of consuming added sugars on weight gain are inconsistent for solid foods.[119]

The review of the literature which has been carried out by the team of writers, with the same inclusion criteria methods, on publications which have evaluated the relationship between the consumption of acaloric sweeteners and the prevention of weight gain in adults between 1996 and 2011, has found no relevant study.

EVIDENCE

32. The evidence regarding free or total sugars intake (with the exception of sugared beverages) in relation to body weight gain is controversial.

4.4. Sugary drinks

The consumption of sugary drinks is currently a cause for concern. The Dietary Guidelines Advisory

Committee of the United States, with the assistance of the Cochrane Collaboration, has recently stated that, in order to reduce the incidence and prevalence of obesity in the United States, the consumption of sugary drinks must be avoided.[16] As indicated by Johnson and Yon,[295] the word "avoid" is, to date, the most emphatic recommendation made in a document of this type, indicating the strength of the association between the consumption of this type of drink and obesity.

Several systematic reviews have been considered to analyse the possible impact of the consumption of sugary drinks on obesity in adults.[283-285] The reviews by Malik et al.[283] and Wolf & Dansinger,[284] covered an extended search period (from 1996 to the end of 2006) and they included transversal studies, prospective cohort studies and experimental studies. Among the transversal studies, the study by Liebman et al.[286] carried out with 1,817 subjects (Rockies Study) found a significantly high probability ($p < 0.05$) of overweight and obesity in subjects who drank one or more soft drinks in a week. Among the prospective cohort studies of adults,[287-289] two of them[287,289] presented statistically significant results. The most numerous study (51,603 women from the Nurses' Health Study II) was carried out by Schulze et al.[287] with an 8 year follow-up, and it estimated that there was a significant association between the consumption of drinks sweetened with sugar and fruit juices and weight gain and increases in the BMI. In the Spanish population, Bes-Rastrollo et al.[289] analysed the data from 7,194 adults with an average age of 41 for 28 and a half months, finding a significant association between the consumption of sugary drinks and weight gain. Kvaavik et al.[188] did not observe a significant association between sugary drinks and change in BMI among younger adults (aged 23-27) during a follow-up period of 8 years.

With an average follow-up of 4 years in the Framingham Heart Study,[290] the consumption of one or more drinks per day was associated with the increase in the OR of suffering from obesity and an increase in the abdominal circumference in comparison with those who did not consume them. Palmer et al.,[291] included sugary drinks and fruit juices in the analysis of type 2 diabetes in Afro-American women, observing that they gained weight during the study, but that this increase was smaller among those who reduced the consumption of this type of drink.

The prospective cohort study carried out in the USA by Chen et al. in 2009[140] examined how changes in the consumption of sugary drinks affects body weight in adults (810 adults from the PREMIER Study). A reduction of one daily ration of sugary drinks was associated with a loss of 0.49 kg at six months and 0.65 kg at 18 months. These authors also observed a significant dose-response trend between changes in body weight and the consumption of sugary drinks.

Two intervention studies in adults,[148,292] with few subjects, 15 and 41 adults respectively, observed significant increases in body weight and BMI, observing significantly more in the subjects who consumed sugary drinks than those who consumed solid sweets or drinks with sweeteners.

A meta-analysis[293] published in the American Journal of Public Health showed a clear and consistent association between the consumption of sugary drinks and an increase in energy intake and body weight. Although this meta-analysis contains data from studies with a wide range of ages which fall outside this review, the results suggest that it could be wise to recommend that people reduce their consumption of the aforementioned drinks.

In contrast, Gibson[294] reviewed six longitudinal studies, observing that in two of them the evidence was strong, in one it was probable, while in the other three it was not conclusive.

Evidence

33. Frequent intake of sugared beverages is associated with a higher Body Mass Index (Evidence Level 2+).

Recommendations

13. Restricting the frequency of sugared beverages intake may lead to a lower body weight gain over time (Recommendation Degree A).

4.5. Olive oil

The consumption of olive oil, one of the characteristic foods of the Mediterranean diet, has been associated with numerous effects which are beneficial to health,[295,296] possibly because of its role as a protector against cardiovascular disease.[297,298]

The intake of olive oil in Spain is notably higher that that observed in other countries in Europe,[177,178] which makes it important to assess its possible effect on unintentional weight gain, despite it being a food with a high calorie density.

A transversal study carried out in Spain by González CA et al.[299] with a sample of 37,663 adults of both sexes aged between 29 and 69 did not observe a significant association between the consumption of olive oil and the BMI.

In 2009, Soriguer et al.[300] published a cohort study with 613 randomly selected adults in Pizarra (Malaga). They assessed the consumption of olive oil and other oils and its relationship with weight gain after a follow-up of 6 years. The weight gain and the incidence of obesity were lower in volunteers who regularly consumed olive oil, after adjustments were made for various possible confounding factors.

In the SUN cohort, Bes-Rastrollo et al.[301] evaluated the association between the consumption of olive oil and the risk of gaining weight after an average follow-up of 28.5 months. No statistically significant associations were found between olive oil intake and the risk of weight gain.

Evidence

34. The intake of olive oil does not seem to be associated with a significant body weight gain risk in healthy adults (Evidence Level 2–).

4.6. Nuts

The regular consumption of nuts has been associated with numerous health benefits, including a lower risk of mortality.[302-304] The National Survey on Dietary Intake in Spain of 2011[118] indicates that the consumption of nuts in Spain is 2.6 rations/week.

Although the health benefits of nuts are beyond doubt, doubts have emerged about promoting their consumption as there are concerns that they may generate undesired increases in the BMI because of their energy and fat content, which is higher than in other plant based foods.

In a prospective study of 8,865 adults from the SUN cohort (University of Navarra Follow-Up) in 2007, Bes-Rastrollo et al.[305] studied the association between the consumption of nuts and the risk of weight gain, after a follow-up of 28 months. After adjustments were made for confounding variables, it was observed that the participants who consumed nuts twice or more every week had a lower risk of gaining weight (OR = 0.69, CI 95%: 0.53; 0.90) than those who never or almost never ate them.

Another prospective cohort study by Bes-Rastrollo et al. which was carried out in the USA in 2009[306] researched the long term relationship between the consumption of nuts or peanut butter and weight change among 51,188 women participating in the Nurses' Health Study II. After an eight year follow-up, the women who declared that they ate nuts more than twice every week had a slightly lower weight gain than those who consumed them sporadically (5.04 ± 0.12 kg *vs.* 5.55 ± 0.04 kg, $p < 0.001$); the results were similar when the subjects where divided by normal weight, overweight and obesity. After adjustments for confounding variables, the consumers of nuts (more than twice a week) presented a lower risk of suffering obesity than those who never or almost never consumed them (RR = 0.77; CI 95%: 0.57-1.02; p = 0.003).

In 2005, Sabaté et al.[307] carried out an RCT in the USA in which they assessed potential weight changes and body composition associated with the consumption of nuts in 90 volunteers, over a period of six months. The group which supplemented its diet with nuts increased its energy intake by 133 kcal, increasing their weight (0.4 ± 0.1 kg, $p < 0.01$) and BMI (0.2 ± 0.1 kg/m^2, $p < 0.05$). However, after adjustments were made for the energy difference between the diets, no significant differences were observed in weight and body composition, but they were in BMI (0.1 ± 0.1 kg/m^2, $p < 0.05$). The conclusion of the authors is that the regular consumption of nuts caused a lower than expected weight gain, although it was not significant after the adjustment for energy intake.

In 2008, Salas-Salvadó et al.[308] published the results obtained after a one year follow-up of the PREDIMED study. In this multicentre study 1,224 volunteers were randomly selected to receive three different dietary interventions: control (diet low in fat), Mediterranean diet supplemented with extra virgin olive oil or Mediterranean diet supplemented with nuts. This latter group showed a significant reduction in the prevalence of abdominal obesity in comparison with the control group.

Evidence

35. The addition of nuts to the usual diet is not associated with body weight gain (Evidence Level 2+).

Recommendations

14. A moderate intake of nuts is advantageous in the prevention of chronic diseases, but does not influence the body weight gain risk (Recommendation Degree C).

4.7. Miscellaneous

Meat and body weight

In the review of literature carried out in the section about animal protein (3.3) the descriptor "meat" and the heading "Animal protein" were included, so the review contains the evaluation of evidence associated with the consumption of meat or processed meat and weight gain.

Evidence

36. A high intake of meat and processed meat products might increase weight gain and the abdominal circumference (Evidence Level 2+).

Recommendations

15. A restriction of the intake of meat and processed meat products might prevent the body weight gain due to this factor (Recommendation Degree C).

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