Relationship between bread and obesity

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Abstract

Some studies have indicated that promoting the Mediterranean diet pattern as a model of healthy eating may help to prevent weight gain and the development of overweight/obesity. Bread consumption, which has been part of the traditional Mediterranean diet, has continued to decline in Spain and in the rest of the world, because the opinion of the general public is that bread fattens. The present study was conducted to assess whether or not eating patterns that include bread are associated with obesity and excess abdominal adiposity, both in the population at large or in subjects undergoing obesity management. The results of the present review indicate that reducing white bread, but not whole-grain bread, consumption within a Mediterranean-style food pattern setting is associated with lower gains in weight and abdominal fat. It appears that the different composition between whole-grain bread and white bread varies in its effect on body weight and abdominal fat. However, the term ‘whole-grain bread’ needs to be defined for use in epidemiological studies. Finally, additional studies employing traditional ways of bread production should analyse this effect on body-weight and metabolic regulation.

Key words: Mediterranean diet; Refined bread; Whole-grain bread; Obesity; Abdominal obesity

Several epidemiological studies investigating the relationship between diet and general or abdominal obesity have obtained inconsistent results(1).

The characteristics of the Mediterranean diet include the following: high consumption of olive oil; high consumption of legumes; high consumption of unrefined cereals (including bread); high consumption of fruits; high consumption of vegetables; moderate consumption of dairy products, mostly as cheese and yogurt; moderate to high consumption of fish; low consumption of meat and meat products; moderate consumption of wine(2,3). Some studies have indicated that promoting the Mediterranean diet pattern as a model of healthy eating may help to prevent weight gain and the development of obesity(4,5).

Within the cereal group, bread is an important dietary constituent from a nutritional point of view. However, a long-standing belief held by the general public is that bread fattens. This encourages many people to restrict or even eliminate bread from their diet. Thus, consumption of bread, which has been part of the traditional Spanish diet (the Mediterranean diet), has continued to fall in Spain and in the rest of the world(6). However, although bread consumption has been decreasing over the past decades, the global epidemic of obesity has been increasing(7).

Some studies have specifically investigated the associations between cereal consumption and BMI or abdominal fat. They showed inverse associations with anthropometric variables for consumption of whole-grain cereals, but yielded conflicting results for consumption of refined cereals(8). Factors such as postprandial insulin responses, gastric emptying after consuming a high-glycaemic index (GI) meal and

Abbreviations: GI, glycaemic index; PREvención con DIeta MEDiterránea; VAT, visceral adipose tissue; WC, waist circumference.

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other factors could be implicated in a potential differential effect of refined vs. whole-grain cereals on adiposity.

The present study was conducted to assess whether or not eating patterns that include bread as well as bread consumption itself (refined and whole-grain bread) were associated with overall obesity and abdominal adiposity, both in the general population and in subjects undergoing obesity management. Additionally, the objective of the present study was to address the relationship between bread consumption and changes in weight or waist circumference (WC) over time. Moreover, we considered it interesting to investigate whether bread consumption had been decreasing and if so, which food groups were being consumed in its place.

This knowledge would assist efforts in developing public health messages and recommendations regarding healthy eating habits that help individuals to maintain an appropriate BMI and to prevent abdominal obesity. Achieving better nutritional status for the general population would be an additional beneficial outcome.

### Scientific evidence

We have recently published a systematic review about the influence of bread intake on body weight and abdominal fat distribution. The literature search strategy was designed to identify original studies conducted on the association between bread intake and variations in ponderal status. The search was limited to English- or Spanish-language publications from a 30-year period (1978–2008). To identify publications in scientific journals, the search was conducted in MEDLINE and in the Spanish Medical Index (Indice Médico Español).

Articles identified in the initial search were eligible for inclusion if the following criteria were met: (1) the research

<table>
<thead>
<tr>
<th>First author and year</th>
<th>Differentiate bread type?</th>
<th>Study conclusions</th>
<th>Relationship</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tillotson et al. (1997)</td>
<td>Yes</td>
<td>Food group that included whole-meal bread was associated with a lower BMI and WHR; group that included refined bread was associated with a slight WHR increase</td>
<td>Group of food items that included whole-meal bread was beneficial</td>
</tr>
<tr>
<td>Jacobs et al. (1998)</td>
<td>No</td>
<td>No relationship was observed between bread and cereal intake and BMI</td>
<td>None</td>
</tr>
<tr>
<td>Greenwood et al. (2000)</td>
<td>Yes</td>
<td>Food group that included whole-meal bread was inversely related to BMI</td>
<td>Group of food items that included whole-meal bread was beneficial</td>
</tr>
<tr>
<td>Cleveland et al. (2000)</td>
<td>Yes</td>
<td>Whole-meal bread within the consumption of grains with fibre was associated with lower excess weight</td>
<td>Group of food items that included whole-meal bread was beneficial</td>
</tr>
<tr>
<td>Cho et al. (2003)</td>
<td>Yes</td>
<td>Food group that included bread at breakfast was associated with a lower BMI</td>
<td>Beneficial</td>
</tr>
<tr>
<td>Moreira &amp; Padrão (2006)</td>
<td>No</td>
<td>No relationship was observed between the food group that included bread and the risk of presenting with obesity</td>
<td>None</td>
</tr>
<tr>
<td>Schulz et al. (2002)</td>
<td>Yes</td>
<td>Food group that included whole-meal bread was predictive of avoiding weight gain</td>
<td>Beneficial</td>
</tr>
<tr>
<td>Liu et al. (2003)</td>
<td>Yes</td>
<td>Whole-meal bread included in the food group was associated with no weight gain over time; the opposite for refined bread</td>
<td>Food group that included whole-meal bread was beneficial, Food group that included refined bread was associated with weight gain</td>
</tr>
<tr>
<td>Halkjaer et al. (2004)</td>
<td>Yes</td>
<td>High intake of refined bread was associated with increased WC in females</td>
<td>Food group that included refined bread was associated with increased WC, No relationship was observed for the food group that included whole-grain bread</td>
</tr>
<tr>
<td>Togo et al. (2004)</td>
<td>Yes</td>
<td>Bread was not predictive of changes in ponderal status for development of obesity</td>
<td>None</td>
</tr>
<tr>
<td>Koh-Banerjee et al. (2004)</td>
<td>Yes</td>
<td>Whole-meal bread included in the food group was predictive (negatively associated) of ponderal gain; no influence was observed for refined bread</td>
<td>Food group that included whole-grain bread was beneficial, No relationship was observed for the food group that included refined bread</td>
</tr>
<tr>
<td>Halkjaer et al. (2006)</td>
<td>Yes</td>
<td>Refined bread was associated with WC gain in females. This association was not observed for whole-meal bread</td>
<td>Food group that included refined bread was associated with increased WC, No relationship was observed for the food group that included whole-grain bread</td>
</tr>
<tr>
<td>Stamler &amp; Dolecek (1997)</td>
<td>No</td>
<td>Conclusions from intervention studies</td>
<td>Beneficial</td>
</tr>
</tbody>
</table>

WC, waist circumference; WHR, waist:hip ratio.

**Table 1.** Classification of the most relevant studies segregated according to the influence of bread consumption on ponderal status.
We also analysed 2153 participants at a high risk of CVD who were included in the PREvencion con DIeta MEDiterranea (PREDIMED) trial to assess the association between changes in the consumption of certain foods and changes in weight and WC over time. Dietary habits were assessed with a validated FFQ. Seven prospective cohort studies were also included in this analysis.

Table 2. Changes in food group consumption according to the change in total bread consumption

Changes in total bread consumption

<table>
<thead>
<tr>
<th>Food groups</th>
<th>Baseline intake (g/d)</th>
<th>Changes at 4 years of follow-up (g/d)</th>
<th>Q1 (median – 98.72)</th>
<th>Q2–Q3 (median – 8.39)</th>
<th>Q4 (median 84.77)</th>
<th>P (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± Range</td>
<td>Mean ± Range</td>
<td>Mean ± Range</td>
<td></td>
</tr>
<tr>
<td>Legumes</td>
<td>18.9 ± 10.0</td>
<td>2.6 ± 11.8</td>
<td>4.4 ± 3.4 to 5.40</td>
<td>2.4 ± 1.7 to 3.08</td>
<td>1.1 ± 0.2 to 2.21</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Vegetables</td>
<td>32.8 ± 13.5</td>
<td>–2.9 ± 5.4</td>
<td>38.1 ± 25.2 to 50.50</td>
<td>–3.8 ± 12.9 to 5.29</td>
<td>–46.0 ± 58.4 to –33.47</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fruits</td>
<td>35.1 ± 18.7</td>
<td>5.3 ± 11.8</td>
<td>98.8 ± 79.6 to 117.78</td>
<td>48.7 ± 36.4 to 61.03</td>
<td>5.9 ± 26.2 to 25.42</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fish</td>
<td>101.2 ± 51.0</td>
<td>2.3 ± 53.8</td>
<td>14.8 ± 10.9 to 18.65</td>
<td>2.7 ± 0.17 to 5.62</td>
<td>–11.0 ± 16.62 to 5.58</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Meat</td>
<td>134.8 ± 50.9</td>
<td>–12.7 ± 54.0</td>
<td>1.3 ± 5.9 to 3.16</td>
<td>12.0 ± 14.91 to 9.09</td>
<td>–25.4 ± 30.42 to 20.40</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Olive oil</td>
<td>42.7 ± 16.7</td>
<td>7.0 ± 20.6</td>
<td>10.0 ± 8.33 to 11.80</td>
<td>7.7 ± 6.57 to 8.94</td>
<td>2.4 ± 0.66 to 4.15</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Nuts</td>
<td>11.0 ± 13.1</td>
<td>4.5 ± 17.7</td>
<td>7.5 ± 5.90 to 9.28</td>
<td>4.7 ± 3.84 to 5.69</td>
<td>0.8 ± 0.67 to 2.40</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Dairy products</td>
<td>384.4 ± 214.5</td>
<td>–17.0 ± 213.9</td>
<td>32.9 ± 14.56 to 51.23</td>
<td>13.4 ± 25.35 to 1.61</td>
<td>73.9 ± 92.29 to 55.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cereals*</td>
<td>20.4 ± 15.4</td>
<td>–0.7 ± 16.7</td>
<td>1.8 ± 0.54 ± 3.14</td>
<td>–1.0 ± 2.01 to –0.02</td>
<td>–3.0 ± 4.43 to –1.56</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Sweets</td>
<td>32.0 ± 30.6</td>
<td>–5.2 ± 33.0</td>
<td>3.3 ± 0.66 to 5.98</td>
<td>–5.5 ± 7.36 to –3.74</td>
<td>–13.1 ± 16.21 to –10.09</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Q, quartiles.

*Cereals excluding bread.
Table 3. Mean changes in weight and waist circumference according to the quartiles (Q) of change in bread consumption

<table>
<thead>
<tr>
<th>Q1</th>
<th>Q2</th>
<th>Q3</th>
<th>Q4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>Range</td>
<td>Mean</td>
<td>Range</td>
</tr>
<tr>
<td><strong>Change in total bread consumption</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$n$</td>
<td>553</td>
<td>554</td>
<td>553</td>
</tr>
<tr>
<td>Median</td>
<td>107·53</td>
<td>107·53</td>
<td>107·53</td>
</tr>
<tr>
<td>Weight change (kg)*</td>
<td>0·16</td>
<td>1·12 to 1·89</td>
<td>0·16</td>
</tr>
<tr>
<td>Waist change (cm)†</td>
<td>1·00</td>
<td>0·31 to 4·29</td>
<td>1·00</td>
</tr>
<tr>
<td><strong>Change in white bread consumption</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$n$</td>
<td>2</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Median</td>
<td>107·53</td>
<td>107·53</td>
<td>107·53</td>
</tr>
<tr>
<td>Weight change (kg)*</td>
<td>0·14</td>
<td>1·57 to 1·87</td>
<td>0·14</td>
</tr>
<tr>
<td>Waist change (cm)†</td>
<td>1·00</td>
<td>0·31 to 4·29</td>
<td>1·00</td>
</tr>
<tr>
<td><strong>Change in whole-grain bread consumption</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$n$</td>
<td>553</td>
<td>553</td>
<td>554</td>
</tr>
<tr>
<td>Median</td>
<td>55·43</td>
<td>55·43</td>
<td>55·43</td>
</tr>
<tr>
<td>Weight change (kg)*</td>
<td>0·75</td>
<td>0·98 to 2·48</td>
<td>0·75</td>
</tr>
<tr>
<td>Waist change (cm)†</td>
<td>1·00</td>
<td>0·31 to 4·29</td>
<td>1·00</td>
</tr>
</tbody>
</table>

*Multivariate means were calculated using generalised linear models. The means were adjusted for age, sex, intervention group, weight at baseline, change in energy-adjusted white and whole-grain bread consumption; (2) change in body weight after 4 years of follow-up; (3) change in WC after 4 years of follow-up; (4) risk of gaining or losing more than 2 kg of weight; (5) risk of gaining or losing more than 2 cm of WC.

The outcomes after 4 years of follow-up were: (1) changes in food group consumption according to the change in total bread consumption; (2) change in body weight after 4 years of follow-up; (3) change in WC after 4 years of follow-up; (4) risk of gaining or losing more than 2 kg of weight; (5) risk of gaining or losing more than 2 cm of WC.

In general, increases in total bread consumption were associated with more weight gain (mean weight change of 0·16 kg after 4 years in the lowest quartile and of 0·82 kg in the highest quartile, $P$ for trend = 0·019) and with more WC gain (mean waist change of 1 cm after 4 years in the lowest quartile and of 2·34 cm in the highest quartile, $P$ for trend < 0·001). For white bread, the results were very similar for weight (mean weight change of 0·14 kg after the follow-up period in the lowest quartile and of 0·90 kg in the highest quartile, $P$ for trend = 0·003) and for WC gain (mean waist change of 1·11 cm in the lowest quartile and of 2·39 cm in the highest quartile, $P$ for trend < 0·001). No significant dose–response relationship was observed for the change in whole-bread consumption and anthropometric measures. The adjustment for total dietary fibre intake had little effect on these results.

Finally, changes in the consumption of any type of bread during 4 years of follow-up were not associated with the risk of gaining more than 2 cm in WC or more than 2 kg in weight among the PREDIMED participants. When compared with the subjects who were in the lowest quartile of change at baseline and repeatedly every year during 4 years of follow-up. Using multivariate models to adjust for covariates, long-term changes in weight and WC according to the quartiles of change in energy-adjusted white and whole-grain bread consumption were calculated. The PREDIMED trial is the first large randomised controlled trial for the primary prevention of CVD that allocates participants to one of three dietary patterns. These consisted of two Mediterranean type diets (Med-diet) with different fat sources, mixed nuts or virgin olive oil, and one low-fat diet (control group). The study population was composed of men aged between 55 and 80 years and women aged between 60 and 80 years with no previously documented CVD, but at a high risk of CVD.
in white bread consumption, those in the highest quartile showed a significant reduction in the odds of losing weight (>2 kg) and WC (>2 cm) of 33 and 36%, respectively. Moreover, a significant inverse dose–response relationship was found for the increment in the consumption of white bread and the probability of losing weight (P for trend=0.021) and WC (P for trend=0.009).

**Summary of scientific evidence for the influence of bread consumption on general and abdominal obesity**

**Systematic review**

1. The majority of studies following a food pattern that included bread were not associated with an increase in ponderal status.
2. Consumption of whole-grain bread was more beneficial than refined bread, especially in relation to abdominal fat.
   - (a) Whole-grain bread: does not influence weight gain.
   - (b) White bread: possible relationship with excess abdominal fat.

**PREvención con Dieta MEDiterránea study**

1. The results showed that over 4 years, participants in the highest quartile of the change in white bread intake gained 0.76 kg more in weight than those in the lowest quartile and 1.28 cm more in WC than those in the lowest quartile.
2. No significant dose–response relationships were observed for the change in whole-bread consumption and anthropometric measures.
3. Gaining weight (>2 kg) and gaining WC (>2 cm) during the follow-up was not associated with an increase in bread consumption. However, participants in the highest quartile of changes in white bread intake had a 33% reduction in the odds of losing weight (>2 kg) and a 36% reduction in the odds of reducing WC (>2 cm).

**Hypotheses on the mechanism of action whereby bread consumption influences general and abdominal obesity**

We do not know with precision what the mechanism is as whole-grain bread could prevent increased WC and body weight, and a dietary pattern low in refined bread might help to prevent body-weight increase and abdominal fat accumulation.

The possible mechanisms involved in the action of whole-grains (including bread) are:

1. **Energy density**
2. **GI**
3. **Dietary fibre**
4. **Gut microbiota.**

**Energy density**

The lower energy density of products based on whole-grain cereals compared with those made with refined cereals, as well as the satiating effect of whole-grain products, could both play an important role in body-weight regulation. In the case of bread intake, although both types of bread (whole-grain bread and refined bread) have similar energy content, whole-grain bread has the greatest satiating power. This may influence the decreased energy intake observed from other foods.

**Glycaemic index**

Lower plasma glucose and insulin responses to whole-grain cereal intake can also contribute to body-weight regulation. Lower plasma glucose and insulin responses have been observed in diabetic and non-diabetic subjects after the ingestion of a low-GI diet containing pumpernickel bread, pasta and legumes, compared with a high-GI diet containing refined bread and potatoes. In fact, several studies have shown that the consumption of foods or meals with a low GI has a higher satiating effect than those with a high GI, irrespective of the evaluation method utilised (direct or indirect) and the possible contribution of some confounders (palatability and fibre content). The lower rate of nutrient digestion and absorption typical of low-GI foods seems to stimulate the release of gastrointestinal peptides related to satiation and satiety signals. Therefore, the intact food structure that accounts for the lower GI of whole-grain cereals can contribute to body-weight regulation.

Du et al. carried out a prospective cohort study in 89,432 Europeans aged between 20 and 78 years, who were monitored for an average of 6.5 years to assess the effect of the GI and glycaemic load on body weight and WC. The study did not find an effect on the change in body weight. The GI (but not the glycaemic load) was moderately associated with a larger WC. McKeown et al. conducted a study in a sample of the Framingham Heart Study cohort, and they found that a higher intake of whole-grain foods was associated with lower visceral adipose tissue (VAT) in adults, whereas a higher intake of refined grains was associated with higher VAT. In this study, fasting insulin concentrations were observed to attenuate the associations between refined grain intake, but not whole grain intake, and VAT volume, perhaps suggesting an intermediary role of insulin in the positive relationship between refined grain intake and VAT volume. The additional adjustment for insulin did not affect the relationship observed between whole grain intake and VAT.

Finally, Giacco et al., 2013, designed an intervention study evaluating glucose and insulin metabolism in response to long-term consumption of rye and whole wheat compared with a diet containing the same amount of refined cereal foods in individuals with the metabolic syndrome from two European locations (Kuopio, Finland/Naples, Italy). Overall, 146 participants were assigned to a diet based on whole-grain (whole-grain group) or on refined cereal products (control group), each lasting for a duration of 12 weeks. At the end.
of the intervention, insulin sensitivity indices and secretion did not change significantly in the whole-grain and control groups when compared with baseline, and no differences between the two groups were observed.

Dietary fibre

The potential mechanism or mechanisms by which whole grains may be related to regional adiposity are speculative. Whole grains are rich in fermentable carbohydrates such as dietary fibre, resistant starch and oligosaccharides. Cereal fibre influences body weight by multiple mechanisms depending on intrinsic properties, hormonal effects and intestinal fermentation. Specifically, intrinsic properties concern the ability of soluble fibre to bind to water and form a viscous solution that delays gastric emptying and intestinal transit, and limits glucose absorption, thus leading to a lower blood glucose response \(^{(35)}\). The hormonal effects of fibre are mediated by insulin and gastrointestinal hormones. Fibre decreases insulin secretion and, consequently, reduces the risk of reactive hypoglycaemia during the post-absorption period, thus promoting satiety and satiation, increasing fat oxidation and decreasing fat storage. Fibre also influences gut hormone secretion that, independently of plasma glucose response, acts on satiety or modifies glucose homeostasis. Cholecystokinin, secreted by small-bowel cells, stimulates pancreatic secretion, modulates gastric emptying and stimulates the hypothalamic centre of satiety \(^{(36)}\).

In the previously mentioned study of McKeown et al. \(^{(33)}\), added bran and dietary fibre was also adjusted for in the analysis. The association between higher intake of whole-grain foods with lower VAT and higher intake of refined grains with higher VAT persisted, suggesting that these were not the mediating attributes of the diet related to body fat distribution.

We carried out a previously mentioned study \(^{(25)}\) in a sample of PREDIMED subjects evaluating the influence of bread intake and WC. We did not find any differences in the relationship between higher intakes of refined bread and increases in WC, adjusting for dietary fibre.

Gut microbiota

A further mechanism by which whole grains may influence body-weight regulation is via a prebiotic effect modulating the intestinal flora. Available evidence, primarily from investigations in animal models, suggests that the gut microbiota affects nutrient acquisition and energy regulation. Microbiota composition has also been shown to differ in lean vs. obese animals and human subjects \(^{(37)}\). Among the possible mechanisms of this relationship, of particular interest is the hypothesis that the metabolic activities of the gut microbiota facilitate the extraction of energy from ingested dietary substances and help store this energy in host adipose tissue for later use. In fact, gut bacterial flora of obese mice and humans include fewer Bacteroidetes and correspondingly more Firmicutes than that of their lean counterparts, suggesting that differences in energy extraction of ingested food substances may be due to the composition of gut microbiota \(^{(37)}\).

In humans, however, it is not known whether whole grains could influence body fat distribution through an effect on gut incretin hormones. Nonetheless, available data in human subjects show that a diet rich in whole-wheat cereals compared with a diet based on bran wheat is able to increase the number of faecal bifidobacteria and lactobacilli, the target genera for prebiotic intake \(^{(38)}\).

Conclusions

(1) The different composition of whole-grain bread and white bread shows inconsistent results regarding its influence on body weight and abdominal fat.

(2) Reducing white bread, but not whole-grain bread, consumption within a Mediterranean-style food pattern setting is associated with lower gains in weight and abdominal fat. However, a definition of whole-grain bread is needed for application in epidemiological studies.

(3) Additional studies using traditional ways of bread production should analyse this effect on weight and metabolic regulation.

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The authors’ contributions are as follows: L. S.-M. and I. B.-C. prepared the manuscript and wrote the paper with important input and feedback between them. Both authors read and approved the final version of the manuscript.

Neither of the authors has any conflict of interest to declare.

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