Short Communication

Preliminary data about the influence of vitamin D status on the loss of body fat in young overweight/obese women following two types of hypocaloric diet

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The loss of weight was analysed in a group of sixty overweight/obese women of childbearing age (20–35 years) according to their initial vitamin D status. Subjects were randomly assigned to one of two slightly hypocaloric diets: Diet V, in which the consumption of vegetables was increased, or Diet C, in which the relative consumption of cereals (especially breakfast cereals) was increased. Dietetic, anthropometric and biochemical data were collected at the start of the study and again at 2 weeks after dividing the women into groups depending on their having an initial serum 25-hydroxyvitamin D (25(OH)D) concentration of $< 50$ nmol/l (LD) or $\geq 50$ nmol/l (HD). Dietary intervention led to a reduction in energy intake, body weight and BMI in all groups. The HD women showed greater body fat losses during the study than the LD women (1·7 (SD 1·8) kg compared to 0·5 (SD 0·8) kg). A better vitamin D status therefore aided the loss of body fat over the experimental period (OR 0·462; CI 0·271, 0·785; $P$ = 0·001). However, when the dietary groups were analysed separately, this effect was only seen in the C subjects (OR 0·300; CI 0·121, 0·748; $P$ = 0·001). The present results suggest that women with a better vitamin D status respond more positively to hypocaloric diets and lose more body fat; this was especially clear among the C subjects who had a greater vitamin D supply during the experimental period.

Vitamin D: Weight loss: Overweight: Obesity: Young women

Vitamin D is essential for the regulation of serum calcium, phosphate and alkaline phosphatase, and thus helps to produce and maintain bone(1–3). It has also been associated with the prevention of certain cancers, diabetes mellitus, autoimmune disorders, CVD and osteoporosis(1,4). Different studies have observed that there is an inverse correlation between vitamin D and BMI and fat mass(5,6). It could be explained because an adequate vitamin D status results in lower blood parathyroid hormone, diminishing calcium influx into the adipocytes and increasing lipolysis(5). An improvement of vitamin D status, especially when trying to lose weight, may contribute to regulating the accumulation of fat(3–7).

The aim of the present work was to analyse the repercussion of vitamin D status on the loss of body fat in young overweight/obese women group following two slightly hypocaloric diets.

Materials and methods

Study subjects

The study subjects were sixty women aged 20–35 years. According to the requirements of the Ethics Committee of the Faculty of Pharmacy, all subjects signed a witnessed form of consent to be included.

The study was performed during the winter (November to March).

Interventions

The experimental diets to which the subjects were randomly assigned were designed to provide a mean of approximately 20 % less than their theoretical energy requirements. Theoretical energy expenditure was established by taking into account the age, body weight and physical activity(8) of all subjects, using equations proposed by the WHO(9). Both diets were structured with the idea of approximating them to the theoretical ideal by increasing the relative consumption of either vegetables or cereals; earlier studies have shown that these foods are those with the greatest differences between their observed and recommended intakes(10,11).

The weight control measures were based on restricting the consumption of energy-rich foods and increasing the consumption of cereals (Diet C) or vegetables (Diet V).

Abbreviations: Diet C, increased consumption of breakfast cereals; Diet V, increased consumption of vegetables; HD group, initial serum 25(OH)D concentration $\geq 50$ nmol/l; LD group, initial serum 25(OH)D concentration $< 50$ nmol/l; 25(OH)D, 25-hydroxyvitamin D.

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Diet C. Enriched-breakfast cereals and cereal bars were particularly recommended (a minimum of three times/d) since, apart from carbohydrate, they also provide fibre, minerals and vitamins (particularly vitamin D). However, the subjects were also advised to eat other cereals, e.g. bread, rice and pasta.

Diet V. Increasing the intake of vegetables (minimum three times/d) was recommended.

The women were allowed to create their own menus following the established rules. The full characteristics of the diets followed and other methodological details are described elsewhere.[12]

Methods

The following data were collected from all subjects during the pre-intervention stage and again at 2 weeks.

Anthropometric information. Weight and height were determined using a Seca Alpha digital electronic balance (range 0·1–150 kg) and a Harpenden digital stadiometer (range 70–205 cm), respectively. A pair of Holtain skinfold calipers was used to measure the biceps, triceps, subscapular and suprailiac skinfold thickness. For these measurements, subjects were barefoot and wore only underwear. All data were collected by trained personnel following norms set out by the WHO[13].

Subject BMI was calculated as weight (kg)/height$^2$ (m$^2$). The percentage of body fat (%BF) was determined from the body density using the equation of Siri[14]:

\[ BF = \frac{495}{\text{body density}} - 450. \]

Body density was calculated from the formulae of Durnin & Womersley[15]:

\[
\text{body density} = 1.1567 - 0.0717 \times \log(\text{sum of skin fold thicknesses: biceps + triceps + subscapular + suprailiac}).
\]

Using the value for %BF and subject body weight, the following were calculated:

Fat mass (FM) (kg) = %BF × body weight (kg)/100

Fat-free mass (kg) = body weight – FM.

Dietetic study. A ‘food and drink record’ was used to register all intakes for 3 d, including a Sunday[16]. The aim was to have as true a record as possible; subjects were asked to record all intakes, even though they broke the ‘rules’ of their diet.

The energy and nutrient contents of these foods were then determined using food composition tables[17]. Special attention was paid to the intake of energy and vitamin D. DIAL software (Alce Ingenierı́a, 2004) was used to process all data[18].

Serum analysis. 25-Hydroxyvitamin D (25(OH)D) levels were measured by RIA using the IDS RIA kit (IDS Ltd, UK) (CV 7·4%)[19]. The limit established by Gordon et al.[20] (50 nmol/l) was used in the present study to consider mild vitamin D deficiency.

Statistical analysis

Means and standard deviations were calculated for all variables. Student’s t test for paired samples was used to analyse the change in variables over time in each diet group. The comparison of group C and group V results was performed using the Student’s t test (or the Mann–Whitney test if the distribution of results was not homogeneous). Linear correlation coefficients were calculated using the Pearson test. Comparisons between proportions were performed using the $\chi^2$ test. Logistic regression analysis was used to identify risk or protection factors that might modify any variables. Significance was set at $P<0.05$.

Results

Tables 1 and 2 show the initial results and those recorded at 2 weeks for the women who followed the C and V diets, and with respect to whether they had initial serum 25(OH)D concentrations of $<50$ nmol/l (LD) or $\geq 50$ nmol/l (HD).

The C subjects came to have a higher cereal intake than the V subjects, and the V subjects to have a higher vegetable intake than they showed at the beginning of the experiment. The intake of fruits increased and the consumption of meat, fish and eggs fell over the study period (Table 1).

Vitamin D intake and 25(OH)D levels improved in the C women – both those of the LD and HD subgroups. It is reasonable that serum 25(OH)D levels should rise with vitamin D intake (Table 2).

The body weight and BMI of the subjects in all the subgroups fell since the diets reduced the energy intake. Body fat was reduced in three of the four subgroups. This reduction was significantly greater among HD women, and especially among those who followed Diet C; these subjects also enjoyed a significant increase in their fat-free mass (Table 2).

Taking all subjects together, the HD women showed greater losses of body fat at 2 weeks than LD women (1·7 (SD 1·8) kg compared to 0·5 (SD 0·8) kg). It would therefore appear that having better vitamin D status aids in the loss of body fat (OR 0·462; CI 0·271, 0·785; $P<0.001$). However, when the same analyses were performed for V and C subjects separately, only in HD–C subjects was the aid maintained (OR 0·300; CI 0·121, 0·748; $P<0.001$).

Discussion

Bearing in mind that the initial intake of cereals/legumes, vegetables/greens and fruits was lower than recommended in all subjects[10–12], the design of the two slightly hypocaloric intervention diets, based on approximating the intakes of cereals or vegetables to the theoretical ideal, is justified. One of the interventions, the V diet, was more conventional, while the other, the C diet, was more unusual, allowing the differences in the effect of extra vitamin D intake to be examined.

The initial vitamin D intake and serum 25(OH)D levels of the subjects were similar to those recorded in other studies[21,22], and, like these, showed that many women have an inadequate vitamin D status[1,21,22] (Table 2).

The diets followed led to a reduction in energy intake, body weight, BMI and body fat (Tables 1 and 2), which agrees with results reported in other studies[12,23]. The C diet led to an increase in vitamin D intake and an increase in serum 25(OH)D (Tables 1 and 2), and a correlation was found at the end of the study between cereals and vitamin D intake...
Corporal fat loss since start (kg): 0.4, 0.5, 0.6, 1.1, 0.5, 0.8, 0.7, 1.2, 2.7†‡, 1.8, 1.7‡, 1.8.

Mean values were significantly different from those of the baseline (pre-intervention): *, Diet C, increased consumption of breakfast cereals; Diet V, increased consumption of vegetables, FFM, fat-free mass.

Table 1. Changes in food composition, energy and vitamin D intake over the dietary intervention period: differences depending on initial serum 25-hydroxyvitamin D (25(OH)D) concentrations

<table>
<thead>
<tr>
<th></th>
<th>Initial 25(OH)D concentration &lt; 50 nmol/l</th>
<th></th>
<th>Initial 25(OH)D concentration ≥ 50 nmol/l</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Diet V (n 17)</td>
<td>Diet C (n 14)</td>
<td>Total (n 31)</td>
<td>Diet V (n 14)</td>
</tr>
<tr>
<td></td>
<td>Mean SD</td>
<td>Mean SD</td>
<td>Mean SD</td>
<td>Mean SD</td>
</tr>
<tr>
<td>Cereals and pulses (servings/d)</td>
<td>4.6 ± 1.4</td>
<td>5.5 ± 1.7</td>
<td>1.7 ± 1.0</td>
<td>5.1 ± 1.6</td>
</tr>
<tr>
<td>Vegetables (servings/d)</td>
<td>2.9 ± 1.3</td>
<td>3.1 ± 1.6</td>
<td>1.6 ± 1.5</td>
<td>3.0 ± 1.5</td>
</tr>
<tr>
<td>Fruits (servings/d)</td>
<td>1.1 ± 0.8</td>
<td>1.5 ± 0.9</td>
<td>0.9 ± 0.9</td>
<td>1.3 ± 0.9</td>
</tr>
<tr>
<td>Milk products (servings/d)</td>
<td>1.7 ± 0.9</td>
<td>2.1 ± 0.8</td>
<td>0.8 ± 0.9</td>
<td>1.9 ± 0.9</td>
</tr>
<tr>
<td>Meat, fish and eggs (servings/d)</td>
<td>4.3 ± 1.8</td>
<td>4.3 ± 1.9</td>
<td>1.9 ± 1.8</td>
<td>4.3 ± 1.8</td>
</tr>
<tr>
<td>Energy (kJ/d)</td>
<td>9434 ± 2428</td>
<td>9901 ± 2401</td>
<td>2401 ± 9645</td>
<td>2387 ± 8232</td>
</tr>
<tr>
<td>Vitamin D (µg/d)</td>
<td>3.1 ± 2.1</td>
<td>2.8 ± 1.8</td>
<td>1.8 ± 1.5</td>
<td>3.0 ± 1.9</td>
</tr>
<tr>
<td></td>
<td>Final data</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cereals and pulses (servings/d)</td>
<td>3.6 ± 1.2</td>
<td>5.3‡ ± 0.6</td>
<td>4.4* ± 1.3</td>
<td>4.7 ± 1.3</td>
</tr>
<tr>
<td>Vegetables (servings/d)</td>
<td>5.12* ± 1.6</td>
<td>4.0 ± 1.9</td>
<td>1.9 ± 1.9</td>
<td>4.6* ± 1.8</td>
</tr>
<tr>
<td>Fruits (servings/d)</td>
<td>2.7* ± 1.1</td>
<td>4.3* ± 1.9</td>
<td>1.6 ± 1.2</td>
<td>4.7* ± 1.2</td>
</tr>
<tr>
<td>Milk products (servings/d)</td>
<td>2.0 ± 0.6</td>
<td>1.9 ± 0.8</td>
<td>0.9 ± 0.7</td>
<td>1.9 ± 0.7</td>
</tr>
<tr>
<td>Meat, fish and eggs (servings/d)</td>
<td>3.0* ± 1.5</td>
<td>2.1* ± 1.0</td>
<td>1.0 ± 1.4</td>
<td>2.6* ± 1.4</td>
</tr>
<tr>
<td>Energy (kJ/d)</td>
<td>6742* ± 754</td>
<td>6813* ± 1322</td>
<td>1322 ± 6774</td>
<td>1030 ± 6246</td>
</tr>
<tr>
<td>Vitamin D (µg/d)</td>
<td>3.2 ± 2.4</td>
<td>6.0‡ ± 1.2</td>
<td>4.5* ± 2.4</td>
<td>4.6 ± 2.6</td>
</tr>
</tbody>
</table>

Diet C, increased consumption of breakfast cereals; Diet V, increased consumption of vegetables. Mean values were significantly different from those of the baseline (pre-intervention): *P < 0.05. Mean values were significantly different from those of the Diet V group: †P < 0.05. Mean values were significantly different from those of the women with an initial serum 25(OH)D concentration < 50 nmol/l: ‡P < 0.05.

The importance of vitamin D in weight control(3,5,6,24,25) is shown in the greater loss of body fat in the HD women. Vitamin D therefore appears to aid in the loss of body fat.

The reduction in energy intake in HD women (2912 (SD 2043) kJ) was different to that seen in the LD women (2871 (SD 2379) kJ). Therefore, it cannot be the cause of their greater body weight and fat losses (Table 2). Similarly, the HD women following the C diet lost more body fat than their LD counterparts yet there was no significant difference in the reduction of their energy intakes (3776 (SD 2144) kJ in HD–C subjects; 3088 (SD 2444) kJ in LD–C subjects).

Recent evidence suggests that vitamin D intakes above current recommendations may be associated with better health(1,4), and that the vitamin D status of the general population could be improved(21,22). Strategies to increase vitamin D intake, including the fortification of food, should

Table 2. Changes in anthropometric and biochemical data over the dietary intervention period: differences depending on initial serum 25-hydroxyvitamin D (25(OH)D) concentrations

<table>
<thead>
<tr>
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<th>Initial 25(OH)D concentration &lt; 50 nmol/l</th>
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<td></td>
<td>Mean SD</td>
<td>Mean SD</td>
<td>Mean SD</td>
<td>Mean SD</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72.3 ± 8.4</td>
<td>78.1 ± 12.3</td>
<td>74.9 ± 10.6</td>
<td>75.6 ± 9.0</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.1 ± 3.4</td>
<td>28.3 ± 3.8</td>
<td>28.2 ± 3.5</td>
<td>28.4 ± 3.2</td>
</tr>
<tr>
<td>BMI &gt; 30 kg/m² (%)</td>
<td>29.4</td>
<td>28.6</td>
<td>29.0</td>
<td>33.3</td>
</tr>
<tr>
<td>Fat (kg)</td>
<td>4.9 ± 2.9</td>
<td>29.3 ± 1.4</td>
<td>28.3 ± 5.6</td>
<td>4.3 ± 2.3</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td>44.8</td>
<td>48.8* ± 6.2</td>
<td>46.6 ± 5.4</td>
<td>46.2 ± 4.6</td>
</tr>
<tr>
<td>Serum 25(OH)D (nmol/l)</td>
<td>30.5 ± 12.0</td>
<td>37.5 ± 8.1</td>
<td>33.7 ± 10.8</td>
<td>50.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75.9* ± 8.2</td>
<td>76.6* ± 12.4</td>
<td>73.8* ± 10.6</td>
<td>73.4* ± 8.7</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.9* ± 3.4</td>
<td>27.7* ± 3.9</td>
<td>27.8* ± 3.6</td>
<td>27.9* ± 3.0</td>
</tr>
<tr>
<td>BMI &gt; 30 kg/m² (%)</td>
<td>29.4</td>
<td>28.6</td>
<td>29.0</td>
<td>26.7</td>
</tr>
<tr>
<td>Fat (kg)</td>
<td>27.1* ± 5.0</td>
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<td>44.4* ± 4.0</td>
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<td>46.0* ± 5.3</td>
<td>47.2* ± 5.0</td>
</tr>
<tr>
<td>Serum 25(OH)D (nmol/l)</td>
<td>32.9 ± 8.9</td>
<td>43.7* ± 12.3</td>
<td>38.0 ± 12.3</td>
<td>40.0* ± 45.5</td>
</tr>
<tr>
<td>Corporal fat loss since start (kg)</td>
<td>0.4 ± 0.5</td>
<td>0.6 ± 1.1</td>
<td>0.5 ± 0.8</td>
<td>0.7 ± 1.2</td>
</tr>
</tbody>
</table>

Diet C, increased consumption of breakfast cereals; Diet V, increased consumption of vegetables, FFM, fat-free mass. Mean values were significantly different from those of the baseline (pre-intervention): *P < 0.05. Mean values were significantly different from those of the Diet V group: †P < 0.05. Mean values were significantly different from those of the women with an initial serum 25(OH)D concentration < 50 nmol/l: ‡P < 0.05.
Acknowledgements

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References