OBJECTIVE: To evaluate weight gain and its relation to anxiety in a group of smokers after 3 months of cessation treatment.

PATIENTS AND METHODS: The target population for this prospective, analytical, longitudinal study was smokers being treated in a specialist smoking cessation clinic who were still abstinent at the conclusion of a 3-month treatment program. The following variables were analyzed: age, sex, nicotine dependence (Fagerström test), daily cigarette consumption, number of pack-years, pharmacological treatment (nicotine replacement/bupropion), use of nicotine gum (yes/no), weight gain, body mass index, and degree of state and trait anxiety. Successful cessation was defined as self-reported abstinence at the conclusion of a 3-month treatment program. Results for the quantitative variables were expressed as means (SD), and results for the qualitative variables were expressed as percentages and absolute frequencies.

RESULTS: The study population consisted of 122 individuals, 76 of whom were men (62%) and 46 of whom were women (38%). The mean age was 45.9 (9.9) years, and mean nicotine dependence according to the Fagerström scale was 6.2 (2.2) points. Average weight gain was 2.6 kg (3.6%), with no significant difference between the sexes. Weight gain in 25% of this population was greater than 4.2 kg, and maximum weight gain was 9.2 kg. Levels of state anxiety fell progressively as weight increased, although there was no evident relationship between the 2 variables.

CONCLUSIONS: Weight gain is moderate as smokers quit. Anxiety levels, which are greater in the first few weeks after cessation, do not explain weight variation, which is more related to the metabolic effects of nicotine rather than to psychological variables.

Key words: Smoking cessation. Weight gain. Nicotine. Anxiety. Weight. Body mass index.

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Introduction

Nicotine, which is the psychoactive substance responsible for smoking addiction, has a number of effects on the human organism.1 When nicotine is...
inhaled with cigarette smoke, it takes 7 to 10 seconds to reach the brain, where it produces direct effects and releases a variety of neurotransmitters. As a psychoactive substance, nicotine alters the functioning of the central nervous system by producing perceptible mood, cognitive, affective, and behavioral changes. Since these changes are perceived by the smoker as beneficial, they contribute to developing and maintaining nicotine addiction.

Nicotine effects other than those on the central nervous system have been described that include cardiovascular, endocrine and metabolic effects, which occur as a consequence of nicotine acting on endocrine and neuroendocrine system components, such as catecholamines, serotonin, and glucocorticosteroids.

Unrelated to the effects on the central nervous system, however, many of these changes are not perceived by the smoker.

A widely held belief in regard to the effects of smoking is that it helps keep body weight under control. A number of studies demonstrate that smokers weigh less than nonsmokers and that giving up smoking results in weight gain. That said, the mechanism by which weight is increased is not fully understood. The withdrawal syndrome resulting from nicotine suppression is characterized by both an increase in appetite and the development of psychological symptoms (anxiety and irritability). However, it is quite possible that the psychological symptoms contribute indirectly to weight gain in ex-smokers.

Anxiety is a negative emotion which, like any other, affects at least 3 response components or systems, namely the subjective (or cognitive), motor (or behavioral), and physiological (or somatic) systems. The most characteristic physiological response is an increase in activity levels in the autonomic nervous system that are reflected in both external and internal changes. These changes may all lead to increased food intake, which may or may not be provoked by a subjective feeling of hunger. The outcome is weight gain, which often reflects a state of anxiety.

Our aim was to evaluate weight gain and to assess its relationship with anxiety levels in a group of smokers after 3 months of cessation treatment.

**Patients and Methods**

The target population for this prospective, analytical, longitudinal study was smokers being treated in a specialist smoking cessation clinic who were still abstinent at the conclusion of a 3-month treatment program. Multicomponent treatment consisting of medication and 9 sessions of cognitive-behavioral group therapy was implemented over a period of 3 months. Individuals admitted to the cessation program were smokers who requested the treatment and who agreed in writing to comply with the program conditions. Excluded were smokers with a severe psychiatric disorder, smokers who were dependent on other drugs, and/or smokers who were pregnant. Prior to commencing the treatment program, the smoking-related medical histories of the participants were recorded, and psychological treatment—bupropion, or nicotine replacement therapy (NRT) in the form of patches—was decided. Patients were also recommended to use nicotine gum at will.

The following variables were analyzed: age, sex, nicotine dependence (measured with the Fagerström test), daily cigarette consumption, number of pack-years, pharmacological treatment (bupropion or NRT), nicotine gum use (yes/no), weight gain (expressed in kilograms and as a percentage), body mass index (BMI), and degree of state and trait anxiety.

A sustained abstinence criterion was applied from the outset of treatment. As in similar studies, this was evaluated by means of patient self-reported abstinence and confirmed by expired carbon monoxide (CO) measured at each follow-up visit. A patient was considered to no longer smoke when expired CO measured with a CO-oximeter (Mini Smokerlyzer, Bedfont Scientific Ltd., Rochester, UK) was less than or equal to 10 parts per million (ppm). Weight was recorded in the same conditions for all patients (dressed and barefoot), using a Seca digital electronic scale. BMI was calculated by measuring height and applying the standard formula (weight in kg divided by height in m^2). Applying current recommendations for the Spanish population, we established 4 BMI weight categories (underweight, normal weight, overweight, and obese).

Anxiety was evaluated using the Spielberg State–Trait Anxiety Inventory questionnaire adapted for use in Spain; this instrument consists of self-appraisal scales that measure 2 independent conceptual aspects of anxiety: state anxiety (20 items) and trait anxiety (20 items). As in similar studies, this was evaluated at the outset of treatment.

State anxiety and weight were measured on 5 occasions during the treatment program: before smoking cessation, and at the end of week 1 (week 2 in the case of weight), month 1, month 2 and month 3 after cessation. Weight was measured after 2 weeks because weight gain after just 1 week was likely to be minimal.

**Statistical Analysis**

Descriptive statistics and other analyses were performed with version 11.5 of the Statistical Package for Social Sciences (SPSS, Chicago, Illinois, USA) for Windows. Results were expressed as means (SD) for quantitative variables and as absolute frequencies and percentages for the qualitative variables. The Pearson χ² test was used to analyze relationships between qualitative variables. To identify possible relationships between weight gain and the remaining variables, the Student t test was used to compare means for qualitative variables, and dispersion diagrams were used to compare quantitative variables.

Finally, analysis of variance (ANOVA) was conducted, using the Tukey test for post hoc multiple comparisons, in order to evaluate the relationship between weight gain and the Fagerström test variables, namely, time to the first cigarette of the day and number of cigarettes smoked a day. Results were represented in the form of an error bar graph for a 95% level of confidence. All the tests were considered statistically significant at P≤.05.

**Results**

The study population was composed of 122 individuals: 76 men (62%) and 46 women (38%). All subjects were still abstinent 3 months after giving up smoking. The mean (SD) age of patients was 43.9 (9.9) years: men 43.9 (9.8) years, and women 44 (10.2)
years. Table 1 summarises sample characteristics according to sex, age, smoking habits (including expired CO level), Fagerström test scores, and trait anxiety. The means comparison revealed significant differences between men and women for number of cigarettes smoked a day \( (P=0.026) \), number of pack-years \( (P=0.036) \), trait anxiety \( (P=0.001) \), and expired CO level \( (P=0.025) \).

Table 2 describes the pharmacological treatment program undertaken (bupropion or NRT) in terms of frequencies and percentages for the entire sample and for men and women. Nicotine gum was used by almost three quarters of the population.

Table 3 shows weight gain (expressed in kilograms and as a percentage) for the entire study population and for men and women. There were no significant weight gain differences between men and women \( (P=0.94 \text{ for kilograms gained}, \text{ and } P=0.22 \text{ for percentage gain}) \). Only

\[
\begin{align*}
\text{Kilograms} & : 2.6 (2.4) & 2.6 (2.2) & 2.6 (2.6) \\
\text{Percentage} & : 3.6 (3.3) & 3.2 (2.8) & 4.1 (4.0)
\end{align*}
\]

\*Values are expressed as mean (SD).

\*BMI indicates body mass index.

Figure 1. Classification of patients in body mass index (BMI) categories.

### Table 1
**Quantitative Variables for the Sample and by Sex**

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>43.9 (9.9)</td>
<td>43.9 (9.8)</td>
<td>44.0 (10.2)</td>
</tr>
<tr>
<td>Fagerström test</td>
<td>6.2 (2.2)</td>
<td>6.4 (2.1)</td>
<td>5.8 (2.3)</td>
</tr>
<tr>
<td>Cigarettes/d†</td>
<td>26.1 (10.2)</td>
<td>27.3 (10.7)</td>
<td>23.5 (8.8)</td>
</tr>
<tr>
<td>Pack-years†</td>
<td>35.1 (18.7)</td>
<td>37.9 (19.4)</td>
<td>30.6 (16.7)</td>
</tr>
<tr>
<td>Trait anxiety†</td>
<td>20.6 (9.5)</td>
<td>18.5 (8.9)</td>
<td>24.1 (9.4)</td>
</tr>
<tr>
<td>Carbon monoxide, ppm</td>
<td>25.2 (14.4)</td>
<td>27.5 (15.1)</td>
<td>21.4 (12.3)</td>
</tr>
</tbody>
</table>

\*Values are expressed as mean (SD); ppm indicates parts per million.

\†Variables for which statistically significant differences in the sexes were observed.

### Table 2
**Qualitative Variables for the Sample and by Sex**

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nicotine replacement therapy</td>
<td>95 (77.9%)</td>
<td>62 (81.6%)</td>
<td>33 (71.7%)</td>
</tr>
<tr>
<td>Bupropion</td>
<td>27 (22.1%)</td>
<td>14 (18.4%)</td>
<td>13 (28.3%)</td>
</tr>
<tr>
<td>Nicotine gum</td>
<td>91 (74.6%)</td>
<td>57 (75.0%)</td>
<td>34 (73.9%)</td>
</tr>
</tbody>
</table>

### Table 3
**Weight Gain for the Sample and by Sex**

<table>
<thead>
<tr>
<th>Weight Gain</th>
<th>Total</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kilograms</td>
<td>2.6 (2.4)</td>
<td>2.6 (2.2)</td>
<td>2.6 (2.6)</td>
</tr>
<tr>
<td>Percentage</td>
<td>3.6 (3.3)</td>
<td>3.2 (2.8)</td>
<td>4.1 (4.0)</td>
</tr>
</tbody>
</table>

**Table 4
** Weight gain according to BMI category

<table>
<thead>
<tr>
<th>Weight</th>
<th>BMI, kg/m²</th>
<th>Weight Gain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Kilograms</td>
<td>Percentage</td>
</tr>
<tr>
<td>Underweight</td>
<td>&lt;18.5</td>
<td>4.8</td>
</tr>
<tr>
<td>Normal weight</td>
<td>18.5-25</td>
<td>2.6</td>
</tr>
<tr>
<td>Overweight</td>
<td>25-30</td>
<td>2.4</td>
</tr>
<tr>
<td>Obese</td>
<td>&gt;30</td>
<td>2.9</td>
</tr>
</tbody>
</table>

25% of the population experienced a weight gain of more than 4.2 kg, and no patient gained more than 9.2 kg. Figure 1 depicts the study population classified according to BMI categories, and Table 4 summarizes data on weight gain (in kilograms and as percentages) for each BMI category. It can be observed that there was a tendency for patients in the higher BMI categories to put on slightly less weight. The ANOVA between BMI category and weight gain in kilograms and between BMI category and percentage weight gain revealed no significant differences \( (P=0.481 \text{ and } P=0.085, \text{ respectively}) \).

No statistically significant differences emerged from comparisons to detect relationships between weight gain in kilograms and the number of cigarettes smoked a day, Fagerström test score, type of pharmacological treatment (bupropion or NRT), nicotine gum use, and trait and state anxiety scores on the day before giving up smoking.

An analysis of how state anxiety and weight in kilograms evolved over the 3 months of treatment revealed that state anxiety decreased whereas weight increased (Figures 2 and 3). Nonetheless, it must be emphasized that the relationship is not necessarily an inverse one, and a dispersion diagram, in fact, confirmed that there was no relationship between those variables.

Finally, Table 5 shows the results of ANOVA between weight gain in kilograms and the 2 Fagerström test items—"time to the first cigarette of the day" and "number of cigarettes smoked a day." Significant differences were found between categories for the item "time to the first cigarette of the day" \( (P=0.022) \).
post hoc analysis (Tukey test) revealed that the most important differences occurred between the category “more than 60 minutes” and the categories “between 30 and 60 minutes” and “between 6 and 30 minutes” ($P=0.027$ and $P=0.031$, respectively). This clearly demonstrates that the individuals who gained least weight were those who did not smoke their first cigarette until 60 minutes after waking (Figure 4). The same analysis performed for men and women revealed that there were no significant differences for men ($P=0.672$) (Figure 5), whereas weight gain did change according to smoking patterns for women ($P=0.012$) (Figure 6). The Tukey post hoc test confirmed the difference for women ($P=0.01$ and $P=0.04$, respectively).

Of note is the fact that the same findings were observed when percentage weight gain was analyzed.

### Discussion

The analysis of anxiety demonstrated that trait anxiety was greater in women smokers than in men smokers, a pattern which reflects the situation in the

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**TABLE 5**

<table>
<thead>
<tr>
<th>Relationship betwen weight gain and selected Fagerström test items</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight gain, kg, versus number of cigarettes</td>
<td>.043*</td>
</tr>
<tr>
<td>Weight gain, kg, versus time to the first cigarette of the day</td>
<td>.022*</td>
</tr>
</tbody>
</table>

*Statistically significant differences.
nonsmoking population. State anxiety was observed to decrease gradually over the 3 months of treatment, while weight increased gradually over the same period even though no relationship was demonstrated between the 2 variables. Since pharmacological treatment acts fundamentally on the abstinence syndrome, of which anxiety is one of the main symptoms, it is likely that the reduction in anxiety was due at least in part to the treatment. The fact that a gradual decrease in anxiety has been described in other studies would seem to indicate that nicotine increases rather than diminishes anxiety. Although anxiety levels are generally greater in the first few weeks after cessation, our study would suggest that weight gain is not a consequence of anxiety but is more related to the metabolic effects of nicotine.

Mean weight gain at 2.6 kg (3.6%) 3 months after smoking cessation was moderate; consistent with other similar studies was the fact that only 25% of the studied population gained over 4 kg, and no study participant gained more than 10 kg. A number of studies have demonstrated, in fact, that only a small percentage of individuals experience substantial weight gain. Most studies, however, have described weight gain in kilograms, whereas we reported percentage gains; this nondimensional measure reflects real gain more accurately, as it takes into account an individual’s weight/height relationship. The sample distribution according to BMI categories reflects the distribution of the Spanish population. The smaller weight gains reported for subjects in higher BMI categories are consistent with our findings, although the differences we observed between categories were not statistically significant. Although the relationship between weight gain and smoking cessation has been widely documented, the underlying reasons for this association remain unclear. A number of possible contributory factors have been evaluated, including calorie intake, physical activity, and, above all, the metabolic effects of nicotine. In experiments with animals and human volunteers, nicotine intake has been shown to produce a 10% increase in calorie expenditure. Nicotine suppression, on the other hand, results in a fall in the metabolic rate, which some authors have estimated at between 12% and 16%. Nicotine also releases noradrenaline and serotonin, substances that suppress appetite. These facts would explain the weight gain in ex-smokers, as well as the relatively lower weight of smokers compared to nonsmokers described in epidemiological studies. The fact that lapsed ex-smokers recover their original weight after a period of abstinence, combined with the inverse relationship between obesity and smoking (reported in a number of population studies), would indirectly confirm the effects of nicotine on weight. Other mechanisms by means of which smoking might contribute to weight loss include the action of nicotine on insulin and lipase. As for changes in eating habits, a factor in weight gain is that ex-smokers have been reported to increase their calorie intake, especially of simple carbohydrates (sugars), even so, strict diets are not recommended, as they may have negative repercussions on the smoking habit.

Figure 5. Weight gain in body mass index categories in relation to the Fagerström test item “time to the first cigarette of the day” for men. CI indicates confidence interval.

Figure 6. Weight gain in body mass index categories in relation to the Fagerström test item “time to the first cigarette of the day” for women. CI indicates confidence interval.
cessation process.2,28 Other less relevant factors affect weight gain and include lifestyle changes that are directly related to nicotine suppression, such as the fact that taste and smell are enhanced, and that gastric emptying is slowed down.27

One of the recommendations for avoiding or slowing down weight gain is the use of nicotine gum.2,29 In our study, weight gain was 5% higher in the group that used nicotine gum at will, no weight gain differences were found between those who used gum (almost 75% of participants) and those who did not.2,30 Although bupropion has been reported to help avoid weight gain, the evidence is not conclusive. In our study we found no differences between the pharmacological treatments administered (bupropion and NRT), although it has been demonstrated that, compared to no treatment, both treatments help minimize weight gain.3,29

In regard to the relationship between weight and the 2 Fagerström test items that measure degree of dependency (time to the first cigarette of the day and number of cigarettes smoked a day), we found differences in former smoking patterns—women smoking less—but no differences between the sexes with regard to weight gain. Most studies have reported greater weight gains among women ex-smokers, a fact which has been described as both a direct relationship between weight and the 2 Fagerström test items that measure degree of dependency (time to the first cigarette of the day and number of cigarettes smoked a day). We found differences in former smoking patterns—women smoking less—but no differences between the sexes with regard to weight gain. Most studies have reported greater weight gains among women ex-smokers, a fact which has been described as both a perceived obstacle to smoking cessation in women and as an initiating factor in teenage girls.3,31

The role that nicotine plays in creating addiction is quite clear. Nonetheless, smoking addiction is a complex process that includes pharmacological, learning, personality, and lifestyle factors. Image is currently an important factor in professional and personal relations, and physical appearance is a value that is more closely associated with esthetics than health.3,31 Therefore, the probability of weight gain after giving up smoking—even if moderate and transient—can become an important barrier to giving up smoking. A number of smoking cessation programs have included strategies aimed at preventing weight gain, such as physical exercise or calorie-controlled diet recommendations.2,29 Results from these programs are contradictory, but the fact remains that none have managed to fulfill the aim of avoiding weight gain. Some authors propose cognitive and behavioral therapies that modify beliefs and that transmit a message that the possible drawback of weight gain is accompanied by enormous benefits that more than compensate for a changed body image.2,33

To sum up, our results show that weight gain after smoking cessation is moderate and that there is no relationship between weight gain and the anxiety that accompanies smoking cessation. This information needs to be transmitted to smokers so that weight gain is not raised as an obstacle to giving up smoking. Smoking cessation protocols should, moreover, be designed to include weight monitoring, physical exercise programs, and cognitive therapy techniques directed toward body image.

REFERENCES
NERÍN I ET AL. WEIGHT GAIN AND ANXIETY LEVELS IN RECENT EX-SMOKERS