Weight Gain During Long-Term Treatment of Obsessive-Compulsive Disorder: A Prospective Comparison Between Serotonin Reuptake Inhibitors

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Background: The effect of extended antidepressant treatment on weight has been poorly investigated. Also unknown is whether different compounds have differential effects. The aim of the present study was to assess changes in weight in obsessive-compulsive disorder (OCD) patients treated for 2.5 years with clomipramine or selective serotonin reuptake inhibitors.

Method: 138 DSM-IV OCD patients who responded to 6-month acute treatment at the Mood and Anxiety Disorders Unit, Department of Neuroscience, University of Turin, Italy, were followed-up for 2 years while receiving open-label clomipramine, citalopram, fluoxetine, fluvoxamine, paroxetine, or sertraline. Patients were consecutively recruited and followed from May 1998 to March 2003. The mean percentage change in weight was compared for each group, as was the proportion of patients who had a ≥ 7% weight increase from baseline.

Results: At the end of the 2.5-year study period, patients had gained a mean of 2.5% of their body weight with respect to baseline (1.58 kg); 14.5% of the total sample experienced a significant (≥ 7%) weight increase. Within each but the fluoxetine treatment group, paired t tests showed a significant increase in weight from baseline to final visit. Analysis of variance showed a significant difference between treatment groups ($p = .009$), with clomipramine being associated with the highest weight increase and fluoxetine and sertraline with the lowest. A higher proportion of clomipramine-treated patients (34.8%) gained ≥ 7% in weight as compared with sertraline and fluoxetine, which accounted for the lowest percentage of patients with a significant weight gain (4.5% and 8.7%, respectively), although this difference was not statistically significant.

Conclusion: Risk of weight gain during extended serotonin reuptake inhibitor treatment for OCD differs depending on which compound is used. The differences among antiobsessive drugs may affect compliance with medication and health risks.

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Weight gain is a common side effect of many psychotropic drugs. Although weight gain could be acceptable for patients treated over a limited period of time, a significant weight gain reported during long-term treatment can result in loss of self-esteem, jeopardize patient compliance, and pose serious health hazards.

Antidepressant-induced weight gain has been clearly established with the use of tricyclic drugs in the acute treatment of major depression.[1–3] Weight gain liabilities of selective serotonin reuptake inhibitors (SSRIs) during the treatment of affective disorders need to be further clarified; indeed, several trials showed no weight change, and some even showed weight loss, during initial antidepressant therapy,[4–8] while over the long-term, results of SSRI treatment on weight are more controversial. In the Sussman et al.[9] review of clinical trials comparing nefazodone versus SSRIs, 17.9% of patients treated long-term with SSRIs reported an "extreme" weight gain (≥ 7%). Similarly, Sachs and Guille[10] reported that between 25% and 33% of patients taking SSRIs for 12 weeks or longer in their clinic gained a substantial amount of weight.

Data from the few placebo-controlled studies that mention weight changes in the long-term treatment of depression suggest, on the contrary, that long-term treatment with an SSRI is not associated with greater weight gain than is treatment with placebo. In the 1-year prevention of recurrence of depression study by Doogan and Caillard,[11] the mean change in body weight was no different between sertraline- and placebo-treated patients. Mackle and Kocsis[6] did not find any difference between citalopram and placebo in determining weight gain after 6 months of therapy, although 3.9% of subjects treated with citalopram experienced a ≥ 7% weight gain (vs. 2.8% of placebo-treated subjects). In the only prospective, double-blind, placebo-controlled study of weight gain during

long-term antidepressant treatment in the literature, Michelson et al.[8] found no difference in weight gain among patients treated with fluoxetine and placebo for 1 year: an increase in body weight of ≥ 7% was reported in 4.8% of fluoxetine-treated patients, a rate comparable with that of placebo-treated patients (6.3%). In line with this finding, Wade et al.[12] found that 4.7% of 541 patients treated with citalopram reported weight gain of > 5 kg after 1 year of treatment.

Some authors have suggested that the observed weight gain during long-term antidepressant therapy could be associated mainly with recovery from depression, rather than with the pharmacodynamic effect of antidepressants. In a study of 100 remitted depressed patients, Benazzi[13] found that 72% of patients experienced weight gain associated with antidepressant-induced remission of depression without any association with psychoactive drugs patients had taken during the depressive episode.

Nonetheless, Fava et al.[14] and Lecrubier and Judge[15] argued against the idea that the described weight gain could be fully explained as a recovery effect, as differences exist between different antidepressant classes in their potential to induce an increase in body weight. Fava et al.,[14] in a double-blind study comparing paroxetine, fluoxetine, and sertraline treatment for a total of 26 to 32 weeks, suggested that differences exist even within the same class of antidepressant, with paroxetine accounting for the maximum weight gain among the SSRIs. Paroxetine was associated with a significant increase in weight of about 3.6% over 6 months versus a slight increase for sertraline (1.0%) and a slight decrease for fluoxetine (–0.2%). In addition, a significantly higher proportion of paroxetine-treated patients (25.5%) gained ≥ 7% in weight compared with the other treatment groups (fluoxetine, 6.8%; sertraline, 4.2%).

Furthermore, in a double-blind, randomized trial conducted over 48 weeks on 176 patients with panic disorder,[15] 5.9% of paroxetine-treated and 14.3% of clomipramine-treated patients reported weight gain at the end of the trial, while no patients in the placebo arm experienced this side effect. This study reported weight gain in a condition not usually associated with weight loss during the acute symptomatic phase and therefore not susceptible to a recovery effect, suggesting that weight gain could be a significant side effect of treatments.

Although weight gain during antidepressant treatment has been specifically studied mainly in samples of depressed patients, unfortunately, this issue has not been addressed in obsessive-compulsive disorder (OCD) and other clinical conditions that are usually treated with this class of drugs.

Some studies performed on OCD patients but not focused specifically on weight gain have noted an increase in weight during acute treatment with clomipramine,[16–18] while failing to report such a weight gain during acute treatment with fluoxetine.[16] Data regarding weight gain with fluvoxamine and sertraline are more confused, with different studies reporting conflicting results.[17–19] No data are available on weight changes occurring during long-term treatment of OCD.[20–25]

The aim of the present study was to establish whether long-term pharmacotherapy of OCD could lead to an increase in weight, and whether this weight gain is to be considered clinically significant. Further, we wanted to verify whether patients taking different antiobsessive drugs differently experience this side effect.

MATERIALS AND METHOD

Sample

A sample of 292 OCD outpatients consecutively referred to the Mood and Anxiety Disorders Unit, Department of Neuroscience, University of Turin, Torino, Italy, first entered a 6-month, open-label, noncomparative, acute treatment phase. All patients had to meet DSM-IV criteria for OCD; 267 (91.4%) completed the acute treatment phase (25 discontinued: 15 due to side effects [7, nausea; 8, anxiety] and 10 due to incomplete adherence to treatment). A total of 149 patients (55.8%) met the criteria for response (a decrease of ≥ 35% in Yale-Brown Obsessive Compulsive Scale [YBOCS][27,28] total score) at the end of the 6-month acute treatment phase and entered a 2-year extension. Eleven patients were lost to follow-up and did not complete the extension phase (9 patients relapsed and consequently had their treatment regimen changed, and 2 were lost because of other reasons). Thus, 138 patients completed the 2-year follow-up.

All patients were over 18 years of age and gave their informed consent prior to enrollment after the procedure of the study had been fully explained. Patients were recruited and followed from May 1998 to March 2003.

The DSM-IV diagnosis of OCD was made by administering the Structured Clinical Interview for DSM-IV Axis I Disorders-Patient Edition[26] during the screening visit; moreover, patients were required to exhibit a YBOCS score greater than or equal to 16.[27,28] Patients were excluded from the study for any of the following reasons: pregnancy; lactation; a current or past diagnosis of eating disorder, schizophrenia, or other psychotic disorders; substance use disorders; an organic mental disorder; or a medical illness that would contraindicate the use of serotonin reuptake inhibitors (SRIs). Furthermore, we excluded from the sample patients currently meeting diagnostic criteria for a major depressive episode (both in the context of a major depressive disorder and in the context of a bipolar disorder diagnosis) or having a 17-item Hamilton Rating Scale for Depression (HAM-D) total score of ≥ 15 at the screening visit, in order to avoid the confounding effect on weight of an improvement of depression after long-term pharmacotherapy.

Table 1. Patient Characteristics at Baseline, Final Weight Gain, and Proportion of Patients With an Extreme Weight Gain (≥ 7%) by Treatment Group

Variable	Clomipramine (N = 23)	Citalopram (N = 21)	Fluoxetine (N = 23)	Fluvoxamine (N = 28)	Paroxetine (N = 21)	Sertraline (N = 22)	Statistical Analysis (ANOVA or χ^2 test)
Age, mean ± SD, y	33.0 ± 8.5	35.6 ± 9.4	35.3 ± 8.9	33.9 ± 8.4	35.2 ± 9.3	36.7 ± 9.6	F = 0.494; df = 5, p = .781
Sex, female/male, N	12/11	10/11	11/12	14/14	11/10	12/10	χ^2 = 0.335; df = 5, p = .997
Weight, mean ± SD, kg	67.6 ± 12.4	66.9 ± 13.0	65.3 ± 13.3	67.5 ± 14.2	67.6 ± 13.2	66.0 ± 13.6	F = 0.121; df = 5, p = .988
Weight gain, mean ± SD, %	4.9 ± 5.0	2.5 ± 3.5	0.9 ± 3.8	2.6 ± 2.8	2.6 ± 3.2	1.6 ± 2.8	F = 3.187; df = 5, p = .009[a]
Weight gain, mean ± SD, kg	2.9 ± 2.6	1.5 ± 1.9	0.5 ± 2.4	1.7 ± 1.8	1.7 ± 2.1	1.0 ± 1.7	F = 3.318; df = 5, p = .007[b]
Patients with ≥ 7% weight gain, % (N)	34.8 (8)	14.3 (3)	8.7 (2)	10.7 (3)	14.3 (3)	4.5 (1)	χ^2 = 10.345; df = 5, p = .066

[a]Post hoc test (Bonferroni): clomipramine vs. fluoxetine p = .004; clomipramine vs. sertraline p = .038.
[b]Post hoc test (Bonferroni): clomipramine vs. fluoxetine p = .003; clomipramine vs. sertraline p = .053.
Abbreviation: ANOVA = analysis of variance.

Study Design

Our study is a naturalistic and prospective analysis of the weight gain liabilities of clomipramine, citalopram, fluoxetine, fluvoxamine, paroxetine, and sertraline over the long-term in the treatment of OCD subjects who completed 2.5 years of pharmacotherapy.

Patients were evenly distributed across 6 treatment groups: clomipramine, citalopram, fluoxetine, fluvoxamine, paroxetine, and sertraline; clinical decision regarding patients' assignment to the treatment subgroups was not affected by patients' weight at baseline. All these compounds have been found to be effective in the acute treatment of OCD in double-blind, placebo-controlled studies.[19,22,29–32]

Patients were maintained on drug treatment for at least 2.5 years. During this period, dosages could be adjusted according to tolerability and response to treatment.

To systematically assess the effects of extended SRI treatment on weight, patients' weight was recorded at the beginning of the drug treatment (T0) and prospectively at 6-month intervals (T6, T12, T18, T24, and T30) for the whole follow-up period (2.5 years). Visits were performed in the morning; patients were weighed with empty stomachs on the same balance. Patients' food intake was not influenced by advice or specific medical instructions.

Statistical Analysis

Treatment group comparisons of patient demographic characteristics were done using the Pearson χ^2 test and analysis of variance (ANOVA). Statistical significance was defined as a 2-sided p value ≤ .05.

In order to evaluate weight gain liabilities of different antiobsessive drugs over an extended treatment period, we compared the mean weight change in kilograms across time among the different treatment groups for all patients who completed the follow-up period; moreover, we compared the mean percentage change in weight across time among SRIs (percentage weight change was defined as weight at a given time [i.e., T30] minus initial weight [T0], divided by initial weight [T0]). We also examined the number of patients who experienced a ≥ 7% increase in weight, considered the standard of "extreme" weight gain in clinical trials.

Changes in weight (mean weight change in kilograms and mean percentage weight change) were compared using the ANOVA; within-treatment changes were tested using a paired t test. The proportions of patients with ≥ 7% weight increase were compared using the Pearson χ^2 test.

RESULTS

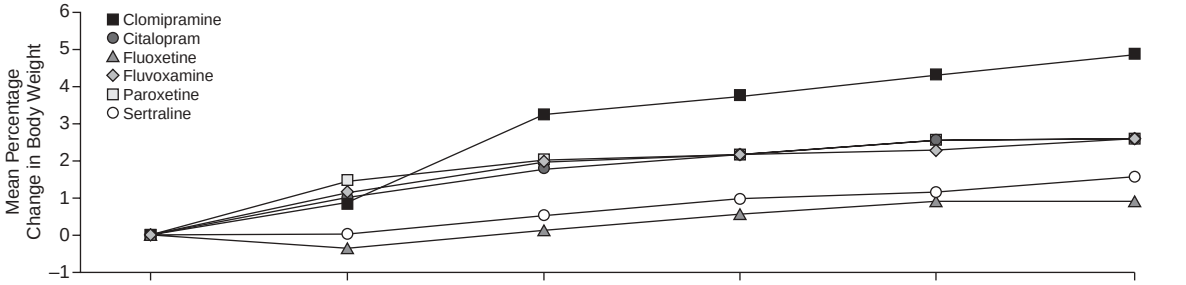
One hundred thirty-eight patients (70 females and 68 males) completed the 2-year follow-up after the 6-month acute treatment phase. They were treated with 1 of the following SRIs: clomipramine (N = 23), citalopram (N = 21), fluoxetine (N = 23), fluvoxamine (N = 28), paroxetine (N = 21), and sertraline (N = 22). Their mean ± SD age was 34.9 ± 8.9 years; the mean ± SD duration of illness was 12.1 ± 8.8 years. The mean ± SD baseline YBOCS total score was 25.0 ± 3.7, and the mean ± SD HAM-D total score was 9.8 ± 2.3.

Drug-treatment groups were not different at baseline for age, gender distribution, or weight (Table 1). During the 2-year follow-up period, dosages varied within each treatment group according to tolerability and response; daily dosages, however, were within the following ranges at any time during the follow-up: clomipramine, 150 to 250 mg; citalopram, 40 to 80 mg; fluoxetine, 40 to 80 mg; fluvoxamine, 200 to 300 mg; paroxetine, 40 to 80 mg; and sertraline, 150 to 200 mg.

At the end of the 2.5-year study period (6 months of acute treatment and 2 years of maintenance treatment), patients had gained a mean of 2.5% of body weight with respect to baseline, equal to 1.58 kg; on the basis of paired t tests, the increase in body weight (kg) for the whole sample was statistically significant (t = –8.444, df = 137, p < .001). When considering "extreme" weight gain, 14.5% of the total sample experienced a significant (≥ 7%) increase in weight at the end of the study period.

Table 1 reports the mean final weight changes and the proportions of subjects experiencing a significant weight

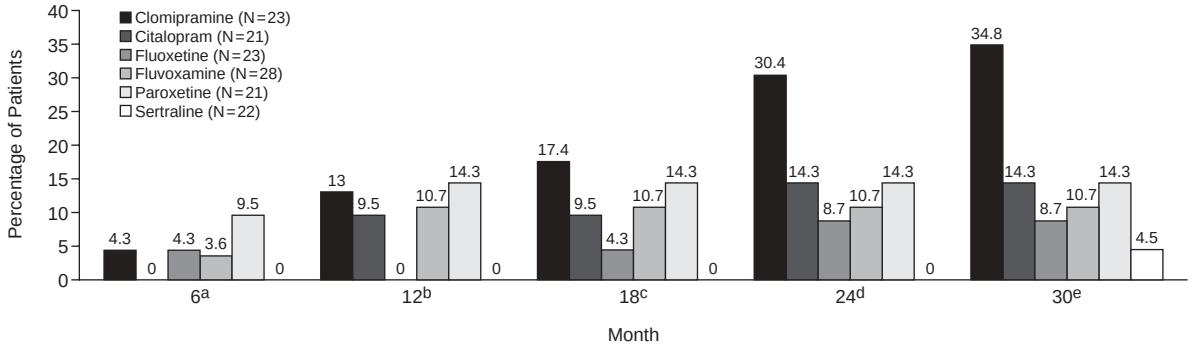
Figure 1. Mean Percentage Increase in Weight Across Time by Treatment Group



	Month 6	Month 12	Month 18	Month 24	Month 30
Clomipramine (N = 23)	0.89 ± 2.19	3.24 ± 4.22	3.73 ± 4.38	4.32 ± 4.79	4.86 ± 5.04
Citalopram (N = 21)	1.06 ± 1.65	1.79 ± 2.72	2.04 ± 2.88	2.57 ± 3.67	2.53 ± 3.49
Fluoxetine (N = 23)	–0.40 ± 2.11	0.11 ± 2.53	0.57 ± 3.21	0.92 ± 3.84	0.92 ± 3.84
Fluvoxamine (N = 28)	1.14 ± 2.15	1.92 ± 2.67	2.08 ± 2.66	2.29 ± 2.67	2.61 ± 2.82
Paroxetine (N = 21)	1.43 ± 2.58	1.96 ± 2.72	2.16 ± 2.85	2.52 ± 3.18	2.59 ± 3.17
Sertraline (N = 22)	0.03 ± 1.21	0.54 ± 1.53	0.97 ± 2.03	1.17 ± 2.23	1.57 ± 2.78
ANOVA	F = 2.796; df = 5, p = .020	F = 3.555; df = 5, p = .005	F = 2.924; df = 5, p = .015	F = 2.786; df = 5, p = .020	F = 3.187; df = 5, p = .009
Post hoc test (Bonferroni)	FLX vs PAR p = .050	CMI vs FLX p = .004 CMI vs SERT p = .028	CMI vs FLX p = .011 CMI vs SERT p = .048	CMI vs FLX p = .017 CMI vs SERT p = .044	CMI vs FLX p = .004 CMI vs SERT p = .038

Abbreviations: ANOVA = analysis of variance, CMI = clomipramine, FLX = fluoxetine, PAR = paroxetine, SERT = sertraline.

Figure 2. Cumulative Proportion of Patients With an Extreme Weight Gain (≥ 7%) Across Time by Treatment Group

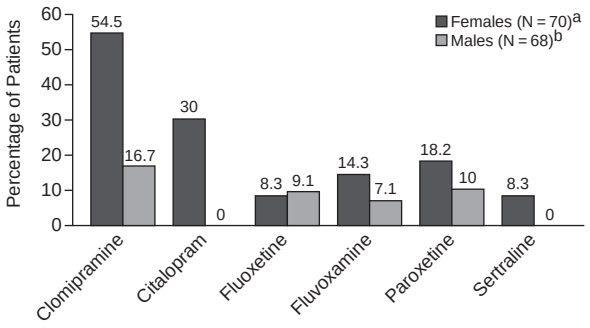


[a]Pearson $\chi^2 = 3.780$, df = 5, p = .582.
[b]Pearson $\chi^2 = 6.202$, df = 5, p = .287.
[c]Pearson $\chi^2 = 5.332$, df = 5, p = .377.
[d]Pearson $\chi^2 = 10.008$, df = 5, p = .075.
[e]Pearson $\chi^2 = 10.345$, df = 5, p = .066.

gain for each treatment group. Within each but the fluoxetine treatment group, paired t tests showed a significant increase in weight (kg) from baseline to final visit (T30) (clomipramine: t = –5.389, df = 22, p < .001; citalopram: t = –3.600, df = 20, p = .002; fluoxetine: t = –1.055, df = 22, p = .303; fluvoxamine: t = –4.976, df = 27, p < .001; paroxetine: t = –3.739, df = 20, p = .001; sertraline: t = –2.832, df = 21, p = .010). The ANOVA showed a significant difference between treatment groups, with clomipramine being associated with the highest mean increase in weight and fluoxetine and sertraline with the lowest. Between-group differences emerged also when considering the proportions of subjects with extreme weight gain, although the statistical analysis only approached significance: a higher proportion of clomipramine-treated patients (34.8%) gained ≥ 7% in weight as compared with sertraline and fluoxetine, which accounted for the lowest percentage of patients with a significant weight gain (4.5% and 8.7%, respectively). Finally, citalopram, fluvoxamine, and paroxetine were intermediate in causing weight gain (Table 1).

Figures 1 and 2 show, respectively, the mean percentage increase in weight and the cumulative proportion of patients with "extreme" weight gain at each time interval for each treatment group, with statistical comparisons between the different SRIs. When considering between-group differences in mean weight changes, a statistical difference between clomipramine on the one hand and fluoxetine and sertraline on the other emerged at month 12 and remained for the whole study period, while at

Figure 3. Cumulative Proportion of Patients With an Extreme Weight Gain (≥ 7%) at Month 30 by Treatment Group and Gender



[a]Pearson $\chi^2 = 10.539$, df = 5, p = .061.
[b]Pearson $\chi^2 = 3.347$, df = 5, p = .647.

month 6, a difference emerged between fluoxetine (causing a 0.4% decrease in weight) and paroxetine (associated with the highest increase in weight [1.4%]).

When patients were categorized by gender, female patients (N = 70) experienced a higher mean final increase in weight than male patients (N = 68) (3.25 ± 4.56% vs. 1.77 ± 2.44% of body weight; t = 2.374, df = 136, p = .019). Moreover, the difference in mean final weight gain (%) between clomipramine-treated subjects and the other groups was found to be significant only in the female subsample (ANOVA: F = 2.925, df = 5, p = .019), not in the male one (ANOVA: F = 0.904, df = 5, p = .484). The same trend was evident when we considered the proportions of patients experiencing a ≥ 7% increase in weight by gender: a trend toward significance was found by treatment group only in the female sample (Figure 3).

DISCUSSION

Weight gain as a side effect of long-term antidepressant treatment has only been studied in samples of depressed patients, although an often longer antidepressant treatment at high doses is needed to treat other psychiatric conditions such as generalized anxiety disorder, panic disorder, or OCD.

The aim of the present study was to establish, in a prospective although naturalistic study, whether long-term pharmacotherapy of OCD could lead to an increase in weight and whether this weight gain is to be considered clinically significant. Further, we wanted to verify whether patients taking different antiobsessive drugs differently experience this side effect.

To our knowledge, this is the first study to specifically address the issue of antidepressant-induced weight change in OCD patients. Double-blind comparative studies[16–19,33] on the acute treatment of OCD mentioned weight changes after 8 to 16 weeks of treatment. These studies suggest that antidepressant-induced weight changes might occur during the acute treatment of OCD and that differences between the single drugs might exist. No data are available on weight changes occurring during long-term treatment of OCD, as the main focus of studies on long-term pharmacotherapy was to demonstrate the liability of serotonergic compounds to maintain improvements obtained in the acute phase as well as to prevent relapses of the disorder.[20–25]

Results of our study show that patients who completed the 2.5-year follow-up actually reported an increase in weight (2.5% of body weight, 1.58 kg) at the completion of the study; although this weight change could be judged as unremarkable, a considerable proportion of the whole sample (14.5%) achieved a clinically significant weight gain: that is, according to the definition used by several authors,[8,9,14] greater than or equal to 7% with respect to baseline.

Our secondary objective was to analyze whether different antiobsessive compounds have differential effects on body weight. The tricyclic clomipramine accounted for the maximum increase in weight (4.9%; 2.9 kg) as well as for the highest percentage of patients achieving at least a 7% increase in body weight: more than one third of clomipramine-treated patients experienced a clinically significant increase in weight. Among the SSRIs, citalopram, fluvoxamine, and paroxetine were responsible for a lesser weight gain, while fluoxetine and sertraline appeared not to cause this side effect. When statistically comparing the different compounds, a significant difference was only found between clomipramine on the one hand and sertraline and fluoxetine on the other. The small size of the sample for each treatment arm prevented us from finding statistically significant differences among the single SSRIs in their potential to induce weight changes. Our results, however, together with the data of Fava et al.[14] on differences between paroxetine, sertraline, and fluoxetine in the long-term treatment of depression, suggest that differences among the SSRIs exist even in OCD and point toward the opportunity of double-blind studies examining this issue.

Our objective was to examine weight changes in patients treated for extensive periods of time and, accordingly, we selected only patients who completed the 2.5-year follow-up. Our results do not take into account dropouts due to adverse events related to weight changes. It has to be pointed out, however, that we had no dropouts due to weight gain.

A limitation of our study is its open-label although prospective design; the absence of a placebo arm makes it difficult to establish if the observed weight gain could have been due to a placebo effect or to the pharmacologic effect of the drugs. Nevertheless, the 34.8% rate of subjects with significant weight gain in the clomipramine group seems too high to be fully explained by a placebo

effect, which in literature (depression studies) accounts for a rate of ≥ 7% weight gain among patients that varies from 2.8% to 6.3%.[6,8] Of interest is the fact that sertraline and fluoxetine were associated in our study with a proportion of subjects with an extreme weight gain, 4.5% and 8.7%, respectively, comparable to that of placebo.

It has to be considered that some OCD patients, especially those affected by contamination obsessions or nosophobia, could restrict their dietary regimen, consequently losing weight; their weight could increase due to clinical improvement during pharmacotherapy. Nevertheless, according to our clinical experience, we consider this "recovery effect" rare enough not to have affected the overall results. We also excluded subjects with concomitant major depressive episodes as well as those with current and lifetime eating disorders from the present study in order to exclude potential confounding factors for the analysis of data.

We did not look at subthreshold comorbidities because the absence of well-established diagnostic criteria for subthreshold disorders makes them difficult to be evaluated. Nevertheless, we assume that this confounding effect should have been widespread to all subgroups, although this remains to be proved. The fact that fluoxetine was associated with weight loss—although not statistically significant—from baseline to month 6 can be taken as further confirmation of the absence of a depression-linked recovery effect.

In light of these considerations, our results appear to be of considerable clinical importance, allowing us to make the assumption that antidepressant-induced weight gain during long-term OCD treatment can be regarded as a true medication side effect. Some authors[34] have postulated that the possible mechanism responsible for weight gain could be carbohydrate craving or changes in serotonin 5-HT_{2C} receptor activity, although Wirshing et al.,[35] who specifically investigated the link between serotonergic activity and weight gain liability of the novel antipsychotics, could not establish such a relationship. Further research is needed to ascertain the causes that lead to this underestimated and not fully explicated side effect.

An important finding we did not expect was that the increase in weight was greater and statistically different in women than in men. Moreover, when we examined the different role of each compound in inducing weight changes, a significant difference between treatment groups could be found only in the female sample. Fava and colleagues[14] also noted that extreme weight gain among paroxetine-treated patients was more prevalent in women (39.1%) than in men (12.5%); although these data refer to depressed patients, their results and ours point toward a role of gender in antidepressant-induced weight changes. There is evidence of the recently discovered role of leptins in influencing body weight. The way psychotropic drugs interact with the leptin system might help to explain weight gain during pharmacotherapy.[38] Furthermore, relevant gender-based differences in leptin levels exist, with higher levels in women at birth that persist throughout life[39]: this difference might contribute to explaining why women, especially, seem to gain weight during pharmacotherapy. It is worth reporting, however, that other authors could not correlate weight gain with sex[36,37]; thus, this finding deserves further investigation.

Finally, a limitation in our study is the lack of body mass index values, although we followed the methodology of other published studies that specifically addressed the problem of weight gain during pharmacotherapy.[35,40]

Despite all limitations discussed above, our study provides the first evidence that both clomipramine and SSRIs are associated with weight gain in the long-term treatment of patients with OCD. Among antiobsessive drugs, clomipramine appears to have the greatest potential to induce weight gain and sertraline and fluoxetine the least. The clinical implication of our study is that, as clinicians, during long-term OCD pharmacotherapy, we must keep in mind the possibility of a clinically relevant weight gain, especially if we are prescribing clomipramine to a female patient; not taking this side effect into account could raise the risk of noncompliance or worsen the patient's quality of life.

Drug names: citalopram (Celexa), clomipramine (Anafranil and others), fluoxetine (Prozac and others), paroxetine (Paxil and others), sertraline (Zoloft).

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