Late Onset Eating Disorders in Spain: Clinical Characteristics and Therapeutic Implications

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Objective: The literature on later age of onset (LAO) in women with eating disorders is scarce. We compared the severity of eating disorders, eating disorder subtype, and personality profiles in a clinical sample of consecutively assessed women with eating disorders with later age of onset (LAO, > = 25 years) to women with typical age of onset (TAO, < 25 years).

Method: All eating disorder patients met the Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV) criteria and were admitted to the Eating Disorder Unit of the University Hospital of Bellvitge in Barcelona, Spain. Ninety-six patients were classified as LAO and 759 as TAO.

Assessment: Measures included the Eating Attitude Test-40 (EAT-40), Eating Disorders Inventory-2 (EDI-2), Bulimic Investigatory Test Edinburgh (BITE), Symptom Checklist Revised (SCL-90-R), and the Temperament and Character Inventory-Revised (TCI-R), as well as other clinical and psychopathological indices.

Results: LAO individuals reported significantly fewer weekly vomiting episodes, fewer self-harming behaviours, less drug abuse, and lower scores on the BITE symptoms, the EDI-2 drive for thinness, and the TCI-R harm avoidance scales than TAO individuals. Conversely, the LAO group reported more current and premorbid obesity than the TAO group.

Conclusion: LAO eating disorder patients in this sample presented with milder symptomatology and less extreme personality traits. Premorbid obesity may be more relevant to LAO than TAO eating disorders and should be routinely assessed and considered when planning treatment.


Keywords: eating disorders; anorexia nervosa; bulimia nervosa; EDNOS; age of onset; psychopathology; obesity; personality

Eating disorders affect approximately 2%-4% of the female population in Western countries (Smink, van Hoeken, & Hoek, 2012). Spain is no exception, with several population-based studies yielding prevalence estimates ranging from 1% to 3.5% (Pelaez Fernandez, Labrador, & Raich, 2007; Sepulveda, Carrobles, & Gandarillas, 2008). The mean prevalence estimate for anorexia nervosa (AN) is 0.3% and for bulimia nervosa (BN) 1.5% (Hoek, 2006; Hoek & van

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Financial support was received from Fondo de Investigación Sanitaria -FIS (PI11/210) and AGAUR (2009SGR1554). It was also partially supported by the European Commission under the Seventh Framework Programme (FP7-ICT-215839-2007- Playmancer project). CIBER Fisiopatología de la Obesidad y Nutrición (CIBEROBN) and CIBER Salud Mental (CIBERSam), are an initiative of ISCIII. Dr. Krug was supported by a Marie-Curie Intra European Fellowship within the 7th European Community Framework Programme, as No 254774 (call reference: FP7-PEOPLE-2009-IEF).

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Hoeken, 2003). The lifetime prevalence of binge eating disorder (BED) in six European countries, including Spain, is estimated to be 1.9% (Preti et al., 2009), slightly lower than estimates from the United States (3.5%; Hudson, Hiripi, Pope, & Kessler, 2007). Little is known about the prevalence of eating disorder not otherwise specified (EDNOS), which could partially be attributable to the heterogeneous nature of the category. One of the few studies assessing the occurrence of EDNOS in the general population reported a prevalence of 2.37% (Machado, Machado, Goncalves, & Hoek, 2007).

The Epidemiology of Later Age of Onset (LAO)

The typical age at onset (TAO) of AN is early adolescence (between 14 and 18 years of age), and the TAO of BN and BED is somewhat later, usually in late adolescence and early adulthood (American Psychiatric Association [APA], 2000; Koruth, Nevison, & Schwannauer, 2012). Little is known about age of onset of EDNOS. Although most patients with eating disorders report TAO, later onsets do occur; however, our understanding of these cases is limited to a few studies on AN (Boast, Coker, & Wakeling, 1992; Tobin, Molteni, & Elin, 1995) and BN (Mitchell et al., 1987), and to isolated case series examining later age of onset (LAO; Inagaki et al., 2002; Kellett, Trimble, & Thorley, 1976). A major United States prevalence study of eating disorders (Hudson et al., 2007) demonstrated that while there were no identified cases of AN with an onset after the mid-20s, BN and BED did commonly onset at older ages. Recent estimates indicate that 1 out of every 10 eating disorder patients was older than 40 years of age (Hoek, 2006; Hoek & van Hoeken, 2003). While some of these cases represented relapses of disorders at earlier ages, others reported a LAO with no previous history, a finding confirmed by other studies (Forman & Davis, 2005; Scholtz, Hill, & Lacey, 2010). These population-based statistics are supported by clinic-based research (Cumella & Kally, 2008b; Forman & Davis, 2005), which has revealed increasing admissions to inpatient eating disorder services for females aged 35 years and older in recent years (Wiseman, Sunday, Klapper, Harris, & Halmi, 2001).

Definition of LAO

The definition of LAO in eating disorders has been inconsistent (Mangweth-Matzek et al., 2006) and there is substantial heterogeneity with regard to the criteria used to identify onsets outside the typical age range (Joughin, Crisp, Gowers, & Bhat, 1991; Rabe-Jablonska, 2003). In fact, the International Statistical Classification of Diseases and Related Health Problems (WHO, 1993) 10th (ICD-10) excludes AN diagnosis if onset is after menopause, and the Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV) states that “the onset of this disorder rarely occurs in females over the age of forty” (APA, 2000, p:587). Many of the existing studies anchor their definition in Feighner’s criteria (Feighner et al., 1972) conceptualization of AN, which defines five necessary conditions for the diagnosis, including onset before 25 years of age. Additional definitions have been somewhat arbitrary, with Joughin and colleagues (1991) and later Tobin and colleagues (1995), characterizing LAO as onset of the disorder at or after 30 years of age citing the rationale that “this group was well after the more characteristic age at onset, and seemed likely to emphasize any differences from the more typical sample” (Joughin et al., 1991, p:976). It is important to note that these definitions were developed prior to the shifts in demographics towards older age at presentation, which we are currently seeing in clinical referrals.

Risk Factors for LAO

Although age of onset of eating disorders has been thoroughly researched (Hudson et al., 2007; Scholtz et al., 2010), the relation among risk factors, eating symptomatology, comorbid psychopathology, personality, and LAO has received very little scientific attention (Goldfein, Walsh, & Midlarsky, 2000). The few studies that have explored risk factors for LAO eating disorders revealed less family history of major psychiatric disorders (Russell & Gilbert, 1992), and lower comorbidity (Beck et al., 1996; Boast et al., 1992), but more bereavement (Hill, Haslett,
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& Kumar, 2001; Inagaki et al., 2002), marital conflicts, and separation or divorce (Dally, 1984; Inagaki et al., 2002). However, most of these studies were based on clinical case series which limits their generalizability.

LAO and Eating Disorder Symptomatology

LAO has been associated with less severe eating disorder symptoms in terms of weight, fear of food, and body image distortion (Dally, 1984; Runfola et al., 2012) and poorer prognosis and outcome (Boast et al., 1992; Inagaki et al., 2002). However, these differences have not been observed consistently, as some studies have failed to identify any substantial clinical differences, between LAO and TAO eating disorder patients on several dimensions, including age at menarche or duration of illness prior to presentation (Joughin et al., 1991), weight at presentation, and weight control methods (Joughin et al., 1991). Conversely, a more recent study (Cumella & Kally, 2008a), revealed more pronounced eating disorder severity, denial of the disorder, and increased emotional and behavioural over-control in women with a midlife eating disorder onset compared to individuals with TAO. The same study assessed eating disorder diagnoses between both onset groups and found that inpatient women with LAO presented significantly more commonly with AN-restricting and AN-binge purging, but fewer BN diagnoses than the women with TAO. Clear patterns are not yet identifiable to clarify these inconsistencies.

LAO and Comorbidity

The problem of co-occurring disorders is important to consider when examining the aetiology of LAO eating disorders because adulthood may be a time during which people experience depressive symptoms associated with, for example, the loss of their parents, relationship breakups, and divorce (Hill et al., 2001; Inagaki et al., 2002). Hence, the majority of individuals presenting with midlife-onset eating disorders report comorbid depression and approximately 65% also presented with anxiety (Cumella & Kally, 2008a,b; Forman & Davis, 2005). In many cases, the eating disorder symptoms decreased once the depression was managed, which raises the question as to whether the association between life stressors and eating disorders might be mediated by depression.

To our knowledge, the comorbidity of impulsive behaviours in eating disorder women with LAO onset has been assessed only in two previous studies (Cumella & Kally, 2008a,b), revealing that substance abuse/dependence, self-harming behaviours, and suicidality were less common in LAO than TAO inpatients. The authors attributed their finding to reflect generational differences in impulsive behaviours.

LAO and Personality

Specific personality traits such as obsessionality and perfectionism have been found to be present before, during, and after an eating disorder diagnosis (Halmi et al., 2005; Lilenfeld, 2011). Evidence that certain personality traits may be associated with certain age at onset in women with eating disorders is lacking (Cumella & Kally, 2008a; Forman & Davis, 2005). Identifying differences in personality traits relative to age of eating disorder onset is important as it may inform both our understanding about course of illness and treatment (Midlarsky & Nitzburg, 2008).

Importance of Assessing LAO

Our knowledge about LAO eating disorders is limited to case reports or convenience samples with considerable methodological shortcomings, including the absence of structured interviews, limiting observations to one eating disorder subtype, and a virtual absence of research on personality traits. As the prevalence of eating disorders among LAO is receiving more recognition, it is essential to evaluate any differences associated with this onset pattern. Because the aetiology and pathophysiology of LAO eating disorders are at present unknown, larger scale studies that
contrast the etiologic factors, clinical manifestation, comorbidity, and personality of eating disorders between TAO and LAO can aid in the development of suitable treatment and prevention programs for different age groups (Peat, Peyerl, & Muehlenkamp, 2008).

Aims of the Study

We aimed to overcome some of the limitations of previous studies by studying a large sample of consecutively assessed individuals with eating disorders from Catalonia, Spain and comparing those individuals with LAO versus TAO on a broad range of clinical and personality measures. Our specific goals were twofold: (a) to explore differential features (clinical, psychopathological, and personality) between individuals with LAO and TAO eating disorders and (b) to assess whether LAO and TAO patients differ on these measures across eating disorders subtypes (AN, BN, and EDNOS). Based on the limited information from the literature, we hypothesized that compared to TAO, LAO eating disorder patients would evidence lower scores on psychopathology indices and have a higher body mass index (BMI).

Method

Participants

Entry into the study was between January 2002 and December 2006. The final sample included 855 female eating disorder patients consecutively admitted to the eating disorder unit at the University Hospital of Bellvitge (HUB) in Barcelona, Spain. All participants were diagnosed according to DSM-IV (APA, 2000) criteria, using a semistructured clinical interview (SCID-I; First, Gibbon, Spitzer, & Williams, 1996), conducted by experienced psychologists and psychiatrists. Individuals were excluded from the analyses if they had missing values for any diagnostic items. For the present analysis, from an initial sample of 993 eating disorder patients, the following individuals were excluded: 71 males, 52 females with BED due to the small sample size, and 73 patients with missing relevant data. With regard to diagnostic subtypes of the final 798 included patients, 175 (21.9%) presented with AN, 452 (56.6%) with BN, and 171 (21.4%) with EDNOS [excluding (BED)]. Ninety-two of these eating disorder patients presented with LAO (19.6% AN; 64.1% BN; 16.3% EDNOS) and were compared with the remaining 706 female eating disorder individuals with TAO (22.2% AN, 55.7% BN; 22.1% EDNOS). The mean age of the total sample was 26.2 years (standard deviation [SD] = 7.1). The range of age of onset for the TAO group was 7–25 years and for the LAO group it was 26–53 years. The Ethics Committee of our institution approved this study and informed consent was obtained from all participants.

Definition of LAO and TAO

To help determine the optimal definition of later versus typical age of onset, we explored age at onset data from a large international independent study, the Genetics of Anorexia Nervosa (Wang et al., 2011). In this sample, the mean age of onset was 16.9 (SD = 4.3) for the 841 female participants with a lifetime history of AN. Exploring the age at onset distribution, individuals with an age of onset of 25 years or older had an age of onset that was at least two standard deviations above the mean. This information, corroborated with additional information in the literature (Inagaki et al., 2002; Kimura, Tonoike, Muroya, Yoshida, & Ozaki, 2007), was used to determine our cutoff for LAO for the current study. Those with an age of onset of 25 years or older were placed in the LAO group. All other participants comprised the TAO group.

Assessment

We used a comprehensive battery of assessments to capture eating disorder symptoms and clinical presentation, general psychopathology, personality and impulsive behaviours. The battery included the Eating Attitudes Test-40 (EAT-40; Garner & Garfinkel, 1979), the Eating Disorders Inventory-2 (EDI-2; Garner, 1998), the Bulimic Investigatory Test Edinburgh
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(BITE; Henderson & Freeman, 1987), the Symptom Checklist-Revised (SCL-90-R; Derogatis, 1990), and the Temperament and Character Inventory-Revised (TCI-R; Cloninger, 1999), as well as further questions on impulsive behaviours, sociodemographics, and relevant clinical variables for eating disorders.

**EAT-40 (Garner & Garfinkel, 1979)**. The EAT-40 contains 40 items, including symptoms and behaviours common to eating disordered patients and provides an index of the severity of the disorder. Scores on this questionnaire range from 0 to 120. The higher the scores, the more disturbed the eating behaviour. The cutoff score is generally set at 30, and differentiates between a pathological and a nonpathological population. This questionnaire has been adapted for the Spanish population and has exhibited a high internal consistency (Cronbach's alpha coefficient = .93) in a previous study (Castro et al., 1991).

**EDI 2 (Garner, 1998)**. The EDI-2 is a reliable and validated 91-item, multidimensional, self-report questionnaire that assesses different cognitive and behavioural characteristics, which are typical for eating disorders. The EDI-2 retains the 64 items (grouped into eight scales: Drive for Thinness, Bulimia, Body Dissatisfaction, Ineffectiveness, Perfectionism, Interpersonal Distrust, and Interceptive Awareness, Maturity Fears) of the EDI and adds 27 new items into three provisional scales: Asceticism, Impulse Regulation, and Social Insecurity. All of these scales are answered on a 6-point Likert scale and provide standardized subscale scores. This instrument was validated in a Spanish population and was found to have a mean internal consistency of .63 (coefficient alpha) in an earlier study (Garner, 1998).

**BITE (Henderson & Freeman, 1987)**. The BITE contains 33 items that measure the presence and the severity of bulimic symptoms. There are two subscales: the Symptomatology scale (30 items), which assesses the seriousness of the symptoms, and the Severity scale (three items), which offers a severity index. The cutoff points for the Symptomatology scale scores for the present study were as follows: <10 = no symptomatology; 10–20 threshold symptoms and > 20 threshold gravity symptoms. For the Severity scale, higher scores indicate greater severity of the eating disorder. This questionnaire has been adapted to the Spanish population and has been found to have a high internal consistency (Cronbach’s alpha coefficient: .96) in a previous study (Rivas, Bernabé, & Jiménez, 2004).

**SCL-90-R (Derogatis, 1990)**. We used the SCL-90-R to assess a broad range of psychopathological symptoms. This test contains 90 items and helps to measure nine primary symptom dimensions: Somatization, Obsession-Compulsion, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism. In addition, it includes three global indices: (a) a global severity index (GSI), designed to measure overall psychological distress; (b) a positive symptom distress index (PSDI), designed to measure the intensity of symptoms; and (c) a positive symptom total (PST). The GSI can be used as a summary of the test. Derogatis (2002) validated this scale in a Spanish population and reported a mean internal consistency (Coefficient alpha) of .75.

**TCI-R (Cloninger, 1999)**. The TCI-R (Cloninger, 1999) is a 240-item, reliable, and validated questionnaire that measures, as in the original TCI version (Cloninger, Svrakic, & Przybeck, 1993), seven dimensions of personality: four temperament (Harm Avoidance, Novelty Seeking, Reward Dependence, and Persistence) and three character dimensions (Self-Directedness, Cooperativeness, and Self-Transcendence). All items are measured with a 5-point Likert scale. The performance on the Spanish version of the original questionnaire (Gutierrez et al., 2001) and the revised version (Gutierrez-Zotes et al., 2004) have been documented. In the revised version of this instrument, Gutierrez-Zotes et al. (2004) reported an internal consistency (coefficient alpha) of .87.

**Impulsive behaviours.** The SCID-I (First et al., 1996) for DSM-IV Axis I Disorders was used to assess lifetime alcohol and drug abuse/dependence. In addition, self-harm, defined
as direct and intentional self-injurious behaviours (cutting/burning/hitting/scratching/hair pulling), which did not lead to death and lifetime suicide attempts (with the question “Have you ever attempted suicide?”), was assessed by a semistructured clinical interview (Fernandez-Aranda & Turon, 1998).

**Evaluation of sociodemographic and clinical variables.** Additional demographic information, including age, marital status, education, occupation, living arrangements, parental occupation, clinically relevant variables (weight, height, premorbid obesity [defined as BMI values above 30 kg/m²], childhood obesity, number of previous treatments, weekly frequency of binges, vomiting episodes, laxative use, and diuretic use, were also assessed by semi-structured clinical interview (Fernandez-Aranda & Turon, 1998).

**Procedure**

Experienced psychologists and psychiatrists with master’s or doctoral degrees completed the clinical assessment during two structured face-to-face interviews, before any psychological or pharmacological treatment was initiated. In addition to the clinical interview, demographic information was obtained through self-report questionnaires. Confidentiality was maintained throughout the whole assessment and participants were allowed to withdraw from the study at any time. At the end of the study participants were debriefed. The total duration of each appointment was approximately between 1 and 5 hours.

**Statistical Analysis**

Statistical analyses were carried out with SPSS 15.0.1 for Windows (SPSS Inc., 2006). A 3 × 2 inter-group factor design (eating disorder subtype—AN/BN/EDNOS—and age of onset—LAO/TAO) adjusted by duration of illness was considered. Negative binomial models were used for count data outcomes (dependent variables as the number of previous treatments and weekly frequency of binges, vomiting episodes, diuretics, and laxatives) and analysis of variance (ANOVA) procedures for metric outcomes (psychological, clinical, and other psychometric scores). For both procedures, the interaction of the two independent factors (eating disorder subtype and age of onset group) was first evaluated. For nonsignificant interaction terms ($p > .05$), only main effects were interpreted. For models with significant interactions, simple effects (parameters that show the effect of one variable within each level of the other variable/s implicated in the interaction) were obtained and interpreted.

Finally, logistic regressions, stratified by age of onset group and adjusted by duration of illness, were conducted to evaluate differences between eating disorder subtype on criteria presence of obesity and impulsivity behaviours (alcohol abuse and suicide attempts) and the predictor eating disorder subtype. Goodness-of-fit of logistics was valued through Hosmer-Lemeshow’s test.

Because of the multiple statistical comparisons, Finner’s correction was applied (Brown & Russell, 1997), through SPSS macros (Domènech, 2008). This method uses a sequential procedure to adjust $p$-values, while retaining statistical power, and has been demonstrated to be a good alternative to Bonferroni’s correction procedure (which is considered too conservative, with low power to detect real significance differences).

**Results**

**LAO and Sociodemographics**

LAO patients were significantly older (mean age: 36.2, $SD = 7.72$) than TAO patients (mean age: 24.4, $SD = 5.8$; $p < .001$) and presented with a significantly shorter eating disorder duration (mean duration: 4.3, $SD = 4.4$) compared with those with TAO (mean duration: 7.0, $SD = 5.8$; $p = .001$). In addition, compared with TAO individuals, LAO eating disorder women were less likely to be single ($p < .029$).
LAO and Eating Disorder Subtype

The accumulated distribution of the variable age of onset for each of the eating disorder subtypes is shown in Figure 1. No statistically significant differences were found between age of onset and eating disorder subtype, $\chi^2(2) = 2.57; p = .277$.

LAO and Clinical Characteristics

Table 1 shows the comparisons between LAO and TAO for clinical characteristics. Patients with LAO reported significantly fewer weekly vomiting episodes ($p = 0.005$) than TAO women. Regarding current BMI, a significant interaction of eating disorder subtype and age of onset group was found only for BN patients, with the LAO group presenting a higher current BMI than the TAO group ($p < 0.005$).

LAO and Eating Disorder Symptomatology

Differences between LAO and TAO eating disorder symptomatology (measured by the EAT-40, the EDI-2, and the BITE) are presented in Table 2. Compared with the TAO group, individuals with LAO reported significantly lower scores on the BITE symptoms scale ($p = 0.001$) and the EDI-2 drive for thinness scale ($p = 0.005$).

LAO, General Psychopathology, and Personality

Table 3 presents results for the comparisons between LAO and TAO in terms of general psychopathology and personality. No statistically significant differences emerged between the two age of onset groups in terms of general psychopathology as measured by the SCL-90-R.

For the TCI-R, a significant interaction between eating disorder subtype and age of onset group was observed for the AN group for the harm-avoidance scale, with the LAO AN patients presenting with a lower mean score than the TAO group ($p = 0.001$).

LAO and Obesity

Significantly more women with LAO than TAO were currently obese (25.8% vs. 5.9%; $p < .001$) and reported premorbid obesity (37.3% vs. 15.6%; $p < .001$). No statistically significant
Table 1

Comparison of Clinical Measures Across Eating Disorder Subtype (AN, BN and EDNOS) and Age of Onset Group (LAO and TAO)

<table>
<thead>
<tr>
<th></th>
<th>Late age of onset (&gt;25 yrs)</th>
<th>Typical age of onset (≤25 yrs)</th>
<th>p-values for Negative binomials (Bonferroni-Finner’s adjustment)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AN</td>
<td>BN</td>
<td>EDNOS</td>
</tr>
<tr>
<td></td>
<td>(n = 18; 19.6%)</td>
<td>(n = 59; 64.1%)</td>
<td>(n = 15; 16.3%)</td>
</tr>
<tr>
<td>N° previous treat.</td>
<td>0.89 (0.96)</td>
<td>0.57 (0.99)</td>
<td>0.42 (0.61)</td>
</tr>
<tr>
<td>Weekly binges</td>
<td>0 (0)</td>
<td>8.09 (7.27)</td>
<td>0.06 (0.24)</td>
</tr>
<tr>
<td>Weekly vomits</td>
<td>1.00 (2.38)</td>
<td>6.33 (8.74)</td>
<td>1.68 (2.54)</td>
</tr>
<tr>
<td>Weekly laxative use</td>
<td>1.94 (5.3)</td>
<td>4.59 (10.5)</td>
<td>3.58 (7.0)</td>
</tr>
<tr>
<td>Weekly diuretic use</td>
<td>0 (0)</td>
<td>2.73 (7.44)</td>
<td>2.37 (9.62)</td>
</tr>
<tr>
<td></td>
<td>Current BMI</td>
<td>Age of onset</td>
<td>Duration ED</td>
</tr>
<tr>
<td></td>
<td>15.20 (1.03)</td>
<td>28.94 (2.82)</td>
<td>26–38</td>
</tr>
<tr>
<td></td>
<td>28.21 (6.42)</td>
<td>33.10 (7.22)</td>
<td>26–53</td>
</tr>
<tr>
<td></td>
<td>20.99 (2.99)</td>
<td>30.80 (4.02)</td>
<td>27–39</td>
</tr>
<tr>
<td></td>
<td>15.78 (1.43)</td>
<td>18.11 (3.04)</td>
<td>4–25</td>
</tr>
<tr>
<td></td>
<td>23.74 (4.75)</td>
<td>17.20 (3.48)</td>
<td>7–25</td>
</tr>
<tr>
<td></td>
<td>21.13 (4.47)</td>
<td>17.31 (3.05)</td>
<td>9–25</td>
</tr>
<tr>
<td></td>
<td>4.81 (5.03)</td>
<td>5.18 (4.77)</td>
<td>7.82 (5.93)</td>
</tr>
<tr>
<td></td>
<td>4.73 (4.43)</td>
<td>7.82 (5.93)</td>
<td>6.83 (5.69)</td>
</tr>
</tbody>
</table>

Note: Non-estimable model due to lack of goodness of fit (cells with means or proportions equal to 0). AN = anorexia nervosa; BN = bulimia nervosa; EDNOS = eating disorder not otherwise specified; LAO = later age of onset; TAO = typical age of onset; SD = standard deviation; ANCOVA = analysis of covariance.
## Table 2

### Comparison of Eating Disorder Symptomatology Measures by Eating Disorder Subtype and Age of Onset Group: ANOVA Adjusted by Duration of Illness

<table>
<thead>
<tr>
<th>Means (SD)</th>
<th>Late age of onset (&lt;25 yrs) (N = 92)</th>
<th>Typical age of onset (≤25 yrs) (N = 706)</th>
<th>ANCOVA (Bonferroni-Finner's adjustment)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AN (n = 18; 19.6%)</td>
<td>BN (n = 59; 64.1%)</td>
<td>EDNOS (n = 15; 16.3%)</td>
</tr>
<tr>
<td>BITE: symptoms</td>
<td>10.00 (6.3)</td>
<td>23.69 (4.9)</td>
<td>11.94 (6.8)</td>
</tr>
<tr>
<td>BITE: severity</td>
<td>3.56 (4.33)</td>
<td>14.63 (6.89)</td>
<td>7.44 (6.35)</td>
</tr>
<tr>
<td>EAT-40 total</td>
<td>44.1 (22.8)</td>
<td>47.9 (19.5)</td>
<td>45.3 (25.5)</td>
</tr>
<tr>
<td>EDI: drive for thinness</td>
<td>6.72 (6.53)</td>
<td>13.22 (6.18)</td>
<td>10.11 (7.47)</td>
</tr>
<tr>
<td>EDI: body dissatisfaction</td>
<td>9.28 (6.50)</td>
<td>19.46 (7.3)</td>
<td>12.06 (8.3)</td>
</tr>
<tr>
<td>EDI: interoception awareness</td>
<td>7.28 (5.29)</td>
<td>11.59 (5.9)</td>
<td>9.56 (7.19)</td>
</tr>
<tr>
<td>EDI: bulimia</td>
<td>1.50 (2.04)</td>
<td>9.95 (5.62)</td>
<td>2.22 (2.10)</td>
</tr>
<tr>
<td>EDI: interoception</td>
<td>4.94 (4.76)</td>
<td>7.14 (5.42)</td>
<td>6.28 (5.23)</td>
</tr>
<tr>
<td>EDI: ineffectiveness</td>
<td>5.28 (4.99)</td>
<td>10.81 (6.53)</td>
<td>9.78 (7.98)</td>
</tr>
<tr>
<td>EDI: bulimia</td>
<td>5.67 (5.89)</td>
<td>8.25 (5.86)</td>
<td>8.17 (6.32)</td>
</tr>
<tr>
<td>EDI: perfectionism</td>
<td>5.11 (4.54)</td>
<td>4.64 (4.01)</td>
<td>2.78 (3.34)</td>
</tr>
<tr>
<td>EDI: impulse regulation</td>
<td>5.28 (4.66)</td>
<td>6.05 (5.13)</td>
<td>6.39 (6.90)</td>
</tr>
<tr>
<td>EDI: ascetism</td>
<td>5.22 (5.61)</td>
<td>7.25 (4.15)</td>
<td>5.06 (4.08)</td>
</tr>
<tr>
<td>EDI: social insecurity</td>
<td>5.72 (4.21)</td>
<td>8.08 (4.93)</td>
<td>8.39 (5.78)</td>
</tr>
<tr>
<td>EDI: total score</td>
<td>62.0 (32.8)</td>
<td>106.5 (40.9)</td>
<td>80.8 (48.8)</td>
</tr>
</tbody>
</table>

**Note:** Non-estimable model due to lack of goodness of fit (cells with means or proportions equal to 0). AN = anorexia nervosa; BN = bulimia nervosa; EDNOS = eating disorder not otherwise specified; LAO = later age of onset; TAO = typical age of onset; SD = standard deviation; ANCOVA = analysis of covariance; ANOVA = analysis of variance; EDI = Eating Disorders Inventory-2; BED = binge eating disorder.
### Table 3
Comparison of Psychopathology and Personality Measures by Eating Disorder Subtype and Age of Onset Group: ANOVA Adjusted by Duration of Illness

<table>
<thead>
<tr>
<th></th>
<th>Late age of onset (&gt;25 yrs)</th>
<th>Typical age of onset (≤25 yrs)</th>
<th>ANCOVA (Bonferroni-Finner’s adjustment)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AN (n = 18; 19.6%)</td>
<td>BN (n = 59; 64.1%)</td>
<td>EDNOS (n = 15;63)</td>
</tr>
<tr>
<td>SCL: somatization</td>
<td>1.81 (1.75)</td>
<td>2.11 (0.94)</td>
<td>1.96 (1.01)</td>
</tr>
<tr>
<td>SCL: obsess-comp.</td>
<td>1.97 (0.86)</td>
<td>2.13 (0.86)</td>
<td>1.91 (1.19)</td>
</tr>
<tr>
<td>SCL: interp sens.</td>
<td>1.64 (0.84)</td>
<td>2.09 (0.86)</td>
<td>1.78 (1.04)</td>
</tr>
<tr>
<td>SCL: depression</td>
<td>2.02 (0.84)</td>
<td>2.40 (0.84)</td>
<td>2.18 (1.23)</td>
</tr>
<tr>
<td>SCL: anxiety</td>
<td>1.87 (0.96)</td>
<td>1.98 (0.92)</td>
<td>1.91 (1.15)</td>
</tr>
<tr>
<td>SCL: hostility</td>
<td>1.33 (1.03)</td>
<td>1.50 (0.97)</td>
<td>1.47 (1.20)</td>
</tr>
<tr>
<td>SCL: phobic anxiety</td>
<td>0.96 (0.91)</td>
<td>1.30 (1.09)</td>
<td>1.35 (1.29)</td>
</tr>
<tr>
<td>SCL: paranoid</td>
<td>1.32 (0.87)</td>
<td>1.48 (0.92)</td>
<td>1.59 (1.14)</td>
</tr>
<tr>
<td>SCL: psychot.</td>
<td>1.03 (0.54)</td>
<td>1.55 (0.76)</td>
<td>1.21 (0.83)</td>
</tr>
<tr>
<td>SCL: GSI</td>
<td>1.59 (0.71)</td>
<td>1.94 (0.75)</td>
<td>1.80 (1.03)</td>
</tr>
<tr>
<td>SCL: PST</td>
<td>60.7 (17.4)</td>
<td>68.6 (16.1)</td>
<td>65.6 (22.9)</td>
</tr>
<tr>
<td>SCL: PSDI</td>
<td>2.26 (0.54)</td>
<td>2.47 (0.61)</td>
<td>2.35 (0.76)</td>
</tr>
<tr>
<td>TCI: novelty seeking</td>
<td>102.2 (17.0)</td>
<td>100.7 (16.7)</td>
<td>98.6 (20.5)</td>
</tr>
<tr>
<td>TCI: harm avoid.</td>
<td>98.56 (17.0)</td>
<td>121.6 (18.0)</td>
<td>110.8 (24.0)</td>
</tr>
<tr>
<td>TCI: reward depend.</td>
<td>101.1 (18.0)</td>
<td>102.0 (17.0)</td>
<td>98.0 (17.7)</td>
</tr>
<tr>
<td>TCI: total persis</td>
<td>124.6 (18.0)</td>
<td>104.8 (21.0)</td>
<td>108.9 (15.0)</td>
</tr>
<tr>
<td>TCI: self-directed.</td>
<td>130.3 (19.0)</td>
<td>113.6 (20.0)</td>
<td>118.9 (25.0)</td>
</tr>
<tr>
<td>TCI: cooperative.</td>
<td>136.9 (14.0)</td>
<td>134.0 (19.0)</td>
<td>129.4 (16.0)</td>
</tr>
<tr>
<td>TCI: self-transcend.</td>
<td>70.7 (16.8)</td>
<td>63.6 (14.7)</td>
<td>58.4 (9.8)</td>
</tr>
</tbody>
</table>

*Note.* Non-estimable model due to lack of goodness of fit (cells with means or proportions equal to 0). AN = anorexia nervosa; BN = bulimia nervosa; EDNOS = eating disorder not otherwise specified; LAO = later age of onset; TAO = typical age of onset; SD = standard deviation; ANCOVA = analysis of covariance; ANOVA = analysis of variance; EDI = Eating Disorders Inventory-2; BED = binge eating disorder.
differences between the two groups were found for childhood obesity. Exploring obesity within eating disorder subtypes, differences emerged in the TAO but not in the LAO group for current obesity ($p < .001$), premorbid obesity ($p < .001$), and childhood obesity ($p = .019$). In the TAO group, participants with BN reported the highest percentages for all obesity measures: 10.2% for current obesity (0% for AN and 2.4% for EDNOS), 25.4% for premorbid obesity (0.7% for AN and 8.7% for EDNOS), and 11.5% for childhood obesity (1.9% for AN and 10.0% for EDNOS).

**LAO and Impulsive Behaviours**

No statistically significant differences between the two age of onset groups were found for alcohol use (13.4% TAO vs. 10.4% LAO; $p = .752$) and suicide attempts (23.9% TAO vs. 17.7% LAO; $p = .441$). However, compared with the TAO group, LAO participants were less likely to report drug abuse (12.5% LAO vs. 23.8% TAO; $p = .015$) and self-harming behaviours (18.8% LAO vs. 33.6% TAO; $p = .008$). No statistically significant differences were found within specific eating disorder subtypes.

**Discussion**

Our observations indicate that potentially meaningful differences do exist between eating disorders patients with late (LAO $\geq 25$ years) versus typical age of onset. Overall LAO patients endorsed lower levels of eating disorders related symptomatology (e.g., lower scores on the BITE and the EDI-2 drive for thinness; fewer weekly vomiting episodes) and less self-harming behaviours and drug abuse than individuals with TAO. Another general trend that emerged was greater premorbid and current obesity in the LAO than the TAO group.

**LAO and Eating Disorder Subtype**

The first main finding of this study was that 11.2% of patients with eating disorders presented with LAO. Considering eating disorder subtypes, LAO was reported in 10.3% of AN, 13.1% of BN, and 8.3% of EDNOS patients. The age of onset distributions reported here are consistent with those in previous reports (Boast et al., 1992; Joughin et al., 1991). Even though most studies (Wentz, Gillberg, Anckarsater, Gillberg, & Rastam, 2009) on age of onset in eating disorders have primarily focused their attention on the clinical characteristics of AN patients, our results suggest that other eating disorder subtypes should also be considered when exploring LAO in eating disorders.

**LAO and Eating Disorder Symptomatology**

Our second main finding was that patients with LAO presented with lower drive for thinness and bulimic symptomatology than those with TAO. In general our findings are in accordance with numerous other studies assessing age of onset in various psychiatric disorders, including ADHD (Karam et al., 2009), alcoholism (Hingson, Edwards, Heeren, & Rosenbloom, 2009), obsessive-compulsive disorder (Rosario-Campos et al., 2001) and bipolar disorder (Schurhoff et al., 2000), which have also reported less severe symptomatology in individuals with LAO than TAO.

On the other hand, the eating disorder literature on this topic is more inconsistent, with some studies (Bravender, Robertson, Woods, Gordon, & Forman, 1999; Cumella & Kally, 2008a) indicating that body image concerns are lower in those with LAO, while others (Runfola et al., 2012; Webster & Tiggemann, 2003) suggest that body dissatisfaction remains stable across the lifespan at least in the general population. With reference to bulimic symptomatology, our results concur with those studies that have shown lower binging and vomiting frequency in those with LAO (Blinder, Cumella, & Sanathara, 2006) not those that have indicated more pathology (Marcus, Bromberger, Wei, Brown, & Kravitz, 2007).
LAO, BMI, and Obesity

An intriguing finding was that both current and premorbid obesity were more commonly associated with late rather than typical onset. Accordingly, a linear relationship between BMI and eating disorders has been reported previously (Runfola et al., 2012), with overweight and obese individuals being at elevated risk for developing eating disorders (Gagne et al., 2012; Westerberg-Jacobson, Ghaderi, & Edlund, 2012). The age of onset of BED patients, who are generally overweight, has also been found to be higher than for the other eating disorder subtypes (French, Jeffery, Sherwood, & Neumark-Sztainer, 1999; Hudson et al., 2007). However, given that the sample size of BED patients in our study was insufficient, we were not able to reveal whether this was the case for our sample.

Our finding that the BN patients with LAO presented with a higher current BMI than the TAO BN group agrees with previous studies (Boast et al., 1992) that have suggested that BMI at the time of first assessment could operate as a vital clinical characteristic to distinguish TAO from LAO individuals. It is known that BN individuals tend to be at the high end of the normal weight range (APA, 2000) possibly developing unhealthy weight control practices to avoid obesity. This observation is also supported by Fairburn et al. (1998) who suggested that familial obesity is a more potent risk factor for BN than other eating disorder subtypes. One possible explanation could be that individuals who develop eating disorders later have a longer period of time to attain their genetically predetermined adult weight prior to engaging in disordered eating or weight control behaviour (Fairburn, Cooper, Doll, Norman, & O’Connor, 2000).

LAO and General Psychopathology

In terms of general psychopathology, we found no significant differences between LAO and TAO eating disorder patients. The literature on general psychopathology and age of onset of eating disorders is contradictory. Whereas a few studies have reported higher levels of depression in LAO BN (Blinder et al., 2006; Cumella & Kally, 2008a; Marcus et al., 2007), other studies (Bravender et al., 1999; Cumella & Kally, 2008a) have not been able to reveal such an association. Methodological differences in the studies may account for these inconsistencies (e.g., smaller sample size, not controlling for duration of the disorder, only considering BN).

LAO and Personality

We observed very few robust differences in personality between individuals with LAO and TAO. LAO AN patients did have significantly lower scores on the harm avoidance scale of the TCI-R than TAO AN patients. Harm avoidance scores are typically higher in AN than other eating disorder subtypes (Lilenfeld, 2011). Our observation is similar to other investigations that suggest that high harm avoidance might predispose to an earlier age of onset of several disorders including alcoholism (Lim et al., 2008), bipolar disorders (Engstrom, Brandstrom, Sigvardsson, Cloninger, & Nylander, 2003), major depressive disorders (Ongur, Farabough, Jiosifescu, Perlis, & Fava, 2005), and pathological gambling (Shin, Lim, Choi, Kim, & Grant, 2009). Because harm avoidance is strongly associated with both depression and anxiety (Cloninger, 1999; Joffe, Bugby, Levitt, Regan, & Parker, 1993), it is possible that the TAO individuals are more likely to have harm avoidance/anxiety as the entrée into their illness, whereas the LAO patients follow another pathway driven by efforts of weight control given their tendency towards overweight (Cumella & Kally, 2008a).

LAO and Impulsive Behaviours

Another important finding of our study was that, compared to the TAO group, the LAO eating disordered individuals were less likely to report drug abuse and self-harming behaviours. Some previous studies (Cumella & Kally, 2008a,b; Krug et al., 2009) have also shown that these impulsive behaviours are more common among TAO patients than LAO eating disorder
patients. Furthermore, psychoactive substance use disorders and impulsivity seem to decline substantially with age (Dom, D’Haene, Hulstijn, & Sabbe, 2006; Kouimtsidis & Padhi, 2007).

**Possible Factors Associated with LAO**

There are several possible interpretations of our findings. First, even though the aetiology of LAO eating disorders may resemble that of TAO, some associated factors may have a unique stage of life dimension (Marcus et al., 2007; Patrick & Stahl, 2009; Peat et al., 2008). In this sense, LAO women, like their younger TAO counterparts, may engage in disordered eating in response to developmental challenges, identity problems, psychological distress, and sociocultural pressures to be thin (Bravender et al., 1999; Cumella & Kally, 2008a; Patrick & Stahl, 2009; Peat et al., 2008). However, individuals with LAO may also encounter age-specific triggers and maintaining factors (e.g., dealing with a career, work and family stress, marital problems, financial concerns, and persistent concerns about overweight or obesity), which may play a more important role for them than in those with TAO (Fox & Leung, 2009; Marcus et al., 2007). Qualitative research approaches may aid in honing the interpretation of our observations (Berge, Loth, Hanson, Croll-Lampert, & Neumark-Sztainer, 2012). However the current data underscore the potential importance of overweight and obesity as a contributing factor to later onset eating disorders (Villarejo et al., 2012).

**Challenges in Defining LAO**

Although reports of LAO eating disorders are becoming more common, care must be taken in the definition of LAO as there is no agreed upon definition. Eating disorders diagnosed at older ages might have developed throughout younger years but remained undiagnosed because these women may have been ashamed or embarrassed by having a “teenage” problem. Second, women with LAO, who require treatment, may have to make significant family and/or career sacrifices, which might make them less likely than their TAO counterparts to seek treatment (Bravender et al., 1999). Finally, our standard assessment instruments lack age specificity. The majority of instruments are most appropriate for young adult females and lack questions regarding features such as age-related triggering factors, somatic symptomatology, and stressful events across the lifespan (Patrick & Stahl, 2009).

**Limitations**

The present study also has several limitations. First, the information regarding age of onset was obtained retrospectively, introducing the possibility of inaccuracy in recall by the patient. Second, the unequal sample sizes (for eating disorder subtypes and the age of onset comparison groups) might have affected the accuracy of our results. Third, our program treats only patients aged 18 years or older and may include a greater number of LAO cases than would be observed in programs that treat patients of all ages. Fourth, even though we adjusted our findings for duration of the disorder, which is a lineal combination of age of onset and age, we cannot know how much of the obtained differences are due to different current age or different age at onset. A healthy control group would be helpful but not resolutive because levels of psychopathology in such a group would be considerably low. Life stress, which generally increases with age and other age-related factors, could therefore have influenced our findings. Finally, we included only female eating disorder patients from Spain. Therefore, we do not know whether our results are generalizable to men, nonclinical populations, and individuals from other ethnic and cultural backgrounds.

**Clinical Implications for LAO**

The findings from the present study may help to shape appropriate prevention and early intervention programs. The current findings indicate that eating disorder patients with LAO present with a somewhat different clinical picture than patients with TAO and that clinicians should
be aware of these differential symptoms and traits when treating LAO patients. Specifically, clinicians should be mindful of the fact that LAO patients have obesity in their background, that they may have been teased in the past about weight and appearance, that they may be fearful of becoming obese again, and therefore might be reluctant to take medication that might cause weight gain.

Given the fact that currently there are no clinical trials comparing eating disorder treatment strategies in LAO versus TAO eating disorder patients, treatment as usual with slight modification relating to the special needs of LAO eating disorder patients appears most suitable. A transgenerational approach may for instance be helpful in treating eating disorders pertaining to different age and age of onset groups, whereby the emphasis is placed on the group members’ diversity of experiences and wisdom. Currently, treatments are being developed for adults that acknowledge the beneficial effect that family participation has in the treatment of youth with eating disorders. These interventions such as Uniting Couples in the treatment of Anorexia Nervosa (UCAN) engage partners in the treatment process to leverage family support in a developmentally appropriate way (Bulik, Baucom, & Kirby, 2012). Such innovations are essential to address the unique developmental challenges associated with eating disorders that present in midlife. In addition, through a combination of different techniques such as relaxation and mindfulness, stress should be recognized, acknowledged, and tackled in the treatment the LAO individuals (Sejcher, Gould Fogerite, & Perlman, 2012).

Conclusion

In conclusion, our results challenge the current conceptualization of LAO eating disorders by showing that these patients present with lower eating disorder symptomatology and body image concerns. It is also remarkable that obesity plays an important role in the development and course of LAO eating disorders. Future research could expand the present results by employing longitudinal designs addressing the potential mediating role of age of onset in the etiological factors and clinical course of eating disorders. Moreover, genetic, neuroimaging, and epigenetic designs could be meaningfully employed to determine whether etiological differences exist on the biological as well as psychological level.

References


