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Latent Class Analysis of Eating Disorders: Relationship to Mortality

Scott J. Crow, **Sonja A. Swanson**, **Carol B. Peterson**
University of Minnesota

Ross D. Crosby, **Stephen A. Wonderlich**, **James E. Mitchell**
Neuropsychiatric Research Institute, University of North Dakota School of Medicine and Health Sciences

Abstract

The current diagnostic nomenclature for eating disorders has shortcomings. Empirical attempts to identify a new nomenclature have found numerous latent structures, but validation of this work has been largely cross-sectional, and nothing is known yet about the relationship of derived latent classes to mortality. This study examined latent diagnostic structure in 1,885 participants seeking outpatient eating disorder treatment over an 18-year period. Eating disorder symptoms were used as indicators, and the main validator was mortality as assessed using computerized linkage to the National Death Index. Six latent classes were derived; three of the six had significantly elevated standardized mortality ratios. It appeared that the latent class structure yielded better delineation of mortality risk than the existing classifications in the *Diagnostic and Statistical Manual of Mental Disorders*. These results provide support for an alternative, empirically derived diagnostic structure.

Keywords

eating disorders; mortality; Latent Class Analysis

Eating disorders (ED) are relatively common (Hudson, Hiripi, Pope, & Kessler, 2007) and have many associated complications. Prominent among these complications is mortality. A prior meta-analysis of mortality studies has suggested that mortality, particularly suicide, may be higher in ED than in any other illness (Harris & Barraclough, 1998). Most evidence has suggested that these elevated mortality rates among individuals with ED occur primarily in individuals with anorexia nervosa (AN). This finding is somewhat surprising, given the high comorbidity of bulimia nervosa (BN) with problems associated with suicide such as depression. Indeed, recent evidence suggests that BN and ED not otherwise specified (EDNOS) may have elevated all cause mortality and elevated risk for suicide (Crow et al., 2009).

Correspondence concerning this article should be addressed to Scott J. Crow, MD., Department of Psychiatry, University of Minnesota Medical School, F282/2A West, 2450 Riverside Avenue, Minneapolis, MN 55454. crowx002@umn.edu.
Scott J. Crow, Sonja A. Swanson, and Carol B. Peterson, Department of Psychiatry, University of Minnesota; Ross D. Crosby, Stephen A. Wonderlich, and James E. Mitchell, Neuropsychiatric Research Institute, University of North Dakota School of Medicine and Health Sciences.

Currently three ED diagnoses (AN, BN, and EDNOS) are described in the *DSM–IV* (American Psychiatric Association, 1994). However, there are problems with the current diagnostic system. First, in most samples, most individuals with an ED meet criteria for an EDNOS diagnosis (Turner & Bryant-Waugh, 2004). Second, distinctions drawn between full and "subsyndromal" AN or BN that currently fall under EDNOS do not seem to denote clinically meaningful differences (Crow, Agras, Halmi, Mitchell, & Kraemer, 2002).

Several attempts have been made to develop classification models for the ED, generally using various forms of latent structure analysis to derive categories empirically. These are shown in Table 1. In general, these studies have used both clinical and nonclinical samples, have employed ED symptoms as indicators, and have used cross-sectional validators. Variations in sample size and sample type may account for the pattern of results, which range from two to six classes. Smaller numbers of classes have been seen in the more diagnostically restricted samples (Myers et al., 2006; Striegel-Moore et al., 2005).

There are significant limitations to existing work using latent structure analytic techniques. First, the majority of these samples have been nonclinical in nature and some, while large, have small numbers of individuals with ED. If the goal of these investigations is to look for meaningful classes within the realm of ED, this type of sampling would tend to diminish the likelihood of finding meaningful classes. Second, the validators used to examine derived classes or profiles have typically focused on concurrent features such as demographic variables or comorbidity. The strength of the evidence for or against any proposed diagnostic structure hinges on the robustness and clinical relevance of the validators used. Inasmuch as the primary clinical goals of diagnosis are to make predictions about long-term course and treatment response, longitudinal validators would be particularly useful, yet they are generally lacking. Third, most studies to date have not examined assumptions about local independence, which makes the interpretation of these studies more difficult (Swanson, Lindenberg, Bauer, & Crosby, 2011; Torrance-Rynard & Walter, 1997; Vacek, 1985). Such analyses proceed on the assumption that other indicators are independent of one another conditioned on the latent structures. In the case of ED, indicators such as Body Mass Index (BMI) and amenorrhea, or binge eating and purging might be expected not to be locally independent. For example, a subject belongs to a specific class and has a BMI of 12, the assumption of the model is that she has the same probability of amenorrhea as another woman in the same class but with a BMI of 17—which is biologically implausible.

The goal of the current study was to conduct a latent class analysis in a large clinical sample of all individuals presenting for treatment in an eating disorders clinic over an 18-year time period. A second goal was to validate the results of the latent class analysis using a highly relevant prospective validator: mortality. We hypothesized that latent class analysis would derive a structure with more classes than defined in the *DSM*, given the clinical heterogeneity of the sample. We further hypothesized that latent class analysis would provide a stronger prediction of mortality than the *DSM–IV* model.

Method

Participants

One thousand, eight hundred eighty-five (1,885) participants seeking treatment in the outpatient ED clinic at the University of Minnesota between 1979 and 1997 participated in the study.

Measures

ED Questionnaire—Each participant completed the Eating Disorders Questionnaire (Mitchell, Hatsukami, Eckert, & Pyle, 1985), a self-report instrument that assesses ED behaviors and cognition; demographic variables; current and prior treatment; other psychopathology; and medical history. The EDQ has been shown to have acceptable diagnostic agreement with structured interview-generated ED diagnoses. ($\kappa = 0.64$; (Keel, Crow, Davis, & Mitchell, 2002).

In addition, self-reported weight and height were used to calculate BMI (weight in kilograms divided by the square of the height in meters). While concern has been raised in the past about the accuracy of self-reported weight and height, there is evidence that this information may be particularly accurate in individuals who have BN (Doll & Fairburn, 1998).

National Death Index—The National Death Index (NDI) is a computerized death index for the entire United States (Acquavella, Donaleski, & Hanis, 1986) established in 1979 and updated yearly by the National Center for Health Statistics. For this project, records were screened through 2004. The NDI has been shown to have specificity and sensitivity above 95% (Sesso, Paffenbarger, & Lee, 2000).

In the current study, an NDI Plus search was used; NDI Plus yields cause of death coding with very high agreement with the method of obtaining actual death certificates followed by expert assignment of cause of death (Doody, Hayes, & Bilgrad, 2001). Causes of death were then classified by the first author into the following groups: suicide, substance abuse-related, traumatic, or medical.

Statistics—Latent structure models were fit using the software Latent GOLD 4.5. Several series of models were fit with increasing numbers of classes until a minimum was found for Bayesian Information Criteria (BIC) and Bozdogan's Criterion (CAIC); consideration for model fit was also given to Sample Size Adjusted Bayesian Information Criteria (ABIC), Akaike's Information Criterion (AIC), and classification error. Potential indicators for these models were selected based on prior results of latent structure analyses in ED samples. Several variable definitions were considered (e.g., defining binge frequency as an ordinal variable vs. a dichotomous variable reflecting the presence/absence of binge episodes). Further, to address the issue of local independence assumption violations, bivariate relaxations of this assumption were used when applicable and model fit was reassessed.

Once a latent structure model was finalized, subjects were assigned to classes based on maximum posterior probability. Standardized mortality ratios (SMR) were calculated for each class and survival analyses were conducted to assess how class membership predicted mortality. To calculate SMR, expected mortality adjusted for age, race, gender, and calendar year was obtained from mortality rates published by the Centers for Disease Control (CDC, 2008a, 2008b).

Results

The demographic data and descriptive characteristics of the sample are found in Table 2. Overall, the sample was predominantly female (95.1%) and white (94.9%). The original *DSM*-based diagnostic classifications were as follows: AN 9.4% (n = 177); BN 48.1% (n = 906); EDNOS 42.5% (n = 802).

A number of models were fit to the data using latent class analysis with the primary objective being to minimize BIC and CAIC. Most models converged on a six-class solution, with some yielding seven class solutions. The best-fitting model yielded six-classes. Indicators used for the best-fitting model included current BMI (≤17.5, 17.5–19, 19–25, 25–30, >30); binge eating frequency (never, less than two per week, two to six per week, greater than or equal to once per day); vomiting frequency (never; less than two times per week, two to six times per week, greater than or equal to once per day); meal skipping (never, less than two per week, two to six per week, greater than or equal to once per day); exercise frequency (never, less than two per week, two to six per week, greater than or equal to once per day); laxative use (yes/no); diuretic use (yes/no); enema use (yes/no); and fear of weight gain (coded yes if excessive; otherwise coded no). Based on bivariate residuals observed with model fitting, it appeared that meal skipping and exercise frequency might violate local independence assumptions; thus, models were run allowing local dependence between meal skipping and exercise. This modification resulted in little change in probability structure or class membership assignment, but did yield smaller classification errors. The conditional and marginal probabilities for the six-class solution are found in Table 3, and bivariate residuals are shown in Table 4.

Analyses conducted with amenorrhea as an indicator yielded a six-class solution but large bivariate residuals were observed for amenorrhea. Model-fitting that allowed local dependence between amenorrhea and current BMI again led to a six-class solution, but amenorrhea was no longer a significant variable (p = .26). Class membership was highly consistent in the models without and with amenorrhea locally dependent with BMI: 97% of classifications were the same in each. For these reasons, amenorrhea was excluded.

Class 1 (comprising 32.7% of the sample, n = 628) was notable for moderate levels of binge eating and purging. Class 2 (26.0% of the sample, n = 513) was most likely to report the highest levels of binge eating and vomiting. Class 3 (15.1% of the sample, n = 258), endorsed moderate levels of binge eating and purging and had the highest conditional probabilities of laxative, diuretic, and enema use. Class 4 (9.5% of the sample, n = 177) consisted mostly of individuals of overweight or obese BMI who endorsed binge eating. Class 5 (8.9% of the sample, n = 163) consisted primarily of individuals with a BMI less

than 19 who were highly likely to endorse excessive fear of weight gain. By contrast, Class 6 (7.9% of the sample, $n = 146$) also consisted mostly of individuals below BMI 19, but endorsement of fear of weight gain was uncommon in this class. The distribution of *DSM-IV* diagnoses across latent classes is shown in Table 6.

Mortality in the overall sample was 4.3%. The SMR for the overall sample was 1.70 (95% C.I., 1.35–2.10) and the suicide standardized mortality ratio was 5.24 (95% C.I., 2.81–9.02). Standardized mortality ratios for the individual classes are shown in Table 5. The highest SMR was observed in Class 6 (3.02; 1.43–5.56) and elevated SMR's were also seen in Class 4 and Class 2. Mortality rates differed significantly between Class 5 and both Class 6 and Class 2 when adjusting for age, race, and sex ($p = .037$ and $p = .047$, respectively). By comparison, mortality for the *DSM*-based diagnostic classifications were as follows: AN 1.70 (0.69–3.51), BN 1.57 (1.09–2.19), EDNOS 1.81 (1.31–2.45).

Discussion

Latent class analysis of this heterogeneous treatment-seeking sample of ED patients yielded a six-class solution. Notably, mortality was elevated in Latent Class 2 (LC2, the class with the highest likelihood of more frequent binge eating and vomiting); Latent Class 4 (LC4, the class with the highest prevalence of binge eating and overweight/obesity), and Latent Class 6 (LC6, the class with low weight and not generally reporting fear of weight gain).

The central question in this study is whether empirically derived classifications provide better predictive validity than those based on the *DSM–IV*. As hypothesized, this study showed that mortality rates were more strongly associated with the latent classes than the *DSM–IV* diagnoses. This appears particularly true for Latent Classes 5 and 6. These classes appear to split a group who would be placed in *DSM–IV* AN or EDNOS into two classes differentiated mostly by presence or absence of fear of weight gain. Class 6 (not reporting fear of weight gain) had a substantially elevated SMR (3.02) while the SMR in Class 5 (with fear of weight gain) was lower (0.56; adjusted OR for mortality: 5.37, $p = .037$); this latter finding is a striking departure from previous work. Because of the nature of the self-report questionnaire used, it is impossible to say with certainty what this represents. It might reflect denial of symptoms, a common phenomenon among people with ED (Vitousek & Stumpf, 2005). Alternatively, it appears that some individuals at low weight with highly restrictive eating behaviors assess themselves as being at low risk for gaining weight while eating so little, and thus express little in the way of fear of weight gain. There has also been interest in the construct of AN without fear of weight gain (Becker, Thomas, & Pike, 2009; Santonastaso et al., 2009) and perhaps that putative group was identified here. A similar group has been identified using latent structure analysis in two other samples (Eddy et al., 2009; Keel et al., 2004). Whatever the cause, this class was associated with substantially elevated mortality, suggesting this as an area worthy of further study.

Elevated mortality in LC4 (resembling BED) is of interest given recent debates about the diagnostic validity of BED (Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009) and provides some evidence about whether the constellation of obesity and binge eating can be separated from other ED. Elevated mortality in LC2, is also of interest, given that methods

of compensatory behaviors often thought to be associated with medical complications (, e.g., laxative or diuretic use) were not particularly common in this group.

What do these results say about the classification of ED? They do not fully support the current nosology. The diagnostic classifications derived empirically in this investigation are best characterized by frequency of binge eating and purging; by the presence of obesity and binge eating; and by low weight status with or without fear of weight gain. These results also serve to emphasize the potential utility of coupling latent structure analysis to prospective validation for other areas of psychopathology.

There are a number of strengths of this study. This was a large, clinically relevant sample composed of all patients seeking treatment over a lengthy period of time. A longitudinal validator, frequently lacking in prior studies, was available. Moreover, this validator, mortality, is one of a great deal of clinical and public health significance. Finally, the use of computerized record linkage allowed for high fidelity measurement of this variable.

There are weaknesses to this study as well. While the longitudinal validator is one of great importance, it is only one data point. Furthermore, while mortality risk is an important question, the relationship between the latent classes derived herein and other measures of longitudinal course is not known. All such analyses are critically dependent on the indicators and validators selected. As such, choosing other indicator variables (other measures of psychopathology, e.g., or personality, or biological variables) might yield different classes. The indicators used are mostly cross-sectional measures of constructs which show some variation over time. This probably reflects a major underlying challenge in constructing symptoms-based diagnostic systems. Similarly, choosing different validators might lead to markedly different validation results. For example, an alternative outcome variable could be the development of type II diabetes mellitus, which could be far more likely in LC4 than in any of the other latent classes. Last, self-reported weight was used and while this may be fairly accurate for those the BN, it may be less so in others.

In summary, this analysis of a large, clinically relevant sample of individuals with ED yields six latent classes with varying patterns of mortality. The results reinforce the public health importance of ED in general and BN and BED in particular, and they suggest a need to further understand the importance of the presence or absence of fear of weight gain with individuals with symptoms resembling AN. The results also further underscore the importance of mortality across the broad range of ED, and the necessity of addressing this risk in all types of ED psychopathology.

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Table 1

Previous Latent Structure Studies in Eating Disorders

Author/year	*N*	Indicators	Validators	Asymptomatic	Low weight/AN	BN-like	BED-like	Other
(Sullivan, Bulik, & Kendler, 1998)	474	BN symptoms	BMI, comorbid psychopathology, personality measures, demographics	0	0	2	2	0
(Bulik, Sullivan, & Kendler, 2000)	1,071	ED symptoms	Demographics, eating/weight variables, comorbidity, personality/attitudinal measures, co-twin risk/resemblance	0	3	1	1	1
(Keel et al., 2004)	1,179	ED symptoms	Demographics, temperament, comorbidity	0	2	2	0	0
(Striegel-Moore, et al., 2005)	234	BN symptoms	BMI, ED diagnosis, EDI subscales, demographics, Stunkard figures, CES-D	0	0	1	1	1
(Wonderlich et al., 2005)	178	Psychiatric disorder and personality scales, 5-HTTLPR genotype	EDE-Q	0	0	3	0	0
(Myers et al., 2006)	125	Impulsivity measures	Comorbidity, trauma history, demographics	0	0	2		
(Wade, Crosby, & Martin, 2006)	1,002	BMI, ED symptoms	MDD, EDE subscales, suicidality, first 16 years of life events	3	1	0	0	1
(Wagner et al., 2006)	55	Personality measures	Comorbid psychopathology, demographics	0	0	0	0	2
(Duncan et al., 2007)	3,723	ED symptoms	Demographics, ED variables, comorbidity, suicidality	1	0	0	0	4
(Mitchell et al., 2007)	403	BMI, ED symptoms	Demographics, EDE-Q subscales	0	1	1	1	2
(Wonderlich et al., 2007)	131	DAPP	Comorbid psychopathology, treatment history, EMA measure	0	0	3	0	0
(Pinheiro, Bulik, Sullivan, & Machado, 2008)	2,028	ED symptoms, BMI, age	EDE-Q subscales, SES	1	0	1	1	1
(Richardson et al., 2008)	89	Lifetime Axis I disorders	EDE, DAPP-BQ, 5-HTTLPR genotype, history of childhood abuse	0	0	2	0	0
(Eddy et al., 2009)	401	BMI, ED symptoms	Demographics, comorbidity, ED symptoms, medical symptoms, treatment utilization history	0	2	2	1	0
(Steiger et al., 2009)	185	DAPP, BIS, CES-D	Demographic, history of childhood abuse, BMI, 5-HTTLPR genotype, ED diagnosis and symptoms, psychiatric medication use	0	0	0	0	3
(Eddy et al., 2008)	401	Percent ideal body weight, ED symptoms	EDE subscales, BDI, self-esteem	0	2	1	0	0
(Thomas, Crosby, Wonderlich, Striegel-Moore, & Becker, 2011)	222	ED symptoms	EDE- Q Clinical Improvement, CES-D	0	0	1	0	1
(Cain, Epler, Steinley, & Sher, 2010)		ED symptoms	Demographics, BMI, BSI, Personal Mastery Scale, positive affect	1	0	1	0	3

Note. BDI = Beck Depression Inventory; BIS = Barratt Impulsivity Scale; CES-D = Center for Epidemiological Studies Depression scale; DAPP = Dimensional Assessment of Personality Pathology; DAPP-BQ = Dimensional Assessment of Personality Pathology Basic Questionnaire; ED = eating disorders; EDE = Eating Disorders Examination; EDE-Q = Eating Disorder Examination Questionnaire; EMA = ecological momentary assessment; LCA = latent class analysis; LPA = latent profile analysis; MDD = major depressive disorder; SES = socioeconomic status.

Table 2

Demographics

	LC1	LC2	LC3	LC4	LC5	LC6
Age, mean (*SD*)	25.3 (6.4)	24.8 (6.6)	25.7 (6.7)	34.6 (8.5)	25.6 (7.3)	25.8 (8.5)
Gender						
Male, *n* (%)	33 (5.3)	19 (3.7)	7 (2.7)	19 (10.7)	7 (4.3)	8 (5.5)
Female, *n* (%)	595 (94.7)	494 (96.3)	251 (97.3)	158 (89.3)	156 (95.7)	138 (94.5)
Race						
White, *n* (%)	600 (95.5)	489 (95.3)	249 (96.5)	153 (86.4)	158 (96.9)	139 (95.2)
African American, *n* (%)	11 (1.8)	4 (0.8)	5 (1.9)	12 (6.8)	0 (0.0)	2 (1.4)
Native American, *n* (%)	1 (0.2)	3 (0.6)	1 (0.4)	0 (0.0)	1 (0.6)	1 (0.7)
Hispanic, *n* (%)	1 (0.2)	3 (0,6)	0 (0.0)	1 (0.6)	1 (0.6)	0 (0.0)
Asian, *n* (%)	2 (0.3)	1 (0.2)	1 (0.4)	1 (0.6)	0 (0.0)	2 (1.4)
Other/unknown, *n* (%)	13 (2.1)	13 (2.5)	2 (0.8)	10 (5.6)	3 (0.2)	2 (1.4)

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Table 3

Estimated Conditional and Marginal Probabilities for Model I

	LC1	LC2	LC3	LC4	LC5	LC6
Cluster size	0.327	0.260	0.151	0.095	0.089	0.079
Current BMI						
≤17.5	0.055	0.109	0.053	0.000	0.545	0.606
17.5–19	0.126	0.173	0.124	0.001	0.229	0.215
19–25	0.652	0.621	0.065	0.071	0.219	0.174
25–30	0.104	0.069	0.106	0.122	0.006	0.004
>30	0.064	0.029	0.066	0.806	0.001	0.000
Binge eating frequency						
Never	0.090	0.000	0.038	0.037	0.613	0.786
<2/Week	0.298	0.000	0.197	0.196	0.314	0.194
2–6/Week	0.419	0.002	0.442	0.442	0.069	0.020
>1/Day	0.192	0.998	0.323	0.323	0.005	0.001
Vomiting frequency						
Never	0.353	0.000	0.246	0.884	0.588	0.826
<2/Week	0.243	0.000	0.221	0.098	0.230	0.136
2–6/Week	0.253	0.001	0.300	0.016	0.135	0.034
>1/Day	0.151	0.999	0.234	0.002	0.046	0.005
Skipping meal frequency						
Never	0.253	0.210	0.109	0.426	0.103	0.381
<2/Week	0.256	0.240	0.177	0.285	0.171	0.283
2–6/Week	0.221	0.232	0.243	0.161	0.243	0.178
>1/Day	0.270	0.318	0.471	0.128	0.484	0.158
Exercising frequency						
Never	0.175	0.225	0.143	0.606	0.089	0.622
<2/Week	0.204	0.225	0.186	0.236	0.147	0.231
2–6/Week	0.302	0.288	0.308	0.117	0.307	0.109
>1/Day	0.319	0.262	0.364	0.041	0.457	0.037
Laxative use						
Yes	0.172	0.309	0.931	0.120	0.409	0.053
Diuretic use						
Yes	0.039	0.094	0.425	0.067	0.141	0.000
Enema use						
Yes	0.002	0.041	0.224	0.011	0.061	0.014
Fear of weight gain						
Yes	0.865	0.920	0.970	0.755	0.980	0.229

Table 4

Bivariate Residuals for a 6-Class Model Including Amenorrhea With Local Dependence

	BMI	Binge	Vomit	Skip meal	Exercise	Laxative	Diuretic	Enema	Fear	Amenorrhea
BMI	•									
Binge	2.7	•								
Vomit	1.9	**5.9**	•							
Skip meal	**4.0**	1.2	3.5	•						
Exercise	2.0	0.1	1.7	**15.8**	•					
Laxative	1.0	0.9	0.5	2.9	0.8	•				
Diuretic	1.5	0.5	1.9	2.1	**4.0**	**4.7**	•			
Enema	1.4	0.3	0.7	2.4	2.8	1.6	1.2	•		
Fear	2.4	1.1	1.0	**6.9**	3.1	1.5	1.5	2.5	•	
Amenorrhea	**17.6**	**28.6**	**29.7**	**7.9**	**13.9**	**9.1**	1.8	2.7	1.9	•

Note. These figures approximate chi-square values. Bivariate residuals >3.8 are bolded to represent significance at $p < .05$.

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Table 5

Mortality of the Latent Classes

	LC1	LC2	LC3	LC4	LC5	LC6
Observed deaths, *n*	24	21	12	15	2	10
Crude mortality rate, %	3.8	4.1	4.7	8.5	1.2	6.8
Cause of death, *n*						
Suicide	6	3	4	0	0	0
Substance use	3	2	2	1	0	2
Trauma	4	2	1	1	0	1
Other medical	11	14	5	13	2	7
SMR	1.55	**1.69**	1.81	**1.85**	0.56	**3.02**
95% CI	0.99–2.31	**1.04-2.58**	0.93–3.16	**1.04-3.06**	0.07–2.03	**1.43-5.56**

Note. SMR = Standardized mortality ratio. Bivariate residuals >3.8 are bolded to represent significance at $p < .05$.

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Table 6

DSM-IV Diagnosis by Latent Classes

	Anorexia nervosa	Bulimia nervosa	Eating disorder not otherwise specified
LC1, *n* (%)	14 (2.2)	270 (43.0)	344 (54.8)
LC2, *n* (%)	37 (7.2)	445 (86.7)	31 (6.0)
LC3, *n* (%)	4 (5.4)	167 (64.7)	77 (29.8)
LC4, *n* (%)	0 (0.0)	24 (13.6)	153 (86.4)
LC5, *n* (%)	97 (59.5)	0 (0.0)	66 (40.5)
LC6, *n* (%)	15 (10.3)	0 (0.0)	131 (89.7)