Different Patterns of Emotional Eating and Visuospatial Deficits Whereas Shared Risk Factors Related with Social Support between Anorexia Nervosa and Bulimia Nervosa

Youl-Ri Kim, Soo-Jin Lim and Janet Treasure

Department of Neuropsychiatry, Seoul Paik Hospital, Inje University College of Medicine, Seoul, Korea

Department of Academic Psychiatry, Guy’s, King’s, and St. Thomas’s Medical School & Institute of Psychiatry, London, UK

Objective  Although it is thought that eating disorders result from the interplay of personal and sociocultural factors, a comprehensive model of eating disorders remains to be established. The aim of this study was to determine the extent to which the childhood factors and deficit in visuoperceptual ability contribute to eating disorders.

Methods  A total of 76 participants - 22 women with anorexia nervosa (AN), 28 women with bulimia nervosa (BN), and 26 healthy women of comparable age, IQ, and years of education - were examined. Neuropsychological tasks were applied to measure the visuoperceptual deficits, viz. the Rey-Osterrieth complex figure test and the group embedded figures test (GEFT). A questionnaire designed to obtain retrospective assessments of the childhood risk factors was administered to the participants.

Results  The women with both AN and BN were less likely to report having supportive figures in their childhood and poor copy accuracy in the Rey-Osterrieth test. The women with AN were more likely to report premorbid anxiety, childhood emotional undereating and showed poor performances in the GEFT. In the final model, the factors independently contributing to the case status were less social support in childhood as a common factor for both AN and BN, and childhood emotional undereating and poor ability in the low-level visuospatial processing for AN.

Conclusion  Our results suggest the disturbance in the food-emotion relationship and the deficit in low-level visuospatial processing in people with AN. Lower social support appears to contribute to an increase in vulnerability to both AN and BN.

Key Words  Childhood risk factors, Emotional eating, Visuospatial ability, Anorexia nervosa, Bulimia nervosa.

INTRODUCTION

The aetiology of eating disorders is complex, with genetic, biological, psychological and sociocultural factors appearing to contribute significantly to their susceptibility. Although it is thought that eating disorders result from the interplay of these different factors, a comprehensive model of eating disorders remains to be established.

There have been a number of studies which examined the role of developmental factors in predicting the onset of eating disorders and their results were summarized in systematic reviews. The putative risk factors for eating disorders include diet vulnerability, less social support, high social anxiety, perfectionism, emotional eating or picky eating. It is unclear to what extent childhood risk factors contribute to eating disorders and what is their relative importance between anorexia nervosa (AN) and bulimia nervosa (BN). Pre-existing cognitive abnormalities may also be relevant to both the development and maintenance of eating disorders. Body image disturbance has been considered to be a key characteristic of patients with both AN and BN, the main neuropsychological component of which is a distorted body perception. A dysfunctional visuoperceptual ability may underpin distorted body perception in AN. Studies of patients with AN have reported impairments of visuospatial ability. The specific profile of visuospatial impairments of AN was in rapid visual information processing, whereas no impairments on tests of spatial span, pattern recog-
nition memory, and spatial working memory. Not only have fewer studies of BN been conducted than of AN, but also their results were more subtle rather than invasive visuospatial cognitive difficulty. Furthermore, the detailed profile of visuospatial impairments and different patterns between AN and BN are uncertain. The stress diathesis model, in which additional environmental factors add to the risk of neuropsychological deficits with regard to obsessive-compulsive disorder, may explain the visuospatial deficits in the aetiological model of eating disorders.

We aimed at the development of coherent model of eating disorders including comprehensive childhood risk factors and visuospatial dysfunction. Thus the present study was designed to answer the following questions; To what extent do childhood risk factors contribute to eating disorders and what is their relative importance between AN and BN: What are the detailed profiles of visuospatial impairment in AN and BN: What is the comprehensive model consisting of both childhood risk factors and visuospatial deficit in AN and BN? The main hypothesis was that patients with AN and BN have certain risk factors in common and that both of them have an impairment of visuospatial ability, but that the pattern of impairment might differ. To test those hypotheses, we chose two neuropsychological tests, the Rey-Osterrieth Complex Figure Test and the Group Embedded Figures Test which were used for measuring the detailed profiles of visuospatial ability in previous studies. We performed a retrospective assessment of various eating disorder-specific-childhood risk factors compiled from an extensive review of the literature made by one of the researchers (J.T.).

METHODS

Study population
A total of 76 participants, including 22 women with AN and 28 women with BN as well as 26 healthy women, were recruited. The diagnoses of eating-disorder patients were as per the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) criteria using the Korean version of the Structured Clinical Interview for DSM-IV Axis I disorders (SCID). Among the AN patients, 15 (68%) had the restrictive subtype and 7 (32%) the purging subtype. Seven (25%) BN patients had a history of AN, whereas none of the AN patients had a history of BN. The controls’ exclusion criteria were the past or current clinically significant eating disorder symptoms and any current psychiatric disorders. The exclusion criteria for all participants were histories of neurologic illness, brain injury, substance abuse disorder, or psychosis. We permitted only the use of selective serotonin reuptake inhibitors (SSRIs) within 2 weeks before the neuropsychological test, as evidence suggests antidepressant medication has a relatively negligible effect on the cognitive profile. Eight (36%) with AN and 16 (57%) with BN were taking SSRIs at the time of the study. This study was approved by the Institutional Review Board of Seoul Paik Hospital in Seoul, Korea. We obtained written informed consent from all of the participants.

Procedure
We matched the groups in terms of general intelligence ability and years of education, administering the Korean version of the Wechsler Adult Intelligent Scale to them to ascertain the former. We measured participants’ depression, anxiety, and obsessionality levels via Korean versions of the Beck Depression Inventory, the Spielberger State and Trait Anxiety Inventory, and the Maudsley Obsessive Compulsive Inventory, respectively. The patients underwent interviews using the Korean version of the 12th edition of the Eating Disorders Examination (EDE) to measure their objective eating-disorder symptoms.

Measures
Childhood risk factors questionnaire
The questionnaire focused on the period before onset of eating disorders to ensure that the exposure preceded the development of the eating disorder. The questionnaire comprised 36 questions, divided into 8 domains which covered (i) Parents or adults concern about thinness (ii) Parents or adults teasing about weight or shape (iii) Social support (iv) Anxiety (v) Perfectionism (vi) Emotional overeating (vii) Emotional undereating (viii) Picky eating. The psychometric properties of the Korean version was assessed with 2 week test-retest reliability for the 8 factors ranging between 0.64 and 0.87, and Cronbach’s alpha coefficient ranging between 0.70 and 0.91.

Group Embedded Figures Test
We applied the group version of the EFT to subjects individually. As no working memory is required in the Group Embedded Figures Test (GEFT), the test is a good perceptual measure for exploring analytical ability in a low-level visuospatial processing modality. The GEFT procedure called for participants to mark the hidden target shapes that were embedded within more complex stimulus patterns, as many times as appropriate. Each participant could check the simple figure against the complex design throughout the whole task. The score was the number of shapes participants correctly located during the given time. The Korean version contains 5 simple geometric forms and allows a maximum of 10 minutes for completing each of the two sections, with 18 complex designs per section. Scores ranged from 0-18 correctly-located shapes per section. According to Korean normative data, the test/retest reliability coeffi-
cient of the Korean version of the GEFT was 0.67, and Cronbach’s alpha was 0.82.34

Rey-Osterrieth Complex Figure Test
The Rey-Osterrieth Complex Figure Test22 is an open-ended measure of visuospatial constructional ability and visual memory.35 In the first stage of this testing procedure, the participants saw a figure and copied it, without knowing that they would be asked to remember it. The second stage, free-recall test took place after 30 minutes (the delayed condition). We used the system Meyer and Meyer26 developed to calculate the score for each condition, evaluating 18 segments of the figures using criteria such as location and accuracy. There were two criteria for each segment, each worth 1 point, resulting in a score range of 0-36.

Statistical analyses
The three groups (AN, BN, controls) were compared in terms of their clinical variables using analysis of variance and t-tests, as appropriate for the measurements. Multinomial logistic regression with the independent variables consisting of the childhood risk factors, RCFT and GEFT was used to identify those factors which have an impact on the group membership, with the controls used as a reference group. We used multivariate logistic regression analyses using the forward variable selection strategy on the putative factors, in order to find those factors independently contributing to the case status. Two-tailed tests with a 5% level of significance were used throughout the analyses.

RESULTS

Demographic and clinical characteristics
Table 1 shows the participants' clinical and demographic characteristics. The AN group had a lower lifetime weight history, whereas the BN group had a more fluctuating lifetime weight history. Both the AN and BN groups had higher scores for depressed mood and for state and trait anxiety than did the nonclinical group. They showed a tendency toward higher obsessive-compulsiveness, but the difference was statistically minimal (the post-hoc Tukey test: p=0.046 for AN vs. controls: p=0.061 for BN vs. controls). The BN group had higher EDE scores than the AN group did, particularly in the subscale of weight concern.

Childhood risk factors
As shown in Table 2, both the AN and BN patients were less likely to report social support (p=0.008 for AN vs. controls: p=0.001 for BN vs. controls). The AN patients were more likely to report childhood emotional undereating (p=0.023) and premor-

Table 1. The clinical and demographic characteristics of the study population

| Variables                  | Women with AN (N=22) | Women with BN (N=28) | Healthy women (N=26) | Analysis |
|----------------------------|----------------------|----------------------|----------------------|----------|
| Age, years                 | 22.00 (6.96)         | 23.04 (4.57)         | 23.46 (4.14)         | F (2,73) 0.482 0.620 |
| Duration of illness, months | 43.52 (50.66)       | 38.50 (34.65)        | n/a                  | t (48)=0.403 0.689 |
| Years of education, years  | 12.82 (3.07)         | 13.54 (2.10)         | 13.42 (0.99)         | 0.755 0.473 |
| WAIS-IQ                    | 105.17 (9.14)        | 110.88 (12.23)       | 113.04 (10.52)       | 2.883 0.063 |
| Body mass index, kg/m²     |                      |                      |                      |          |
| Current                    | 15.63 (1.47)         | 20.40 (2.72)         | 21.36 (2.78)         | 36.649 <0.001 |
| Lowest ever                | 14.05 (1.54)         | 17.35 (2.64)         | 19.28 (2.16)         | 34.130 <0.001 |
| Highest ever               | 21.19 (3.04)         | 23.82 (3.03)         | 23.37 (3.47)         | 4.559 0.014 |
| Beck depression inventory  | 17.33 (13.14)        | 17.20 (13.07)        | 4.15 (3.60)          | 12.507 <0.001 |
| STAI, total score          | 105.43 (24.57)       | 105.48 (24.60)       | 72.38 (12.28)        | 20.638 <0.001 |
| State                      | 50.71 (11.60)        | 51.16 (13.88)        | 34.35 (6.98)         | 18.538 <0.001 |
| Trait                      | 54.71 (13.92)        | 54.32 (11.73)        | 38.04 (6.89)         | 18.591 <0.001 |
| MOCI                       | 10.14 (4.78)         | 8.00 (5.52)          | 6.73 (3.96)          | 2.981 0.057 |
| EDE, global score          | 59.73 (22.47)        | 79.78 (19.14)        | n/a                  | t (48)=3.374 0.001 |
| Restraint                  | 12.18 (8.04)         | 12.52 (6.90)         | n/a                  | t (48)=0.158 0.875 |
| Eating concern             | 14.05 (9.08)         | 18.52 (7.15)         | n/a                  | t (48)=1.930 0.060 |
| Weight concern             | 21.36 (11.49)        | 33.85 (10.06)        | n/a                  | t (48)=4.056 <0.001 |
| Shape concern              | 12.14 (4.68)         | 15.00 (5.74)         | n/a                  | t (48)=1.884 0.066 |

Data are shown as means (standard deviation). Analysis was by ANOVA or t-test as appropriate. *p<0.05, †p<0.01 in post-hoc Tukey test, as contrasted with controls. AN: Anorexia Nervosa, BN: Bulimia Nervosa, WAIS: Wechsler Adult Intelligence Scale, STAI: State and Trait Anxiety Inventory, MOCI: Maudsley Obsessive and Compulsive Inventory, EDE: Eating Disorders Examination Interview, n/a: not associated.
Visuospatial Deficits and Less Social Support in Eating Disorders

Table 2. Comparisons of the retrospective childhood risk factors in the AN and BN patients and the healthy controls

| Variables                                  | Women with AN (N=22) | Women with BN (N=28) | Healthy women (N=26) | Comparison with healthy women |
|--------------------------------------------|-----------------------|-----------------------|----------------------|-------------------------------|
| Parents/adults concerned about thinness    | 2.05 (3.40)           | 1.92 (2.50)           | 1.04 (1.37)          | 2.690 0.261 1.22 (0.93-1.61) 1.20 (0.92-1.57) |
| Parents/adults teasing about weight and shape | 1.86 (2.46)           | 1.60 (1.85)           | 1.00 (1.33)          | 2.793 0.247 1.30 (0.93-1.83) 1.23 (0.88-1.71) |
| Social support                             | 12.29 (8.31)          | 10.72 (7.07)          | 18.08 (5.23)         | 14.590 0.001 0.89 (0.81-0.97)* 0.86 (0.79-0.94)* |
| Anxiety                                    | 1.57 (2.16)           | 0.92 (1.50)           | 0.31 (0.68)          | 9.127 0.010 2.15 (1.14-4.04)* 1.75 (0.93-3.28) |
| Perfectionism                              | 11.67 (5.45)          | 12.64 (5.62)          | 9.77 (4.94)          | 3.843 0.146 1.07 (0.96-1.20) 1.11 (1.00-1.24) |
| Emotional undereating                      | 2.43 (2.82)           | 1.35 (2.60)           | 0.73 (1.12)          | 9.286 0.010 1.46 (1.05-2.02)* 1.18 (0.86-1.63) |
| Emotional overeating                       | 1.95 (2.84)           | 3.92 (2.91)           | 3.58 (2.87)          | 6.187 0.045 0.80 (0.64-1.01) 1.04 (0.86-1.25) |
| Picky eating                               | 3.20 (2.02)           | 3.40 (3.33)           | 4.27 (4.50)          | 1.260 0.533 0.92 (0.77-1.09) 0.93 (0.80-1.10) |

Data are shown as mean (standard deviation). The likelihood ratio statistic, \( \chi^2 \) (with 2 df) and its p value are given for each variable, with the healthy controls used as a reference group. *indicate odds ratios that were significantly different from 1, according to Wald tests. AN: anorexia nervosa, BN: bulimia nervosa, CI: confidence interval

Table 3. Comparisons of the visuospatial ability of the AN and BN patients with the healthy controls

| Variables                   | Women with AN (N=22) | Women with BN (N=28) | Healthy women (N=26) | Comparison with healthy women |
|-----------------------------|----------------------|----------------------|----------------------|-------------------------------|
| GEFT                        | 9.69 (6.67)          | 15.68 (10.06)        | 15.77 (6.99)         | 8.479 0.014 0.91 (0.84-0.98)* 1.00 (0.94-1.07) |
| RCFT Copy accuracy          | 34.98 (1.27)         | 34.95 (1.12)         | 35.56 (0.74)         | 6.292 0.043 0.51 (0.26-1.00)* 0.49 (0.26-0.95)* |
| RCFT Recall accuracy        | 20.80 (6.22)         | 23.75 (5.23)         | 22.67 (4.36)         | 3.899 0.142 0.94 (0.84-1.05) 1.04 (0.94-1.16) |

Data are shown as mean (standard deviation). The likelihood ratio statistic, \( \chi^2 \) (with 2 df) and its p value are given for each variable, with the healthy controls used as a reference group. *indicate odds ratios that were significantly different from 1, according to Wald tests. AN: anorexia nervosa, BN: bulimia nervosa, CI: confidence interval, GEFT: group embedded figures test, RCFT: Rey-Osterrieth complex figure test

Table 4. Factors independently affecting case status compared with healthy controls in final model

| Identified factors                     | Women with AN | Women with BN |
|----------------------------------------|---------------|---------------|
| Childhood risk factors                 |               |               |
| Social support                         | 0.839 (0.735-0.957) | 0.829 (0.737-0.933) |
| Emotional undereating                  | 1.998 (1.170-3.412) | -- |
| Visuospatial ability                   |               |               |
| Low level of visuospatial processing   | 0.892 (0.805-0.988) | -- |
| Copy accuracy                          | --            | --            |

Low-level of visuospatial processing was derived from the group embedded figures test. Copy accuracy was from the Rey-Osterrieth complex figure test. *odds ratio (95% CI) was shown using forward selection in the regression analyses model. AN: Women with anorexia nervosa, BN: Women with bulimia nervosa, CI: confidence interval

bid anxiety (p=0.018).

**Visuospatial ability**

Table 3 shows the results of the neuropsychological tests. On GEFT, the AN group showed poor ability to identify correct shapes during the given time as compared to the healthy controls (p=0.015). On RCFT, both patient groups showed poorer copy accuracy than the healthy controls did; this finding was less prominent in the AN group (p=0.049 for AN vs. controls; p=0.036 for BN vs. controls). The difference in recall accuracy between the patients and the controls did not reach statistical significance. In a subsidiary analysis to investigate the influence of medication state on performance, comparisons between the eating disorder patients who were and were not...
currently receiving antidepressants revealed no significant group differences across the neuropsychological tasks (t=1.145, df=48, p=0.258 for GEFT; t=0.010, df=48, p=0.992 for copy accuracy on RCFT).

Independent contribution of identified factors to case status in final model

The identified neuropsychological variables and childhood risk factors obtained from the univariate analyses were entered into the regression model analyses (Table 4). The childhood risk factors were entered first, followed by the neuropsychological variables. Childhood emotional undereating (p=0.011), less social support (p=0.009) and poor performance in the GEFT (p=0.029) contributed independently to the case status of AN. The factor independently contributing to BN case status was less social support in childhood (p=0.002).

DISCUSSION

This study furthers our understanding of the aetiological model of eating disorders. In our study, the AN patients were more likely to report childhood emotional undereating and performed poorly on GEFT. Both the AN and BN patients were more likely to report lower social support, which factors appear to contribute to an increase in vulnerability to eating disorders case status.

Emotional eating involves integrated emotional regulation, reward processing, and interoception. In our retrospective study, childhood emotional undereating strongly contributed to AN case status, which supports the idea of a trait-related dysfunction in the brain appetite circuit in AN, as proposed by Kaye et al.47 Whereas carbohydrate intake reduces anxiety by increasing extracellular serotonin concentration in the brain in a healthy human,38,39 it can stimulate dysphoric mood in AN people.40 Whereas childhood social support was an independent risk factor of eating disorder case status, it is interesting that our model did not include the factors related to diet vulnerability. Their role may be that of a trigger in the onset of eating disorders.41

In the neuropsychological data, our findings are consistent with previous studies of poorer performance on GEFT in AN,42 which contradict the results of Lopez et al.24 This discrepancy may be attributable to the differences in sample chronicity between theirs and ours (duration of illness: 13.08±11.2 years vs. 3.63±4.2 years, respectively) and methodology details (EFT vs. the group version of EFT, respectively). Those severely impaired AN patients may have a different cognitive impairment pattern from other AN patients.45 Thus, the results of Lopez et al.24 may more properly explain treatment-resistant, chronic AN cases. In addition, the AN group might have felt the pressure of the time limitation of the group version than the non-clinical group did, which could lead to their poor performance in the test. Our results on RCFT in BN is generally consistent with previous studies in respect to poorer accuracy on the copy phase46 and no visuospatial memory impairments.46 It appears that the visuospatial dysfunction in eating disorder were subtle, but overall our findings suggest dysfunctions in low-level visuospatial processing in AN case status. Our results could be explained based on the stress diathesis model, in which the lower social support may add to the risk of the dysfunction of low-level of visuoperceptual processing.

However, the present study contained a few limitations, which need to be considered. The first is the retrospective assessment of the risk factors. Although we carefully assessed the risk factors with a focus on the period of childhood before the onset of the patients’ disorders, to ensure that the period of risk factor exposure preceded the eating disorder's development, such soft judgments as over-concern and expectations may be biased by retrospective recall or affected by the patient's current state. Second, we might not have included all of the risk factors relevant to a more comprehensive neurodevelopmental hypothesis, i.e., perinatal events. Third, we could not use more comprehensive tasks as measures of visuospatial ability. Fourth, the sample size was relatively small.

In conclusion, these findings add to the growing body of evidence for a complex etiologial model of eating disorders. Our results suggest that the disturbance in the food-emotion relationship and the deficit in low-level visuospatial processing in people with AN. Lower social support appears to contribute to an increase in vulnerability to both AN and BN.

Acknowledgments

We wish to thank Drs Carolina A López and Amy J Harrison for their advice on the study. A Korea Research Foundation Grant funded by the Korean Government (MOEHRD) (KRF-2006-331-E00203) supported this work.

REFERENCES

1. Connan F, Campbell IC, Katzman M, Lightman SL, Treasure J. A neordevelopmental model for anorexia nervosa. Psychol Behav 2003;79: 13–24.
2. Fairburn CG, Harrison PJ. Eating disorders. Lancet 2003;361:407–416.
3. Jacobi C, Hayward C, de Zwaan M, Kraemer HC, Agras WS. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. Psychol Bull 2004;130:19–65.
4. Stice E. Risk and maintenance factors for eating pathology: a meta-analytic review. Psychol Bull 2002;128:825–848.
5. Patton GC, Johnson-Sabine E, Wood K, Mann AH, Wakeling A. Abnormal eating attitudes in London schoolgirls—a prospective epidemiological study: outcome at twelve month follow-up. Psychol Med 1990;20:383–394.
6. Tiller JM, Sloane G, Schmidt U, Troop N, Power M, Treasure JL. Social support in patients with anorexia nervosa and bulimia nervosa. Int J Eat Disord 1997;21:31–38.
7. Troop NA, Treasure JL. Setting the scene for eating disorders. II. Childhood helplessness and mastery. Psychol Med 1997;27:531–538.
8. Bulik CM, Sullivan PF, Fear JL, Joyce PR. Eating disorders and anteced-
Visuospatial Deficits and Less Social Support in Eating Disorders

dent anxiety disorders: a controlled study. Acta Psychiatr Scand 1997;96:101-107.
9. Fairburn CG, Cooper Z, Doll HA, Welch SL. Risk factors for anorexia nervosa: three integrated case-control comparisons. Arch Gen Psychiatry 1999;56:468-476.
10. Johnson E, Wardle J. Dietary restraint, body dissatisfaction, and psychological distress: a prospective analysis. J Abnorm Psychol 2005;114:119-125.
11. Kotler LA, Cohen P, Davies M, Pine DS, Walsh BT. Longitudinal relationships between childhoodhood, adolescent, and adult eating disorders. J Am Acad Child Adolesc Psychiatry 2001;40:1434-1440.
12. Lena SM, Fiocco AJ, Leyenaar JR. The role of cognitive deficits in the development of eating disorders. Neuropsychol Rev 2004;14:99-113.
13. Schmidt U, Treasure J. Anorexia nervosa: valued and visible. A cognitive-interpersonal maintenance model and its implications for research and practice. Br J Clin Psychol 2006;45:343-366.
14. Cash TF, Deagle EA 3rd. The nature and extent of body-image disturbances in anorexia nervosa and bulimia nervosa: a meta-analysis. Int J Eat Disord 1997;22:107-125.
15. Garner DM, Garfinkel PE. Bodyimage in anorexia nervosa: measurement, theory and clinical implications. Int J Psychiatry Med 1980;11:263-284.
16. Wagner A, Ruf M, Braus DE, Schmidt MH. Neuronal activity changes and body image distortion in anorexia nervosa. Neuroreport 2003;14:2193-2197.
17. Gillberg IC, Gillberg C, Råstam M, Johansson M. The cognitive profile of anorexia nervosa: a comparative study including a community-based sample. Compr Psychiatry 1996;37:23-30.
18. Kingston K, Szumukler G, Andrews D, Tress B, Desmond P. Neuropsychological and structural brain changes in anorexia nervosa before and after refeeding. Psychol Med 1996;26:15-28.
19. Fowler L, Blackwell A, Jaffa A, Palmer R, Robbins TW, Sahakian BJ, et al. Profile of neurocognitive impairments associated with female in-patients with anorexia nervosa. Psychol Med 2000;36:517-527.
20. Jones BP, Duncan CC, Brouwers P, Mirsky AF. Cognition in eating disorders. J Clin Exp Neuropsychol 1991;13:711-728.
21. Grisham JR, Anderson TM, Poulton R, Moffitt TE, Andrews G. Childhood neuropsychological deficits associated with adult obsessive-compulsive disorder. Br J Psychiatry 2009;195:138-141.
22. Oosterhi L. The test of copying a complex figure: a contribution to the study of perception and memory. Arch Psychol 1944;30:206-356.
23. Witkin H, Oltman P, Raskin E, Karp SA. Group Embedded Figures Test. California: Consulting Psychologists Press; 1971.
24. Cassano GB, Puca F, Scapicchio PL, Trabucchi M. Italian Study Group on Depression in Elderly Patients. Paroxetine and fluoxetine effects on mood and cognitive functions in depressed nondemented elderly patients. J Clin Psychiatry 2002;63:396-402.
25. Yum TH, Park YS, Oh KJ, Kim JG, Lee HY. The manual of Korean-Wechsler adult intelligence scale. Seoul: Korean Guidance Press; 1992.
26. Han HM, Yeon TH, Shin YW. The validity and reliability of Beck Depression Inventory Korean version. J Korean Neuropsychiatr Assoc 1986; 25:487-500.
27. Kim JT, Shin DK. A study based on the standardization of the State and Trait Anxiety Inventory for Korea. New Med J 1978;21:69-75.
28. Min BB, Won HT. Reliability and validity of the Korean translations of Maadles obsessional-compulsive inventory and Padua inventory. Korean J Clin Psychol 1999;18:163-182.
29. Heo SY, Lee MK, Choi YM, Sohn CH, Lee HK, Lee YH. Reliability and factor analysis of the Korean version of Eating Disorder Examination. J Korean Soc Study Obes 2004;13:42-52.
30. Witkin H, Oltman P, Raskin E, Karp SA. Manual for the Embedded Figures Test. California: Consulting Psychologists Press; 1971.
31. Chon YS, Jang HP. Group embedded figures test. Seoul: Korean Testing Centre; 1983.
32. Shin MS, Park SY, Park SR, Seo SH, Kwon JS. Clinical and empirical applications of the Rey-Osterrith Complex Figure Test. Nat Protoc 2006;1:892-899.
33. Meyer J, Meyer K. Rey Complex Figure Test and Recognition Test: Professional Manual. Odessa, FL: Psychological Assessment Resources; 1995.
34. Kaye WH, Fudge JL, Paulus M. New insights into symptoms and neuro-circuit function of anorexia nervosa. Nat Rev Neurosci 2009;10:573-584.
35. Young SN, Gauthier S. Effect of tryptophan administration on tryptophan, 5-hydroxyindoleacetic acid and indoleacetic acid in human lumbar and cisternal cerebrospinal fluid. J Neurol Neurosurg Psychiatry 1981;44:332-338.
36. Tauscher J, Bagby RM, Javanmard M, Christensen BK, Kasper S, Kapur S. Inverse relationship between serotonin 5-HT(1A) receptor binding and anxiety: a [(11)C]WAY-100635 PET investigation in healthy volunteers. Am J Psychiatry 2001;158:1326-1328.
37. Kaye WH, Barbarich NC, Putnam K, Gendall KA, Fernstrom J, Fernstrom M, et al. Anxiolytic effects of acute tryptophan depletion in anorexia nervosa. Int J Eat Disord 2003;33:237-267; discussion 268-270.
38. Pike KM, Hilbert A, Willey DE, Fairburn CG, Dohms FA, Walsh BT, et al. Toward an understanding of risk factors for anorexia nervosa: a case-control study. Psychol Med 2008;38:1443-1453.
39. Basseches HI, Karp SA. Field dependence in young anorectic and obese women. Psychother Psychosom 1984;4:133-37.
40. McLaughlin EF, Karp SA, Herzog DB. Sense of ineffectiveness in women with eating disorders: a clinical study of anorexia nervosa and bulimia. Int J Eat Disord 1985;4:511-523.
41. Jones BP, Duncan CC, Brouwers P, Mirsky AF. Cognition in eating disorders. J Clin Exp Neuropsychol 1991;13:711-728.
42. Kingston K, Szumukler G, Andrews D, Tress B, Desmond P. Neuropsychological and structural brain changes in anorexia nervosa before and after refeeding. Psychol Med 1996;26:15-28.
43. Lopez C, Tchanturia K, Stahl D, Treasure J. Central coherence in women with bulimia nervosa. Int J Eat Disord 2008;41:340-347.
44. Bosanac P, Kurlender S, Stojanovsa L, Hallam K, Norman T, McGrath C, et al. Neuropsychological study of underweight and “weight-recovered” anorexia nervosa compared with bulimia nervosa and normal controls. Int J Eat Disord 2007;40:613-621.