The purpose of the present study was to clarify genetic and environmental origins of psychological traits of eating disorders using a Japanese female twin sample. Participants were 162 pairs of female twins consisting of 116 pairs of monozygotic (MZ) twins and 46 pairs of dizygotic (DZ) twins in their adolescence. Psychological traits of eating disorders were assessed with five subscales of the Eating Disorder Inventory (EDI). As a result of using univariate twin analyses, among five subscales of EDI (maturity fears, ineffectiveness, interpersonal distrust, interoceptive awareness, and perfectionism), perfectionism showed significant additive genetic contributions and individual specific environmental effects. On the other hand, maturity fears, ineffectiveness, interoceptive awareness, and interpersonal distrust indicated significant shared environment contributions and individual specific environment effects. The results suggest the importance of both genetic and shared environmental influences on psychological traits of eating disorders in the present study.

Eating disorders including anorexia nervosa (AN) and bulimia nervosa (BN) have a characteristic psychopathy with serious physical symptoms (Wakeling, 1996). AN, which is characterized by food refusal, amenorrhea and conspicuous weight loss, was first described in Western cultures and was hardly reported in non-Western cultures (Lee, 1996; Srinivasan et al., 1998). However, previous studies of eating disorders in the sociocultural theory have been reporting that case reports of eating disorders are increasing in non-Western cultures including Japan (Lee, 1995; Mukai et al., 1994).

A large number of epidemiological studies of eating disorders in the normal population, using the Eating Attitudes Test (EAT; Garner & Garfinkel, 1979) and the Eating Disorder Inventory (EDI; Garner et al., 1983), have examined risk factors influential in AN and BN. For instance, research showed that certain attitudes and behaviors such as a poor body image and weight loss were risk factors for AN (Grant & Fodor, 1986; Paxton et al., 1991) and that psychological traits such as perfectionism and personality (e.g., negative emotionality and neuroticism) had a relationship with eating disorders (Bulik et al., 2003; Klump et al., 2002a; Wade et al., 2000). Longitudinal studies using the EDI subscales also indicated that the longitudinal risk factors (e.g., poor interoceptive awareness and perfectionism) were associated with disordered eating (Leon et al., 1995; Vohs et al., 1999).

On the other hand, some twin studies examined the genetic epidemiology of eating disorders (Holland et al., 1984; Kendler et al., 1991). For instance, it has been reported that the heritability of AN was about 80% of the variance in a twin female sample with eating disorders (Holland et al., 1988). Rutherford et al. (1993) accounted for the heritability of eating disorders in a female volunteer twin population using EAI and EDI. The results showed that the heritability of the subscales of EDI ranged from 25% to 52% and body dissatisfaction measured by EDI subscale had the highest heritability.

Recently, Wade et al. (1999) suggested that liability for the development of behaviors and attitudes of eating disorders was best explained by genetic factors and non-shared environment. It also has been reported that genetic factors influenced the risk for AN using the Virginia Twin Registry (Wade et al., 2000) and the Danish Twin Registry (Kortegaard et al., 2001). Klump et al. (2000) reported that the relationship between BMI and the EDI subscales in the younger twins group was primarily mediated by genetic effect (and also some shared environment), and in the older twins group common genetic effect (and also some individual specific environment) primarily mediated the relationships.

Although previous twin studies have suggested that additive genetic and individual specific environment were the best explanations of AN and BN (Bulik et al., 2000; Klump et al., 2002b), with some possible additional roles for the shared environment with respect to BN, there has been little evidence to clarify genetic and environmental
A Twin Study of Genetic and Environmental Influences on Psychological Traits of Eating Disorders in a Japanese Female Sample

The sample used in the present study was composed of 162 complete female twin pairs, 116 of whom were monozygotic (MZ) pairs and 46 dizygotic (DZ) pairs. Zygosity was determined by the questionnaire (Ooki et al., 1990). The questionnaire consisted of questions about the twin's resemblance (i.e., Were you and your twin "as alike as two peas in a pod?" Did people mistake the identity of you and your twin as children? If so, by whom were you mistaken?) and established zygosity with 93.2% accuracy. For twin pairs in whom zygosity was borderline, the polymorphism of D4DR and serotonin transporter genes (5-HTT) were examined, providing an accuracy zygosity diagnosis up to 97.8%. The average ages of the sample were 21.2 years (± 4.3).

Measurement

Self-reported eating attitudes and behaviors were assessed with EDI. This measurement consists of three subscales (drive for thinness, bulimia, and body dissatisfaction) and five subscales evaluating psychological traits of eating disorders: interoceptive awareness, ineffectiveness, maturity fears, perfectionism, and interpersonal distrust. To assess psychological traits of eating disorders, five subscales of EDI were administered to the twin participants in this study. In addition, they were told to respond to the questionnaire independently, without discussing their responses with one another. This measurement uses a six-point Likert scale (6 = true; 1 = false). The reliability coefficients (α) of the five subscale of EDI were .85 (interoceptive awareness), .73 (ineffectiveness), .56(maturity fears), .61(perfectionism), and .78 (interpersonal distrust).

Statistical Analysis

The effect of additive genetic (A) can be assumed to be the total of multiple genes (polygene) whose effects are small and additive to a quantitative phenotype. Because MZ twins share all their genes and DZ twins share only half of their genes, (A) should cause intrapair correlation in MZ twins to be twice as large as that in DZ twins. Shared environment (C or common environment) is the environmental effect that makes family members alike with the common elements shared by all family members. Assuming that MZ twins and DZ twins share their environment to the same extent, (C) should cause the correlation in MZ twins and DZ twins to be equal. Individual specific environment (E) is the environmental effect that is unique to each member of the family. It also includes measurement errors. These latent variables influence observed variables through the paths a, c and e respectively. The covariance of eating disorders is shared in a proportion a², c², and e² reflecting each of these paths.

In the univariate models, we specify three sub-models, ACE, AE, and CE (A: additive genetic, C: shared environment, E: individual specific environment). We fit the ACE, AE and CE model. The choice of the preferred model is based on Akaike’s information criteria (AIC) computed as \( \chi^2 - 2 \Delta df \) (degree of freedom). AIC reflects model’s goodness of fit as well as parsimony of the model. AIC is calculated for each model and the model that computes the smallest AIC is regarded as the best fit.

Intraclass correlation coefficients of the phenotypic scores were computed by SPSS. Structural equation modeling using the statistical programs EQS, which use the maximum likelihood estimation technique to compute the specific latent variable loading based on the covariance matrix, were used to compute the contributions of genetic and environment factors.
**Results**

The ratings on each of EDI were summed up to compute intercorrelations in the twin sample. The sums of the scores of EDI subscales (perfectionism, maturity fears, ineffectiveness, interoceptive awareness, and interpersonal distrust) were positively skewed. Therefore, the data reported here were based on a log (base 10) transformation.

Table 1 shows the intercorrelations between EDI sub-scales and the twin intraclass correlations of EDI sub-scales. Ineffectiveness was highly correlated with interoceptive awareness and interpersonal distrust — $r$ (Ineffectiveness, interoceptive awareness) = .49; $r$ (Ineffectiveness, interpersonal distrust) = .51.

For perfectionism and interpersonal distrust, maturity fears, and ineffectiveness, the MZ correlation exceeded those of DZ. By contrast, for interoceptive awareness, the DZ correlation exceeded those of MZ.

However, since the MZ and DZ correlation were almost similar for most EDI sub-scales, we estimated the contributions of genetic and environmental factors in much greater detail using a model-fitting approach.

The computer program SPSS was used to compute the MZ and DZ covariance for EDI sub-scales. We next fitted a number of univariate model-fitting analyses for EDI sub-scales using the computer package EQS. The $\chi^2$ and AIC statistics of three parameters (ACE, AE, and CE) and the relative contributions of genetic and environmental parameters with 95% confidence intervals for EDI sub-scales are shown in Table 2.

The AE model provided an appropriate fit to the data for perfectionism and its AIC indicated the most parsimonious explanation. On the other hand, the CE model provided an appropriate fit to the data for maturity fears, ineffectiveness, interoceptive awareness, and interpersonal distrust. Its AIC indicated the most parsimonious explanation. Perfectionism was under the modest influence of additive genetic factors (37%). For maturity fears, ineffectiveness, interoceptive awareness, and interpersonal distrust, these contributions of shared environment were moderate and estimated at 30% to 50%.

**Discussion**

The results of this study indicated that genetic, shared environmental, and individual specific environmental factors each influenced psychological traits of eating disorders. Our finding also suggested that the mechanism regarding genetic and environmental influences was different among five sub-scales of EDI. In working on AN or BN patients with psychological problems, it may be important to consider this heterogeneous mechanism of psychological traits of eating disorders.

Considering this genetic component, perfectionism was moderately heritable (37%). Previous studies reported that the association between the genetic effects and genes was obscure (Buins-slot et al., 1998). However, subsequently several candidate genes (e.g., 5HT2A) have been identified with regards to eating disorders (Nishiguchi et al., 2001). These findings for genetic effects of eating disorders have been reported further. It is also possible that the genetic

### Table 1

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>MZ</th>
<th>DZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>1)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.38</td>
<td>0.12</td>
</tr>
<tr>
<td>2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.43</td>
<td>0.39</td>
</tr>
<tr>
<td>3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.47</td>
<td>0.44</td>
</tr>
<tr>
<td>4)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.41</td>
<td>0.45</td>
</tr>
<tr>
<td>5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.39</td>
<td>0.31</td>
</tr>
</tbody>
</table>

Note: * $p < .01$

### Table 2

<table>
<thead>
<tr>
<th></th>
<th>$\chi^2$</th>
<th>$p$</th>
<th>AIC</th>
<th>$\chi^2$</th>
<th>$p$</th>
<th>AIC</th>
<th>$\chi^2$</th>
<th>$p$</th>
<th>AIC</th>
<th>$\chi^2$</th>
<th>$p$</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perfectionism</td>
<td>6.03</td>
<td>0.11</td>
<td>0.03</td>
<td>6.05</td>
<td>0.15</td>
<td>-1.95</td>
<td>6.83</td>
<td>0.20</td>
<td>-1.17</td>
<td>0.37</td>
<td>0.66</td>
<td>(0.23–0.77)</td>
</tr>
<tr>
<td>Maturity Fears</td>
<td>9.35</td>
<td>0.02</td>
<td>3.35</td>
<td>11.86</td>
<td>0.02</td>
<td>3.86</td>
<td>9.35</td>
<td>0.05</td>
<td>1.35</td>
<td>0.43</td>
<td>0.57</td>
<td>(0.28–0.71)</td>
</tr>
<tr>
<td>Ineffectiveness</td>
<td>6.30</td>
<td>0.10</td>
<td>0.30</td>
<td>8.46</td>
<td>0.08</td>
<td>0.46</td>
<td>6.60</td>
<td>0.16</td>
<td>-1.40</td>
<td>0.47</td>
<td>0.53</td>
<td>(0.34–0.66)</td>
</tr>
<tr>
<td>Interoceptive Awareness</td>
<td>4.57</td>
<td>0.21</td>
<td>-1.43</td>
<td>6.77</td>
<td>0.16</td>
<td>-1.33</td>
<td>4.82</td>
<td>0.31</td>
<td>-3.18</td>
<td>0.43</td>
<td>0.57</td>
<td>(0.29–0.71)</td>
</tr>
<tr>
<td>Interpersonal Distrust</td>
<td>7.68</td>
<td>0.88</td>
<td>1.68</td>
<td>7.83</td>
<td>0.88</td>
<td>-0.17</td>
<td>6.83</td>
<td>0.87</td>
<td>-1.17</td>
<td>0.34</td>
<td>0.86</td>
<td>(0.20–0.54)</td>
</tr>
</tbody>
</table>

The $\chi^2$ and AIC statistics of three parameters (ACE, AE, and CE) and the relative contributions of genetic and environmental parameters with 95% confidence intervals for EDI sub-scales are shown in Table 2.
effects indirectly contribute to eating disorders. Klump et al. (2002a) reported that genetic factors are more likely to affect the relationships between personality and eating attitudes and behavior than environmental factors. Thus, eating disorders would have a relationship with physical or psychological traits, which have been reported to be heritable in previous studies.

Our results also showed the importance of shared environmental influences on maturity fears, ineffectiveness, interoceptive awareness, and interpersonal distrust. For instance, if an intervention works on shared environmental factors successfully, it might be possible to change eating attitudes and behaviors regarding maturity fears, ineffectiveness, interoceptive awareness, and interpersonal distrust.

Some methodological limitations of our study should be noted. Firstly, we were limited to using EDI subscales to measure the genetic and environmental influences on psychological traits of eating disorders. Therefore, we need to examine the relationship between psychological traits of eating disorders and these traits relevant to a measure of disordered eating. Secondly, it would be necessary to investigate the contribution of both genetic and environmental factors on eating disorders using other non-Western populations. Thirdly, this study included a small number of subjects. Despite these limitations, our study should be useful to generalize the results obtained from twin samples using Western populations. In conclusion, our results indicated the importance of both genetic and shared environmental influences on psychological traits of eating disorders. In future, our twin studies of psychological traits of eating disorders will focus on genetic and shared environmental contributions to chronological change using longitudinal data.

References


