Genetic and Environmental Etiology of Effortful Control

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We examined whether effortful control (EC), a temperament proposed by Rothbart and Bates (1998), has genetically coherent structure. A self-report measure of EC was administered to 450 Japanese twins (151 males and 299 females, ages 17 to 32 years) including 152 monozygotic and 73 dizygotic pairs. Univariate genetic analysis revealed that AE model fit best for the total EC as well as its subscales. The heritability estimate for total EC was 49%, and the estimates for subscales ranged between 32% and 45%. Multivariate genetic analysis revealed that the subscales of EC were genetically correlated to a high degree and environmentally correlated to a moderate degree. These results suggest that EC has substantial genetic basis and genetically coherent structure, supporting the validity of the construct. The implications to molecular genetic study and study of psychopathology were discussed.

Since Ebstein et al. (1996) and Benjamin et al. (1996) reported the association between human personality and a genetic polymorphism, many researchers have been trying to investigate the molecular genetic basis of personality. Ebstein et al. (1996) and Benjamin et al. (1996) observed that individual differences in novelty seeking (Cloninger et al., 1993) were associated with a polymorphism of dopamine receptor D4 (DRD4). Further, Lesch et al. (1996) reported that harm avoidance (Cloninger et al., 1993) was associated with a polymorphism of the serotonin transporter long promoter region (5-HTTLPR). Increasing attempts to replicate the relationships yielded inconsistent results: some studies were successful (Murakami et al., 1999; Ono et al., 1997; Strobel et al., 1999; Tomitaka et al., 1999), whereas others were not (Gelernter et al., 1997; Kumakiri et al., 1999; Mitsuyasu et al., 2001). Finally, recent meta-analysis concluded that such associations were negligible (Kluger et al., 2002; Munafo et al., 2003; Schinka et al., 2002).

Several reasons contribute to the difficulty in obtaining the molecular genetic basis of personality. The first reason is small effect size of a single polymorphism. Even in studies that revealed a significant association, variance of a single polymorphism is typically 2% to 3%. This is because many polymorphisms exert influence on complex psychological traits like personality. Therefore, a typical sample size of 100 to 200 participants is sometimes not sufficient to detect such a small effect size. The second reason is sampling bias. For example, many studies use a psychiatric population as a sample, but having psychopathology may distort the subjects' self-report on personality, thereby obscuring the association between genes and personality. In addition, some associations between genes and personality may be specific to a certain age group, gender and culture. The third reason is the heterogeneity of the personality scale used. For example, anxious personality may be associated with the 5-HTTLPR polymorphism when measured with the Neuroticism scale of the Revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992) but not when measured with the Harm Avoidance scale of the Temperament and Character Inventory (TCI; Cloninger et al., 1993). In fact, this conclusion was reached in a recent meta-analysis (Schinka et al., 2004; Sen et al., 2004). The reason why some scales are more related with certain polymorphisms may be due to (1) slight difference in the content they measure; or (2) low reliability, particularly low internal consistency. Given the high reliability of scales included in standard personality questionnaires such as NEO-PI-R and TCI, it is more plausible to consider that differences in the content, rather than reliability, are responsible for the inconsistent pattern of results.

However, the problem for association studies is that reliability is computed by phenotypic correlations among items or subscales, and not by the

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genetic correlations among them. When subscales defining the same personality traits are phenotypically correlated with each other, the correlation may be due to genetic influence, environmental influence, or both. Thus, items or subscales defining the same trait may not share their etiology: some correlations among them may be due to genetic influences, whereas other correlations may be due to environmental influences. In fact, Ando et al. (2004) showed that a subscale of Novelty Seeking was genetically correlated to a greater extent with subscales of Harm Avoidance than with the other Novelty Seeking subscales. Although no one could predict the results beforehand, this genetic heterogeneity may be one of the reasons for the many inconsistent results of molecular genetic studies that examined the relationships between the DRD4 polymorphism and novelty seeking (Munafo et al., 2003). As such, computing genetic correlations and excluding genetically unrelated subscales or items makes genetically crisp categories (Faraone et al., 1999) and helps the association study by increasing the statistical power. Considering the fact that Cloninger's model is one of most accepted biological models of temperament, any temperamental traits that are theorized to have a specific genetic basis need to be empirically tested for genetic consistency before a molecular genetic study is conducted.

In this paper, we applied this approach to effortful control (EC), a temperament proposed by Rothbart et al. (2000). EC is a unique concept since it captures a self-regulative process that is rarely modeled by other personality theorists. EC is defined as 'the ability to inhibit a dominant response to perform a subdominant response' (Rothbart & Bates, 1998, p. 137) or the 'efficiency of executive attention, including the ability to inhibit a dominant response and/or to activate a subdominant response, to plan, and to detect errors' (Rothbart, personal communication, January 26, 2002, cited in Eisenberg et al., 2004). EC can be measured by questionnaires for various age groups ranging from infancy (3 to 12 months old; Rothbart, 1981), preschool and early school years (3 to 7 years old; Rothbart et al., 2001), early adolescence (9 to 15 years old; Ellis et al., 2004) to adulthood (Rothbart et al., 2000).

The reason why the genetic coherency of EC should be empirically tested is its importance in explaining psychopathology. As EC is in essence a measure of executive functioning, and as many experimental studies have shown that the lack of executive attention is found for various forms of psychopathology (Dobson & Dozois, 2004; Gotlib & Cane, 1987; Homack & Riccio, 2004; Mathews & Macleod, 1985; Sharma et al., 2001; Smith & Waterman, 2003), it is predicted that low EC should also be associated with them. In fact, empirical studies have shown that low EC is associated with both externalizing problems and internalizing problems (Eisenberg et al., 2001; Lemery et al., 2002; Oldehinkel et al., 2004). These findings suggest that low EC may be a common diathesis of both types of problems and explain high comorbidity between them. However, it could be also possible that EC consists of genetically heterogeneous subscales, and this heterogeneity enabled the scale to be associated with both externalizing and internalizing problems. In fact, a subscale of EC assessing control of inhibition tends to be associated more with externalizing problems, whereas another subscale assessing control of attention tends to be associated more with internalizing problems (Eisenberg et al., 2001). These two possibilities have very different implications for the field. If EC was shown to be genetically homogeneous, then the molecular genetic basis of EC should be rigorously examined as it can find the genetic risk factor common to both types of problems. Alternatively, if EC was shown to be genetically heterogeneous, then the reason for comorbidity between externalizing and internalizing problems should be explored elsewhere.

Thus, the current study was purported to examine whether EC is influenced by a homogeneous set of genes using Japanese adolescent and adult twin samples. This was done by first conducting univariate genetic analyses to show genetic influences on EC and its subscales, and then computing genetic and environmental correlations between the subscales.

Method

Participants

The questionnaire booklets were mailed to approximately 600 pairs of twins. All participants were volunteers in the Keio Twin Project (Ando & Ono, 1998), recruited via invitations sent to a population-based twin residential list of Tokyo and its neighboring cities. All subjects received written explanations of the purpose of the study, the research items, protection of their privacy, and their right to cancel their participation at any time if they wished. Subjects completed an informed consent agreement document. Subjects under 20 years of age were also required to obtain their parents' written consent. The final sample consisted of 225 pairs of twins, including 152 pairs of monozygotic (MZ) twins (104 female pairs and 48 male pairs) and 73 pairs of dizygotic (DZ) twins (34 female pairs, 16 male pairs and 23 opposite-sex pairs). The age range of the sample was 17 to 32 years (mean age = 24.15, *SD* = 4.28). Zygosity was determined using the questionnaire developed by Ooki et al. (1990). For a number of pairs, a clear zygosity diagnosis could not be made and their zygosity was determined on the basis of two gene polymorphisms (DRD4 and 5-HTTLPR) and genetic fingerprinting data. The accuracy of zygosity diagnosis is estimated to be between 91% and 95% in the present sample.

Instruments

The instrument used in the present study is the Japanese version of the Effortful Control scale

(Yamagata et al., in press). The EC scale for adults consists of three subscales: Activation Control (ACC), Attentional Control (ATC), and Inhibitory Control (IC). ACC measures the capacity to perform an action when there is a strong tendency to avoid it (e.g., 'I can make myself work on a difficult task even when I don't feel like trying'). ATC measures the capacity to focus attention as well as shift attention when desired (e.g., 'When interrupted or distracted, I usually can easily shift my attention back to whatever I was doing before'). Inhibitory Control measures capacity to suppress inappropriate approach behavior (e.g., 'When I decide to quit a habitual behavioral pattern that I believe to be undesirable, I am usually successful').

The Japanese version was developed through a back-translation procedure of the 35 original items of the Effortful Control scale included in the Adult Temperament Questionnaire (Rothbart et al., 2000). The Japanese version was reported to have good internal consistency (Cronbach's alpha = .90; for subscales, .74 to .84), test–retest reliability (r = .88; for subscales, r = .79 to .89; 3 weeks), and validity (positively correlate with the performance of the Stroop color-word interference task). Participants were asked to fill out the questionnaire with the instruction not to discuss or show their answers to their twin sibling.

Statistical Analysis

In order to assess the genetic and environmental contribution to phenotypic variations of EC and its subtraits, univariate genetic analyses, as described in Neale and Cardon (1992), were conducted with the computer program Mx (Neale et al., 1999). Univariate genetic analysis decomposes the similarities (covariances) of MZ and DZ twin pairs into estimates of additive genetic (A), nonadditive genetic (D), shared environmental (C) and nonshared environmental (E) influences. The effect of additive genetic factors (A) is assumed to be the sum of multiple genes (polygene) whose effects are small and additive to form a quantitative phenotype. The effect of nonadditive genetic factors (D) is assumed to be an interactive (nonadditive) contribution of alleles within a single locus (dominance). Shared environment (C) is the effect that makes family members alike not from heredity but from the common environment shared by all family members. Nonshared environment (E) is the effect that makes family members different even if they live together, such as physical illness and differential parental treatment. It also includes measurement errors.

In order to reveal which factors significantly contribute to phenotypic variances, four models were systematically compared in terms of goodness-of-fit statistics. These are the ACE model in which phenotypic covariances are explained by A, C and E; the ADE model explained by A, D and E; the AE model explained by just A and E; and the CE model explained by just C and E. Akaike's Information Criteria (AIC) was computed and used to determine the best model from these four. The AIC reflects a model's goodness of fit as well as its parsimony, and the model that results in the smallest AIC is regarded as the best. Parameter estimates for A, D, C and E are squared to compute the familiar proportions of the variance symbolized as h^2, d^2, c^2 and e^2.

Table 1

The Mean, SD, Range, and Intraclass Correlations of EC and Its Subscales

					Intraclass correlation	
	M	*SD*	Min.	Max.	MZ	DZ
EC	93.8	13.9	51	130	.45	.21
ACC	33.3	5.9	16	48	.38	.17
ATC	29.5	6.0	12	47	.42	.20
IC	31.0	4.9	14	44	.30	.12

Note: MZ = monozygotic twins; DZ = dizygotic twins; EC = Effortful Control; ACC = Activation Control, ATC = Attentional Control, IC = Inhibitory Control.

In order to compute the genetic and environmental correlations among EC subscales, multivariate genetic analyses were conducted. Multivariate genetic analysis is a model-fitting method to reveal genetic and environmental sources of phenotypic correlations. We first subjected the MZ and DZ within-pair covariances to a Cholesky decomposition using the Mx program as described in Neale and Cardon (1992). Specifically, we fit the AE Cholesky model to the twin covariances to estimate the additive genetic and nonshared environmental covariance matrices. We then converted the parameter estimates to the additive genetic (r_G) and nonshared environmental (r_E) correlations. For example, r_G between variables i and j was calculated from the genetic covariance between i and j (a_{ij}) and the genetic variance of i (a_{ii}) and j (a_{jj}) as such:

$$rG_{ij} = \frac{a_{ij}}{\sqrt{a_{ij} \times a_{jj}}}$$

The genetic and environmental correlations can be interpreted in the same way as any correlation coefficient: they vary from –1.0 to +1.0 to reflect the degree to which two variables are influenced by the same genetic or environmental factors.

Results

Descriptive Statistics

The mean, *SD*, range, and intraclass correlations for both MZ and DZ twins for EC and each of its subscales are shown in Table 1. For total EC as well as its subscales, intraclass correlations for MZ twins were higher than those of DZ twins, suggesting the existence of additive genetic influences.

Univariate Analysis

Table 2 shows the results of univariate genetic analyses of EC and its subscales. In terms of AIC, the AE model fit best for EC as well as its subscales. For the best-fitting AE model, additive genetic effects (h^2) explained 47% of phenotypic variance, whereas non-

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Table 2

Results of Univariate Analyses

	model	χ^2	*df*	*p*	AIC	h^2	d^2	c^2	e^2
EC	CE	16.91	4	.00	8.91				
	ACE	8.93	3	.03	2.93				
	AE	8.93	4	.06	0.93	.49	—	—	.51
	ADE	8.42	3	.04	2.42				
ACC	CE	6.81	4	.15	–1.19				
	ACE	2.95	3	.40	–3.05				
	AE	2.95	4	.57	–5.05	.39	—	—	.61
	ADE	2.79	3	.43	–3.21				
ATC	CE	12.59	4	.01	4.59				
	ACE	6.65	3	.08	0.65				
	AE	6.65	4	.16	–1.36	.45	—	—	.55
	ADE	6.36	3	.10	0.36				
IC	CE	10.47	4	.03	2.47				
	ACE	7.18	3	.07	1.18				
	AE	7.18	4	.13	–0.82	.32	—	—	.68
	ADE	6.71	3	.08	0.71				

shared environmental effects (e^2) explained 53% of the variance. With regard to the subscales, heritability estimates ranged from .31 (Inhibitory Control) to .44 (Attentional Control).

Multivariate Analysis

Table 3 shows the genetic and environmental correlations among EC subscales. Correlations among the subscales were all positive in both the genetic and the environmental matrices. Genetic correlations were especially strong, with r_G ranging from .64 for between Activation Control and Inhibitory Control, to .93 for between Attentional Control and Inhibitory Control.

Discussion

This study examined the genetic and environmental etiology of EC using a Japanese adolescent and adult sample. Results of univariate analysis confirmed that EC as well as its subscales had a genetic basis. It is consistent with the finding of Goldsmith et al. (1997) that EC is heritable in childhood (h^2 = 58%). However, it is not surprising given the well-known fact that in adulthood, additive genetic and nonshared environmental effects account for variability in most personality traits (Bouchard & Loehlin, 2001).

The main focus of this study was rather on the genetic relationship between subscales of EC. As Ando et al. (2004) have recently shown in the case of Novelty Seeking, many temperaments were theorized for their genetic basis, but were in need of empirical examination for their genetic coherency. This was also the case for EC, but the multivariate analysis results in this study showed that subscales of EC were genetically correlated to a substantial degree. This suggests that individual differences in control with regard to activating and inhibiting behavior and attention are influenced by the same set of genes and form 'genetically crisp categories' (Faraone et al., 1999). Thus the EC scale can be readily used for the search of its molecular genetic basis. It also suggests that low EC is associated with both externalizing and internalizing problems because low EC can work as a common diathesis for the two types of problems, not because the scale contains genetic noise. Thus the molecular genetic study of EC is fruitful because it may reveal common genetic risk factors for internalizing and externalizing problems and contribute to their prevention.

Initially, the polymorphism of dopamine receptors and that of monoamine oxidase A (MAOA) may be good candidates. Derryberry and Rothbart (1997) and Rueda et al. (2004) proposed that individual differences in EC were founded on the activities of the anterior attentional system consisting of the anterior cingulate gyrus and the lateral prefrontal cortex that are modulated by dopamine. In fact, several neuroimaging studies revealed that tasks that require control of attention activate the anterior attentional

Table 3

Genetic and Environmental Correlations Among EC Subscales

	ACC	ATC	IC
ACC	—	.49	.34
ATC	.71	—	.31
IC	.64	.93	—

Note: Below diagonal: genetic correlations; above diagonal: environmental correlations.

network (Fan, Flombaum et al., 2003; Leung et al., 2000; Rubia et al., 2001), and that the amount of neural activation in anterior cingulate while the subjects performed the task was heritable (Fan et al., 2001) and associated with polymorphisms of the dopamine receptor gene and the MAOA gene (Fan, Fossella et al., 2003).

Testing genotype and environment (G × E) interaction in the development of EC is another direction for future research. Recently, Caspi et al. (2002) showed that aggressive behavior and conduct problems, which are closely related to low EC (Eisenberg et al., 2000; Ellis et al., 2004) were explained by the interaction between a polymorphism of the MAOA gene and parental maltreatment. It could be possible that this interaction accounts for individual differences in EC, and EC mediates the relationship between the G × E interaction and aggressive behavior.

Finally, some methodological limitations should be noted. First, it is unclear whether the present results can be applied to cultures outside Japan. Rothbart et al. (2001) reported that in children, the structure of EC differed in China, Japan and the United States. Thus, in cultures other than Japan, the etiology of EC in adulthood may be different. Further, the sample size of the present study was not sufficient to examine gender-specific effects. Olson et al. (1990) reported that the relationship between behavioral measures of EC and parenting differs depending on gender. Hence, it is necessary to examine gender-specific effects using a larger sample.

In conclusion, the results of the present study indicate that EC is genetically influenced to a substantial degree and has a genetically coherent structure. These results support the validity of the construct from a genetic point of view and encourage the search for a molecular genetic basis of temperament as a common diathesis for both internalizing and externalizing problems.

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