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Interaction Between Experience Seeking and Genetic and Environmental Influences on General Cognitive Ability

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Although it is well established that experience seeking behavior (ES) is positively related to cognitive functioning, the mechanisms underlying this association are not clearly understood. In a large sample of adult twins and siblings (N = 864, age range 23–75), we studied the causes of covariation between ES and general cognitive ability and we studied whether ES moderates the genetic and environmental causes of variation in general cognitive ability. Results demonstrate a phenotypic correlation of .17 ($p < .001$) between general cognitive ability and ES, with a common genetic and common environmental background. Moreover, the extent to which genetic and environmental factors are shared between general cognitive ability and ES is increased in individuals with either lower or higher levels of ES. In addition, the extent to which genetic and environmental factors influence individual differences in general cognitive ability in adults partly depended on ES. Standardized influences of additive genetic factors on general cognitive ability ranged from 13% to 99%, with lower estimates in higher levels of ES, while standardized estimates of environmental factors ranged from almost 1% to 87%, with higher estimates in higher levels of ES. Hence, ES and cognitive ability are not only associated through common genetic and environmental factors, but also via moderating effects of genetic and environmental influences on cognitive ability by ES. These findings have implications for future studies on the association between ES and general cognitive ability, and for future research on the genetics of cognitive ability.

■ Keywords: twin study, IQ, GxE interaction, heritability, moderation, correlation, personality

Considerable phenotypic correlations (~.30) are consistently reported between the personality trait 'openness to experience' and general cognitive ability (Ackerman & Heggestad, 1997; Higgins, Peterson, Pihl, & Lee, 2007; Moutafi, Furnham, & Crump, 2003; Phillips, Abraham, & Bond, 2003). In line with this, higher levels of experience seeking behavior (ES), a trait related to openness to experience (Fitzgerald, 1966; Zuckerman, Bone, Neary, R, Mangelsdorff, D, & Brustman, 1972), coincide with better focused attention skills (Martin, 1985), increased scientific interests (Kish & Leahy, 1970), better perceptual, arithmetic, and spatial ability (Kish & Busse, 1968; Kish & Leahy, 1970), and higher levels of general cognitive ability (Fagan, 1984; Kish & Leahy, 1970; Zuckerman, 1994). Individuals with high levels of ES are curious, open to new experiences and change, and receptive to new ideas and views, which are all qualities that are related to general cognitive ability (Myers & McCaulley, 1985). Moutafi et al. (2003) suggested a reciprocal relation between ES and general cognitive ability, such that individuals with lower levels of general cognitive ability may become less curious, and less appreciative of, or receptive to, unfamiliar experi-

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ences due to their lower ability to handle novel information. Restricted ability to benefit from novelty in turn makes exposure to new information and experiences less rewarding. On the other hand, curiosity and openness to experience may evolve in individuals with higher levels of general cognitive ability exactly because they profit from these abilities and experience them as stimulating and rewarding. In addition, experience seekers may create for themselves an enriched environment that stimulates cognitive development (Raine, Reynolds, Venables, & Mednick, 2002).

To date, the exact nature of the association between general cognitive ability and ES is largely unknown. The association may be reciprocal, as suggested by Moutafi et al. (2003), but may also be driven by a third factor. For example, as both ES and general cognitive ability are under genetic control (h^2 for ES in adults ranges from 50% to 60% [Koopmans, Boomsma, Heath, & van Doornen, 1995; Wainwright, Wright, Luciano, Geffen, & Martin, 2008]; h^2 for general cognitive ability in adults ranges from 75% to 85% [Bouchard, Jr & McGue, 1981; Plomin, 1999]), genes might mediate the relation between ES and general cognitive ability. Substantial genetic covariation has indeed been reported between openness to experience and general cognitive ability in one study in young adults by Wainwright and colleagues (2008), supporting the possibility of genetic covariation between ES and general cognitive ability in adults.

Wainwright et al. assumed that the association between general cognitive ability and openness to experience is homogeneous across different levels of openness to experience. However, increasing evidence suggests that variation in general cognitive ability is not homogenous across the whole range of cognitive abilities but depends on other traits or environmental factors (Haworth et al., 2010; van der Sluis et al., 2008; Vinkhuyzen, van der Sluis, & Posthuma, 2011). It is therefore conceivable that covariation between general cognitive ability and ES also fluctuates as a function of ES (and/or general cognitive ability).

If the relative contributions of genetic and environmental influences on general cognitive ability differ as a function of ES (i.e., gene–ES interaction), point estimates of these influences merely reflect the average heritability and environmentability of general cognitive ability across the whole range of ES levels. We refer to gene–ES interaction as 'gene–trait interaction' (GTI) to distinguish this from the term gene–environment interaction, in which the moderator is assumed not to be influenced by genetic factors (i.e., is of environmental nature).

Considering GTI may aid our understanding of the role of genetic and environmental influences on individual differences in general cognitive ability, and in our understanding of the underlying mechanisms of the phenotypic correlation between general cognitive ability and ES.

We set up a twin–sibling study (N = 864 adults) to investigate (i) whether or not the covariance between ES and general cognitive ability is partly genetic in nature, (ii) whether or not the relative contribution of genetic and environmental influences to this covariance varies as a function of ES, and (iii) whether or not the relative contribution of genetic and environmental influences to general cognitive ability depends on ES.

Methods

Sample

Data were available for 864 twins and siblings (55.8% women, 288 complete twin pairs, 23 incomplete twin pairs, 265 siblings) from 317 families that were registered at the Netherlands Twin Registry (Boomsma et al., 2006). Mean age of the participants was 46.61 years (SD = 12.40, range 23–75) at the time they completed the Life Experience List (LEL, see *Measures*) (Vinkhuyzen, van der Sluis, de Geus, Boomsma, & Posthuma, 2010). Zygosity of same-sex twins was determined using DNA polymorphisms (127 pairs, 88.2%) or, if information on DNA markers was not available, using questions about physical similarity and confusion of the twins by family members and strangers. Agreement between zygosity based on DNA and zygosity based on survey was 97% (Willemsen, Posthuma, & Boomsma, 2005). The sample was previously shown to be representative of the general Dutch population with regard to educational attainment (Posthuma, de Geus, & Boomsma, 2001). The study was performed with understanding and written consent of each participant, and was approved by the Central Committee on Research Involving Human Subjects of the VU/VUmc Amsterdam, The Netherlands.

Measures

General cognitive ability. General cognitive ability was operationalized as Full Scale Intelligence Quotient (FSIQ) and assessed with the Dutch version of the Wechsler Adult Intelligence Scale–third edition (WAIS-IIIR) (Wechsler, 1997). FSIQ was based on seven subtests of the WAIS-IIIR (N = 74; Block design, Letter-number sequencing, Information, Matrix reasoning, Arithmetic, Vocabulary, and Digit symbol-coding) or 11 subtests of the WAIS-IIIR (N = 785; the above seven subtests plus Similarities, Picture completion, Digit symbol-coding, and Digit-symbol pairing). The correlation between FSIQ assessed with 11 subtests, and FSIQ assessed with seven subtests, was very high, $r(783) = .97$, $p < .001$. Test-retest reliability, studied in 59 participants who completed the WAIS-IIIR twice with an interval of ~10 years, was also high, $r(57) = .85$, $p < .001$.

Experience seeking. The Experience Seeking (ES) scale is one of the four subscales of the Dutch translation of the Sensation Seeking Scale (Feij & van Zuilen, 1984; Zuckerman, 1971; Zuckerman, 1979; Zuckerman et al.,

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1964) and was incorporated in the Life Experiences List (LEL) (Vinkhuyzen et al., 2010a). The ES scale has been described as the 'hippie factor' (Zuckerman, 1971) and refers to desired experiences through wanderlust, exhibitionism, use of marijuana and hallucinatory drugs, association with nonconformist friends, and liking of modern and arousing arts and music. The ES scale consists of 14 multiple choice items measured on a 5-point Likert scale with answer categories ranging from 1 (*definitely disagree*) to 5 (*definitely agree*). All items were scored such that high item scores correspond to high levels of experience seeking behavior. If three or more item responses were missing, overall ES scores were considered unreliable, and the ES data were excluded from analysis. Sum scores calculated across all available items, and divided by the number of valid items, were used as unit of analysis in this study, such that the minimum score was one, and the maximum score was five.

Test-retest reliability of the ES scale, studied in an independent sample of 62 participants (31 parent–offspring pairs, 75.4% women; age range 17–71 years; $M = 39.95$, $SD = 16.19$) who completed the LEL twice within a period of two months, was high (.87, $p < .001$).

Statistical analyses

To start with, the phenotypic correlation between general cognitive ability and ES was calculated in Mplus (Muthen & Muthen, 2005), using option 'complex', to correct for familial relatedness between the participants.

Subsequently, monozygotic (MZ) twin, dizygotic (DZ) twin, and sibling correlations for general cognitive ability and ES were estimated within an unrestrained model. Differences between zygosity groups in means and variances, as well as differences between DZ twin correlations and regular sibling correlations of cognitive ability and ES, were tested using likelihood-ratio-tests.

To detect moderation effects in the presence of possible shared genetic effects or shared environmental factors, a bivariate interaction model was fitted to the data (Purcell, 2002). Within the bivariate interaction model, variances of cognitive ability and ES, as well as the covariance between cognitive ability and ES, were modeled as a function of genetic and environmental effects. Genetic factors 'A' and 'D' and environmental factors 'C' and 'E' were considered. 'A' represents additive genetic effects of alleles summed over all genetic loci (*additive genetic effects*). 'D' represents nonadditive genetic effects within loci (*genetic dominance*). 'C' represents shared environmental influences that render offspring of the same family more alike (*shared environmental factors*). 'E' represents all environmental influences that result in differences between members of a family, including measurement error (*nonshared environmental factors*). To model GTI, variance components of cognitive ability were allowed to vary as a function of ES. Note that C and D are confounded when only data from twins and siblings are available because C and D have opposite effects on the difference between MZ twin and DZ twin correlations. When DZ twin correlations are less than half the MZ twin correlations, as was the case for both ES and cognitive ability in the present data, dominance genetic effects are expected rather than common environmental effects. In that case, a model including A, D, and E is deemed most appropriate.

Within this bivariate interaction model, the moderator ES features twice: as a dependent variable and as an actual moderator. Moreover, moderating effects of ES can be modeled on two types of variance components: the variance components unique to cognitive ability and the variance components shared between cognitive ability and ES (Purcell, 2002).

Given that the twin correlations for cognitive ability and ES were suggestive of genetic dominance, the variance of ES is calculated as:

$$\mathrm{Var(ES)} = a^2 + d^2 + e^2,$$

where a^2 denotes additive genetic variance, d^2 denotes genetic dominance variance, and e^2 denotes nonshared environmental variance; whereas the variance of general cognitive ability is calculated as follows:

$$\mathrm{Var(general\ cognitive\ ability)} = (a_c + a_c\text{'}*\mathrm{Mod}_{tw1} + a_c\text{''}*\mathrm{Mod}^2_{tw1})^2 + (a_u + a_u\text{'}*\mathrm{Mod}_{tw1} + a_u\text{''}*\mathrm{Mod}^2_{tw1})^2 + (d_c + d_c\text{'}*\mathrm{Mod}_{tw1} + d_c\text{''}*\mathrm{Mod}^2_{tw1})^2 + (d_u + d_u\text{'}*\mathrm{Mod}_{tw1} + d_u\text{''}*\mathrm{Mod}^2_{tw1})^2 + (e_c + e_c\text{'}*\mathrm{Mod}_{tw1} + e_c\text{''}*\mathrm{Mod}^2_{tw1})^2 + (e_u + e_u\text{'}*\mathrm{Mod}_{tw1} + ea_u\text{''}*\mathrm{Mod}^2_{tw1})^2,$$

where Mod_{tw1} denotes the value of Twin 1 on the moderator (i.e., the ES score of Twin 1, a_c, d_c, and e_c denote unmoderated regression coefficients of additive genetic, genetic dominance, and nonshared environmental factors, respectively, that are shared between ES and cognitive ability); a_u, d_u, and e_u denote unmoderated regression coefficients of additive genetic, genetic dominance, and nonshared environmental factors that are unique to cognitive ability; a_c', d_c', and e_c' denote linear moderation coefficients (i.e., the regression coefficients of A, D, and E that fluctuate as a function of ES. Similarly, a_c", d_c", and e_c" denote nonlinear moderation coefficients for A, D, and E, respectively. Coefficients correspond to path-coefficients in Figure 1.

To test whether unmoderated variance components (specific to trait and moderator as well as shared between trait and moderator) were significant within the full sample (i.e., average levels of ES), a bivariate model without moderation was specified (models 1–9 in Table 3). Subsequently, a reference model was specified, in which nonsignificant unmoderated variance components were fixed to zero and potential moderation coefficients were freely estimated (model 10 in Table 3), to test significance of linear and nonlinear moderation effects. Since moderation on D has indirect effects on the additive genetic variance, moderation coefficients on the additive genetic variance (i.e., a' and a' in Figure 1) should be included in

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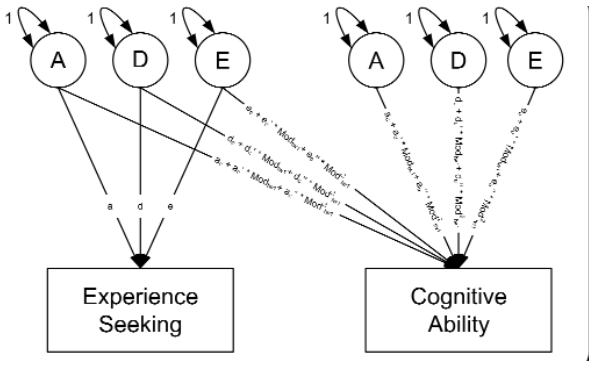

FIGURE 1

Partial bivariate model for one twin including linear and nonlinear moderation effects of the environmental moderator (experience seeking) on the variances of general cognitive ability and on the covariance between cognitive ability and the experience seeking. A = additive genetic effects; D = genetic dominance; E = nonshared environmental effects; a = unmoderated path coefficient for A; d = unmoderated path coefficient for D; e = unmoderated path coefficient for E; a_c = genetic factors shared between moderator and trait; a_u = genetic factors unique to trait; d_c = genetic dominance effects shared between moderator and trait; d_u = genetic dominance effects unique to trait; e_c = nonshared environmental factors shared between moderator and trait; e_u = nonshared environmental factors unique to trait. a_c', d_c', and e_c' denote linear moderation coefficients for A, D, and E, respectively; a_c", d_c", and e_c" denote nonlinear moderation coefficients for A, D, and E, respectively.

the model whenever moderation coefficients on the dominance component are estimated (i.e., d' and d' in Figure 1). Therefore, we first tested the significance of nonlinear and linear dominance-related moderation effects (i.e., d_c', d_u' and d_c', d_u' in Figure 1, models 11–16 in Table 3) before testing nonlinear and linear moderation on additive genetic influences (i.e., a_c', a_u' and a_c', a_u' in Figure 1, models 17–21 in Table 3) and nonshared environmental influences (e_c', e_u' and e_c', e_u' in Figure 1, models 22–25 in Table 3).

The preference of first testing significance of unmoderated variance components in the context of a bivariate model without moderation is because the purpose of the present study was to detect possible moderation effects, and power to detect moderation effects is lower when nonsignificant unmoderated variance components are estimated in the moderator model.

Significance of parameters was tested by comparing the fit of nested (increasingly more restricted) models to the fit of less restricted models. Goodness-of-fit of submodels was assessed by likelihood-ratio-tests. The difference in log-likelihoods between two models was evaluated. A significant χ^2-difference test implies that the constraints imposed on the nested models are not tenable, whereas a nonsignificant χ^2-difference test implies that the nested, more parsimonious model is to be preferred. All measures were corrected for age and sex to avoid spuriously increased similarities in MZ and same-sex DZ twin pairs (McGue & Bouchard, Jr, 1984). Analyses were carried out using the raw data option in Mx (Neale, 1994; Posthuma & Boomsma, 2005).

Results

The phenotypic correlation (corrected for familiarity) between cognitive ability and ES in the sample was .17 (p < .001). Means and variances of cognitive ability, $\chi^2(2, N = 859) = .53$, *ns* and ES $\chi^2(2, N = 549) = .10$, *ns*, could be considered equal between zygosity groups without a significant deterioration of the model fit, implying that there was no heterogeneity in these measures for MZ and DZ twins and their siblings. Table 1 includes information on means and standard deviations of general cognitive ability and ES, as well as information on missingness.

TABLE 1

Descriptive Statistics for General Cognitive Ability and Experience Seeking

	N	% Missing	Mean (SD)
General cognitive ability	859	.6% *	99.68 (14.78)
ES	549	2% **	2.61 (.60)

Notes: N = number of participants; % Missing=percentage of missingness, this is the percentage of participants that (*) participated in the present study but did not complete the IQ test or that (**) returned the LEL but did not complete the questions on experience seeking; Mean=mean score corrected for age and sex effects; SD = standard deviation.

Table 2 shows the sex and age-corrected MZ twin, DZ twin, sibling, and pooled DZ/sibling correlations and standardized variance components for cognitive ability and ES. Sibling correlations did not differ from DZ twin correlations for cognitive ability, $\chi^2(1, N = 746) = 3.72$, *ns*, and ES $\chi^2(1, N = 380) = .25$, *ns*, suggesting no special twin environment. MZ twin correlations exceeded the DZ/sibling correlations for cognitive ability, $\chi^2(1, N = 982) = 77.24$, $p < .001$, and ES $\chi^2(1, N = 463) = 13.15$, $p < .001$, suggesting the presence of genetic influences. As pooled DZ/sibling correlations for cognitive ability (.35) and ES (.28) were less than half the MZ twin correlations (cognitive ability = .82; ES = .60), presence of genetic dominance, rather than common environmental effects, was indicated. Table 2 also shows the results of the uni-

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TABLE 2

Twin and Sibling Correlations (95% confidence intervals) and Standardized Variance Components (95% confidence intervals) for General Cognitive Ability and Experience Seeking

	rMZ (95% CI)	rDZ (95% CI)	rSibling (95% CI)	rDZ/rSibling (95% CI)
General cognitive ability	.82 (.77-.86) N=136	.46 (.32-.57) N=152	.32 (.22-.42) N=594	.35 (.26-.44) N=746
ES	.60 (.50-.71) N=83	.31 (.11-.48) N=67	.26 (.13-.39) N=313	.28 (.16-.39) N=380
	a^2	d^2	e^2	
General cognitive ability	.60 (.24-.85)	.22 (.00-.58)	.18 (.14-.23)	
ES	.57 (.10-.70)	.03 (.00-.53)	.40 (.30-.53)	

Notes: rMZ=MZ twin correlation; rDZ=DZ twin correlation; rSibling=sibling correlation; rDZ/rSibling=pooled DZ and sibling correlation; Twin and sibling correlations were corrected for sex and age; N=number of pairs; 95% CI=95% confidence interval; a^2=standardized additive genetic variance; d^2=standardized dominance genetic variance; e^2=standardized non-shared environmental variance. Standardized variance components are based on full models.

variate variance decomposition of both ES and general cognitive ability. For both general cognitive ability and ES, additive genetic influences explained the major part of the variance. Genetic dominance deviations were not significantly different from zero for both ES and general cognitive ability, although the point estimate of .22 suggested moderate effects for general cognitive ability.

Correlation and Moderation

Model fitting results of a bivariate analysis, in which the variances of cognitive ability and experience seeking as well as their covariance were decomposed into A, D, and E, are presented in Table 3.

Bivariate analyses excluding interaction effects showed that genetic dominance did not significantly contribute to variation in general cognitive ability and ES, or to their covariation (models 2–5). Unmoderated additive genetic effects, however, did significantly contribute to variation and covariation of cognitive ability and ES (models 6–8). Unmoderated nonshared environmental effects did not contribute significantly to the covariance between cognitive ability and ES (model 9).

Bivariate analyses including moderation effects demonstrated that neither linear moderation nor nonlinear moderation of genetic dominance effects were significant (models 11–16), implying that dominance effects were nonsignificant across the entire range of ES. Nonlinear moderation of additive genetic factors specific to cognitive ability (model 18; $\chi^2(1, N = 864) = 8.21, p < .01$) was significant. Furthermore, analyses demonstrated significant nonlinear (model 23; $\chi^2(1, N = 864) = 5.18, p < .05$) and linear (model 23; $\chi^2(1, N = 864) = 10.02, p < .01$) moderation on nonshared environmental factors unique to cognitive ability.

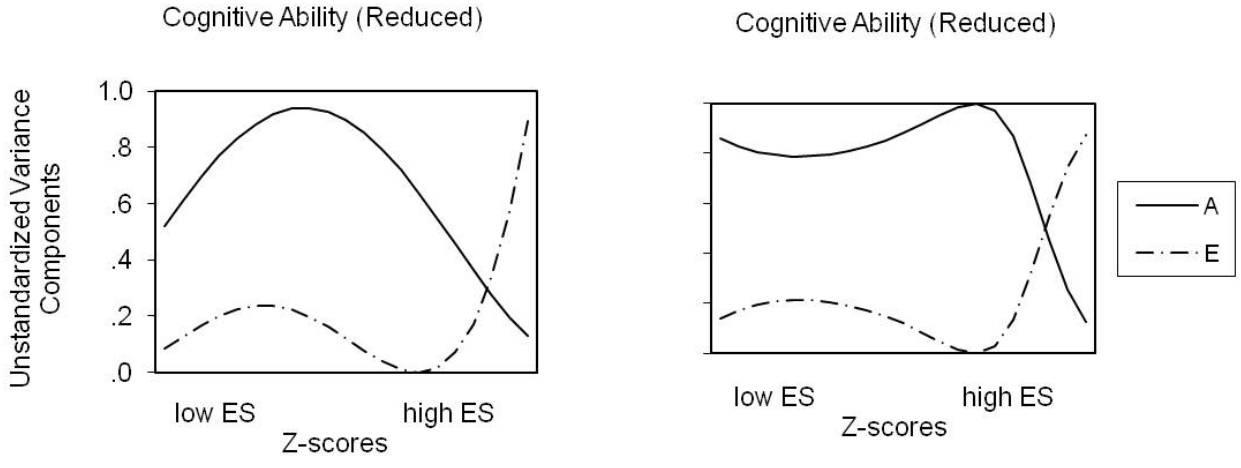

FIGURE 2

Unstandardized (left panel) and standardized (right panel) variance components of cognitive ability as a function of experience seeking. The figures are based on the most reduced models (i.e., on the models in which all nonsignificant effects were fixed at zero). Unstandardized variance components (left panel) refer to the absolute contribution of A (additive genetic effects) and E (nonshared environmental effects); standardized variance components (right panel) refer to the relative contribution to variation in cognitive ability as a function of experience seeking. ES = experience seeking. The heritability of cognitive ability decreases dramatically for individuals with ES scores higher than two standard deviations above the mean.

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TABLE 3

Model Fitting Results for Bivariate ADE Interaction Models of Cognitive Ability with Experience Seeking as Moderator Variable

	model	against	-2LL	est par	df	$\Delta\chi^2$	df	*p*
1	Full model (no moderation)		2865.268	11	1075			
2	drop d	1	2866.393	10	1076	1.125	1	.289
3	drop d_c	1	2866.121	10	1076	.853	1	.356
4	drop d_u	1	2865.467	10	1076	0.199	1	.656
5	drop d, d_c, d_u	1	2867.679	8	1078	2.411	3	.492
6	drop a	5	2924.628	7	1079	56.949	1	.000
7	drop a_c	5	2876.08	7	1079	8.401	1	.004
8	drop a_u	5	2917.836	7	1079	50.157	1	.000
9	drop e_c	5	2867.727	7	1079	.048	1	.827
10	Full model (including moderation)		2843.158	19	1067			
11	drop d_c"	10	2843.158	18	1068	.000	1	1.000
12	drop d_u"	10	2843.198	18	1068	.040	1	.841
13	drop d_c",d_u"	10	2843.198	17	1069	.040	2	.980
14	drop d_c'	13	2843.198	16	1070	.000	1	1.000
15	drop d_u'	13	2843.198	16	1070	.000	1	1.000
16	drop d_c',d_u'	13	2843.198	15	1071	.000	2	1.000
17	drop a_c"	16	2845.732	14	1072	2.534	1	.111
18	drop a_u"	16	2851.404	14	1072	8.206	1	.004
19	drop a_c'	17	2848.539	13	1073	2.807	1	.094
20	drop a_u'	17	2847.143	13	1073	1.411	1	.235
21	drop a_c' a_u'	17	2848.877	12	1074	3.145	2	.208
22	drop e_c"	21	2852.229	11	1075	3.352	1	.067
23	drop e_u"	21	2854.058	11	1075	5.181	1	.023
24	drop e_c' (= final model)	22	2852.58	10	1076	.351	1	.554
25	drop e_u'	22	2862.245	10	1076	10.016	1	.002

	low ES	intermediate ES	high ES
rA	.30	.24	.30
rE	.00	.00	.00
%A	1	1	1
%E	.00	.00	.00

Notes: -2LL=minus 2 log likelihood; par=estimated parameters; χ^2=Chi square (difference in -2LL); *p*=*p*-value; a_c, d_c, and e_c = unmoderated parts of additive genetic, genetic dominance, and non-shared environmental factors, respectively, that are shared between experience seeking and cognitive ability; a_u, d_u, and e_u = unmoderated parts of additive genetic, genetic dominance, and non-shared environmental factors that are unique to cognitive ability; a_c', d_c', and e_c' = linear moderation coefficients, i.e., the parts of A, D and E that fluctuate as a function of experience seeking, respectively; a_c", d_c", and e_c" = non-linear moderation coefficients for A, D, and E that fluctuate as a function of experience seeking, respectively; r_A=genetic correlation; r_E=non-shared environmental correlation; $\%_A$=percentage of phenotypic correlation explained by genetic factors for Low, Intermediate, and High levels of experience seeking; $\%_E$=percentage of phenotypic correlation explained by non-shared environmental factors for Low, Intermediate, and High levels of experience seeking; significant (moderation) effects are printed in bold font.

Within the preferred model (model 24), standardized estimates of additive genetic factors for cognitive ability varied from 13% to 99%, with smaller additive genetic effects observed for higher levels of ES. Standardized estimates of nonshared environmental factors varied concordantly from 87% to almost 1%. Figure 1 shows standardized and unstandardized variance components of cognitive ability as a function of ES.

Table 3 shows genetic and environmental correlations between general cognitive ability and ES, as well as the extent to which the phenotypic correlation between cognitive ability and ES is due to genetic and environmental factors, as a function of the level of ES. The coefficients are therefore reported for low (–2 *SD*), intermediate (± 0 *SD*), and high (+2 *SD*) levels of (standardized) ES. The genetic correlation (r_G) between cognitive ability and ES was somewhat increased in individuals with low and higher levels of ES.

As the contribution of genetic factors to the phenotypic correlation between ES and general cognitive ability was dependent on the level of the moderator, the phenotypic correlation was also dependent of the moderator. Although the mean phenotypic correlation was .17, the phenotypic correlation was relatively increased in individuals with low and high ($r \sim .22$; +2 *SD*) levels of ES.

Within the present sample, we were not able to model both genetic dominance (D) and shared environmental factors (C). Instead, an interaction model with additive

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TABLE 4

Model Fitting Results for Bivariate ACE Interaction Models of Cognitive Ability with Experience Seeking Behavior as Moderator Variable

	model	against	-2LL	est par	df	$\Delta\chi^2$	df	*p*
1	Full model (no moderation)		2867.679	11	1075			
2	drop c	1	2867.679	10	1076	.000	1	1.000
3	drop c_c	1	2867.679	10	1076	.000	1	1.000
4	drop c_u	1	2867.679	10	1076	0.000	1	1.000
5	drop c, c_c, c_u	1	2867.679	8	1078	0	3	1.000
6	drop a	5	2924.628	7	1079	56.949	1	.000
7	drop a_c	5	2876.08	7	1079	8.401	1	.004
8	drop a_u	5	2917.836	7	1079	50.157	1	.000
9	drop e_c	5	2867.727	7	1079	.048	1	.827
10	Full model (including moderation)		2843.198	19	1067			
11	drop c_c"	10	2843.198	18	1068	.000	1	1.000
12	drop c_u"	10	2843.198	18	1068	.000	1	1.000
13	drop c_c",c_u"	10	2843.198	17	1069	.000	2	1.000
14	drop c_c'	13	2843.198	16	1070	.000	1	1.000
15	drop c_u'	13	2843.198	16	1070	.000	1	1.000
16	drop c_c',c_u'	13	2843.198	15	1071	.000	2	1.000
17	drop a_c"	16	2845.732	14	1072	2.534	1	.111
18	drop a_u"	16	2851.404	14	1072	8.206	1	.004
19	drop a_c'	17	2848.539	13	1073	2.807	1	.094
20	drop a_u'	17	2847.143	13	1073	1.411	1	.235
21	drop a_c' au'	17	2848.877	12	1074	3.145	2	.208
22	drop e_c"	21	2852.229	11	1075	3.352	1	.067
23	drop e_u"	21	2854.058	11	1075	5.181	1	.023
24	drop e_c' (= final model)	22	2852.58	10	1076	.351	1	.554
25	drop e_u'	22	2862.245	10	1076	10.016	1	.002

	low ES	intermediate ES	high ES
rA	.30	.24	.43
rE	.00	.00	.00
%A	1	1	1
%E	.00	.00	.00

Notes: -2LL=minus 2 log likelihood; par=estimated parameters; χ^2=Chi square (difference in -2LL); p=p-value; a_c, c_c, and e_c = unmoderated parts of additive genetic, shared-environmental and non-shared environmental factors, respectively, that are shared between experience seeking and cognitive ability; a_u, c_u, and e_u = unmoderated parts of additive genetic, shared-environmental and non-shared environmental factors that are unique to cognitive ability; a_c', c_c', and e_c' = linear moderation coefficients, i.e., the parts of A, C and E that fluctuate as a function of experience seeking, respectively; a_c", c_c", and e_c" = non-linear moderation coefficients for A, C, and E that fluctuate as a function of experience seeking, respectively; r_A=genetic correlation; r_E=non-shared environmental correlation; $\%_A$=percentage of phenotypic correlation explained by genetic factors for Low, Intermediate, and High levels of experience seeking; $\%_E$=percentage of phenotypic correlation explained by non-shared environmental factors for Low, Intermediate, and High levels of experience seeking; significant (moderation) effects are printed in bold font.

genetic factors, genetic dominance, and nonshared environmental factors was considered (i.e., a bivariate ADE interaction model), assuming shared environmental factors to be absent. The choice of an ADE model was based on univariate twin correlations for cognitive ability and ES that suggested absence of shared environmental factors and possible presence of genetic dominance deviation. These correlations were, however, based on the overall sample while moderation was assumed to be absent. Previous studies (Kremen et al., 2005; van der Sluis et al., 2008; Vinkhuyzen et al., 2010b), however, demonstrated that shared environmental factors for general cognitive ability can seem absent in the full sample, while they are actually substantial for specific levels of a moderator. We therefore specified a second series of interaction models in which the variances of cognitive ability and ES, as well as their covariance, were specified as a function of additive genetic factors, and shared and nonshared environmental factors (i.e., a bivariate ACE interaction model). Within this ACE interaction model, dominance genetic (interaction) effects were assumed to be absent, as is in line with the results presented in Table 3. These bivariate ACE interaction analyses (Table 4), however, showed no significant moderation of shared environmental factors.

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Discussion

In order to elucidate the association between experience seeking behavior and general cognitive ability, we decomposed their covariance into genetic and environmental effects and tested whether ES moderates the causes of variation in general cognitive ability. Within the present study, a positive, but modest, association between ES and general cognitive ability was confirmed (.17). This phenotypic association between ES and general cognitive ability, however, depended on the level of ES, with highest correlation in individuals with low and high levels of ES (.23). This was due to moderation of genetic influences, common to ES and general cognitive ability. Furthermore, ES significantly moderated additive genetic (A) and nonshared environmental (E) variance components of cognitive ability. Standardized heritability estimates were generally high (above 80%) but decreased substantially in individuals with high levels of ES (to 13%), while environmental factors increased (to 87%). Thus, individual differences in cognitive ability are, on average, best explained by a large contribution of genetic factors, while environmental factors gain in importance when ES levels are high.

Although moderation effects of ES have not been studied before, these results support previous theories on moderation of variance components of general cognitive ability that suggest that genetic and environmental factors do not simply add up, but have a more complex relation (Eaves, Last, Martin, & Jinks, 1977; Loehlin & DeFries, 1987). Our analyses suggest that the relative contribution of environmental influences on individual differences in general cognitive ability tends to increase, while the genetic contribution tends to decrease, with higher levels of ES. That is, environmental factors are more important and genetic influences are less important in explaining individual differences in general cognitive ability in those subgroups that actively seek out exposure to a wide variety of experiences. Individuals with high levels of ES are likely to seek out environments that optimize the probability to be exposed to new experiences; the observation that environmental factors gain in importance in individuals with high levels of ES is therefore expected. Although the relative influence of genetic factors decreases as a function of increasing ES, we also observed a decrease in absolute contribution of genetic factors due to moderation. The decrease of variation due to genetic factors, however, is more complicated. Our results suggest that genetic effects on general cognitive ability are conditional on environmental exposure (i.e., related to ES), but the mechanisms underlying this interaction remain as yet unknown. Future studies may focus, for example, on epigenetic effects; for example, environmental factors may cause epigenetic changes that may reduce gene expression.

The genetic overlap between ES and general cognitive ability across different levels of ES is of particular interest in the context of the role of dopamine in both ES and general cognitive ability. For example, the D4 dopamine receptor gene (D4DR) is expressed in limbic (Van Tol et al., 1991) and prefrontal (De La & Madras, 2000; Mrzljak et al., 1996; Primus et al., 1997) areas that are involved in general cognitive ability. A positive association has been demonstrated between blockade of dopamine D4 receptors and cognitive impairment. At the same time, an association between novelty seeking and dopamine transmission has been proposed (Cloninger, 1987). Ebstein et al. (1996) and Benjamin, Li, Patterson, Greenberg, Murphy, & Hamer (1996) demonstrated an association between higher levels of novelty seeking behavior and the 7 repeat allele in the D4DR gene. As individual differences in general cognitive ability and individual differences in novelty seeking behavior are related to genetic variability in dopamine transmission, future studies may investigate whether the association between ES and cognitive ability is moderated by dopamine receptor genes such as the D4DR gene.

Although the size of our sample was considerable, the limited sample size of the present study might affect the power to reliably test the moderation of the variance components of cognitive ability; therefore, these results need replication in a larger sample. A larger sample that not only includes twins and siblings but also parents and/or children of the twins and siblings will also allow simultaneous estimation of both shared environmental influences and genetic dominance. The phenotypic as well as the genetic association between cognitive ability and openness to experience have been reported before, but this is the first study to show that the association is not homogenous across different levels of openness to experience. Furthermore, these results are based on a population based sample that was representative of the Dutch population; whether the same mechanisms of moderation apply to other populations has yet to be studied.

To conclude, we demonstrated that general cognitive ability and ES are not only associated through common genetic factors, but also via moderating effects of the underlying variance components of general cognitive ability by ES. These results are valuable in understanding the underlying mechanisms of the phenotypic association between general cognitive ability and ES, as well as in understanding individual differences in general cognitive ability.

Conflict of interest

The authors declare no conflict of interest.

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