Not All Risks Are Created Equal: A Twin Study and Meta-Analyses of Risk Taking Across Seven Domains

X. T. (Xiao-Tian) Wang
Chinese Academy of Sciences, Beijing, People's Republic of China and University of South Dakota

Rui Zheng, Yan-Hua Xuan, Jie Chen, and Shu Li
Chinese Academy of Sciences, Beijing, People's Republic of China

Humans routinely deal with both traditional and novel risks. Different kinds of risks have been a driving force for both evolutionary adaptations and personal development. This study explored the genetic and environmental influences on human risk taking in different task domains. Our approach was threefold. First, we integrated several scales of domain-specific risk-taking propensity and developed a synthetic scale, including both evolutionarily typical and modern risks in the following 7 domains: cooperation/competition, safety, reproduction, natural/physical risk, moral risk, financial risk, and gambling. Second, we conducted a twin study using the scale to estimate the contributions of genes and environment to risk taking in each of these 7 domains. Third, we conducted a series of meta-analyses of extant twin studies across the 7 risk domains. The results showed that individual differences in risk-taking propensity and its consistency across domains were mainly regulated by additive genetic influences and individually unique environmental experiences. The heritability estimates from the meta-analyses ranged from 29% in financial risk taking to 55% in safety. Supporting the notion of risk-domain specificity, both the behavioral and genetic correlations among the 7 domains were generally low. Among the relatively few correlations between pairs of risk domains, our analysis revealed a common genetic factor that regulates moral, financial, and natural/physical risk taking. This is the first effort to separate genetic and environmental influences on risk taking across multiple domains in a single study and integrate the findings of extant twin studies via a series of meta-analyses conducted in different task domains.

Keywords: risk, risk-taking propensity, risk domains, twin study, meta-analysis

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Risks—whether old or new, typical or novel—are ubiquitous in human life. Similar to our ancestors, we learn to live and cope with risks in various task situations (Slovic, 2000). Different kinds of risks (natural, social, moral, financial, and recreational) have been a driving force for both evolutionary adaptations and personal development (Wang, Kruger, & Wilke, 2009). According to the notion of evolutionary domain specificity, not all risks are created equal (Cosmides & Tooby, 1992, 1996; Gigerenzer & Selten, 2002; Wang, 1996, 2002). Throughout hominid evolution, our ancestors have encountered recurring natural and social risks that demanded specific adaptations, such as safety monitoring, reproductive risk avoidance, and cooperation and competition within and between groups. At different stages during their life spans, humans also engage in tradeoffs between different types of risk taking, such as taking financial and health risks to raise offspring and thus reduce the risk of reproductive failure. Likewise, more recent technological developments and social transformations have created modern forms of risk and have resulted in new risk-taking behaviors, such as high-speed driving or incurring a debt to invest in stocks. Considering the phenotypic differences among individuals, we explore the impacts of heredity and environment on risk-taking propensity in both evolutionarily typical and modern risk domains.

Previous studies have shown that risk-taking propensity is domain specific (Blais & Weber, 2001; Horvath & Zuckerman, 1993; Zuckerman & Kuhlman, 2000), and such findings have been confirmed in multiple countries (Blais & Weber, 2006). For example, a person might invest one tenth of his annual income in a stock, but the same person would never bet a day's income on a

single casino game. Another may enjoy bungee jumping but would never take the health risk of tasting a delicious but potentially poisonous food item. A woman may postpone reproduction for the sake of financial freedom but refuses to take a relational risk to argue against her boss regarding the unfair treatment of a co-worker.

Two schemes for classifying risk domains have been adopted in the literature on domain-specific risk taking. One is based on the types of risky scenarios often encountered in modern life. Using this classification, Weber, Blais, and Betz (2002) developed and subsequently revised a domain-specific risk-taking scale across five risk domains (ethical, financial, health/safety, recreational, and social risks). Likewise, using Chinese samples, Hu and Xie (2012) derived a domain-specific scale of risk taking over four domains (ethical, recreational, health/safety, and gambling). A second scheme of classification is based on evolutionary psychology and life-history analysis. Kruger, Wang, and Wilke (2007) identified five modern versions of evolutionarily recurrent risks: between-group competition, within-group competition, mating and resource allocation, environmental risks, and fertility risks. In the present study, we integrated the aforementioned scales of risk-taking propensity to include both evolutionarily typical and modern risks. This approach allowed us to begin with a valid measuring tool of individual differences in domain-specific risk taking and then to explore differential genetic and environmental influences on risk-taking propensity in a twin study.

The extant twin studies on risk taking have generally focused on a single type of risk, with a few exceptions (e.g., Miles et al., 2001). To the best of our knowledge, there have been no integrative and comparative twin studies based on a scale of domain-specific risk taking. The results of existing twin studies on risk-taking behaviors are inconsistent and often contradictory. Within one specific risk domain, heritability estimates of risk taking differ drastically across studies. For example, the heritability estimates for gambling behavior have ranged from 72% to 0% (Beaver et al., 2010; Slutske & Richmond-Rakerd, 2014; Slutske, Zhu, Meier, & Martin, 2011). These contradictory results call for comparative twin studies with unified measurement tools as well as separate meta-analyses of risk taking in different domains.

A twin study across risk domains enables the investigation of genetic and environmental influences on individual differences in risk-taking propensity. For example, when compared with financial risk taking, which may be shaped to a large degree by the economic environment and personal experiences, physical risk taking may be regulated more by hereditary factors that shape innate preferences for adventurous activities. The results of such analyses could contribute to improving educational programs or clinical interventions through targeting the specific origins of particular risk-taking behaviors.

In addition, twin studies also help us to understand individual differences in risk taking and make better behavioral predictions. Some individuals are more consistently risk seeking or risk averse across domains than others (Soane & Chmiel, 2005). The pattern of risk taking across domains also differs between subsamples of individuals (e.g., bungee jumpers vs. gamblers; Hanoch, Johnson, & Wilke, 2006). The present research also examines how individual differences in risk propensity are affected by specific genetic and environmental factors.

Our approach was threefold. First, we developed a synthetic seven-domain scale through exploratory factor analysis (EFA) and confirmatory factor analysis (CFA), including categories of both evolutionarily typical and modern risks: cooperation/competition, safety, reproduction, natural/physical risk, moral risk, financial risk, and gambling. Second, we conducted a twin study that estimated the relative contributions of genes and environment to risk-taking propensity across the seven domains. Third, we synthesized our results with previous twin studies on risk taking by completing a series of separate meta-analyses across risk domains.

Study 1: Development of the Domain-Specific Risk-Taking Scale Across Seven Domains

Study 1 aimed to develop a valid tool for measuring individual differences in risk-taking propensity across domains in an attempt to encompass both evolutionarily typical (e.g., safety, mating, and reproduction) and modern (e.g., financial) risks. To achieve this objective, we combined several domain-specific risk-taking scales to derive items for further factor analysis and validly tests. The end product (Domain-Specific Risk-Taking Scale Across Seven Domains; DOSPERT-7) described herein is not intended to exhaust risk categories but rather to capture both modern and evolutionarily typical risks.

Method

Participants and procedure. We conducted the following three rounds of sampling: one for EFA with 237 college students (133 females, age = 22.1 ± 2.3 years), one for CFA with 351 college students (199 females, age = 20.4 ± 2.0 years), and one for an additional construct validity analysis with a general public sample of 300 respondents (155 females, age = 32.2 ± 2.7 years). All of the participants were recruited from Beijing, People's Republic of China. The participants rated each of the questionnaire items, presented in Chinese, as to their likelihood of engaging in the described risk-taking behavior on a 5-point scale (very unlikely, unlikely, not sure, likely, or very likely). The questionnaire items were presented in random order. Each participant received a souvenir worth approximately $1 (6 yuan) at the end of the study.

Measurement. We developed the synthetic domain-specific risk-taking scale by combining the following four scales: (a) the Domain-Specific Risk-Taking (DOSPERT) scale developed by Weber and his colleagues (Weber et al., 2002; Blais & Weber, 2001), (b) a revised version of DOSPERT (Blais & Weber, 2006), (c) a Chinese version of the Domain-Specific Risk-Taking Scale (DOSPERT-C) developed by Hu and Xie (2012), and (d) a scale of risk-taking propensity across evolutionary domains of risk (Kruger et al., 2007). All of the items were phrased in terms of choice problems manifested in modern time (see *Appendix*).

Results and Discussion

First, from the aforementioned four scales we derived 58 items for principal component analysis by conducting an EFA with varimax rotation. From this analysis, 20 items were deleted because of low loadings on a factor (<.40) or because an item loaded on more than one factor. Seven factors (one associated with each domain) were retained after the examination of both of the eigen-

values (which had to be >1 for retention) and the scree plot, accounting for 57.4% of the total variance (see Table 1 for factor loading scores of the remaining 38 items across the seven factors/domains).

We adopted orthogonal rotation because of our theoretical assumption that the domains were relatively independent of each other (Vogt, 1993). To test the seven-factor structure with a weaker assumption about their independence, we also performed an EFA with oblique rotation (using the oblimin method). The results showed that the factor structures of the orthogonal and oblique rotations were identical.

To confirm the factor structure found through the EFA, we performed a CFA to test this seven-factor structure, which yielded various indices for goodness of fit. Ten more items were deleted from the model after several iterations of CFA due to lower goodness of fit. The final model contained seven dimensions with 28 items. This model was tested with a sample taken from the general public in addition to a sample of college students. The

results of the model fit tests are presented in Table 2; all 28 items are listed in the *Appendix*, organized by risk domain.

We compared the seven-factor model to other alternative factor solutions. For example, we combined gambling with natural/physical risk to construct a six-factor model, and we also combined gambling with financial risk (Blais & Weber, 2006; Weber et al., 2002) to construct another six-factor model. Furthermore, we combined all three of these domains together to construct a five-factor model. The results showed that the seven-factor solution was still the best-fitting model (see Table 2 for statistical results). As shown in Table 2, the hypothesized seven-factor model (goodness-of-fit index [GFI] = .92, confirmatory factor index [CFI] = .93, incremental fit index [IFI] = .93, root mean square error of approximation [RMSEA] = .05), yielded a better fit than the one-factor model (GFI = .64, CFI = .44, IFI = .44, RMSEA = .12, $\Delta\chi^2$ = 2,966.98, Δdf = 21, $p <$ .001), the five-factor model (GFI = .78, CFI = .71, IFI = .71, RMSEA = .09, $\Delta\chi^2$ = 1,348.23, Δdf = 11, $p <$ .001), or the six-factor

Table 1

Factor Loadings of the 38 Items of the Risk-Taking Scale in Study 1

Domain	Factors						
	1	2	3	4	5	6	7
Moral risk	**.706**	.016	.081	−.050	−.134	.183	−.036
	.688	.063	.159	−.025	−.267	.246	.065
	.655	.099	.146	−.002	.083	.001	.208
	.652	.009	−.013	−.048	.065	.002	−.002
	.631	.111	.140	−.098	.017	.179	.162
	.580	.087	.155	.213	−.043	.099	.221
	.504	.049	.081	−.004	−.084	.047	.192
	.497	.120	−.007	.174	−.028	−.027	−.207
	.450	.049	.147	.192	.187	−.027	.098
Financial risk	.048	**.912**	−.011	−.037	.038	−.006	.081
	.125	**.845**	.058	.003	.106	−.035	.095
	.088	**.814**	−.099	.014	.094	.011	−.124
	.037	**.710**	.148	.210	.086	−.008	.069
	.181	**.682**	−.014	.202	.071	.184	.111
Gambling	.124	.016	**.878**	.228	−.030	.095	−.011
	.146	.014	**.877**	.130	−.010	.110	.074
	.210	−.004	**.831**	.106	.107	.053	.012
	.252	.042	**.777**	.185	−.112	.274	.017
Natural/physical risk	.043	−.046	.196	**.785**	−.125	.226	.078
	−.026	.172	.076	**.757**	.006	.084	.159
	.033	.062	−.016	**.750**	.167	−.090	−.007
	.096	.076	.150	**.717**	.123	.073	−.061
	−.014	.083	.333	**.664**	.013	.148	−.113
Cooperation/competition	−.191	.083	.054	−.031	**.715**	−.076	.187
	−.051	.117	−.072	−.007	**.693**	.065	.071
	−.080	.107	.055	−.028	**.643**	.123	.110
	.025	.059	.067	.173	**.637**	.017	.129
	.197	.219	−.161	.071	**.530**	−.116	−.118
	−.284	−.083	−.067	−.131	**.443**	−.217	.175
	.244	−.130	.028	.203	**.423**	−.050	.001
Reproduction	.145	−.011	.226	.115	−.162	**.819**	.185
	.139	.031	.099	.209	−.045	**.799**	.142
	−.003	.131	.108	.111	−.011	**.687**	−.181
	.278	−.079	.047	−.080	.203	**.549**	−.035
Safety	.123	−.009	−.045	.044	.102	−.032	**.826**
	−.027	−.029	.079	−.005	.117	−.012	**.791**
	.225	.131	.015	−.109	.182	.065	**.579**
	.229	.141	.016	.146	.095	.045	**.551**

Note. Loadings ≥ .40 are shown in **bold**. The loading scores were from an EFA with varimax rotation. We also performed an EFA using oblimin rotation, which yielded an identical seven-factor structure.

Table 2
Model Fit Indices for Factor Structures in the Student and General Public Samples in Study 1

Model	χ^2	df	χ^2/df	$\Delta\chi^2$	Δdf	p	GFI	CFI	IFI	RMSEA
Student sample										
One-factor	3,736.13	350	10.68	2,966.98	21	<.001	.64	.44	.44	.12 (.12–.13)
Five-factor	2,117.38	340	6.23	1,348.23	11	<.001	.78	.71	.71	.09 (.09–.09)
Six-factor 1	1,537.18	335	4.59	768.03	6	<.001	.84	.80	.80	.07 (.07–.08)
Six-factor 2	1,351.93	335	4.04	582.78	6	<.001	.85	.83	.83	.07 (.07–.07)
Seven-factor	769.15	329	2.34				.92	.93	.93	.05 (.04–.05)
Second-order factor	699.78	343	2.04	–69.37	14	<.001	.87	.90	.90	.06 (.05–.06)
General public sample										
One-factor	1,820.09	350	5.20	1,259.59	21	<.001	.65	.44	.45	.12 (.11–.12)
Five-factor	1,169.34	340	3.44	608.84	11	<.001	.76	.69	.69	.09 (.09–.10)
Six-factor 1	855.31	335	2.55	294.81	6	<.001	.82	.80	.81	.07 (.07–.08)
Six-factor 2	876.13	335	2.62	315.63	6	<.001	.81	.80	.80	.07 (.07–.08)
Seven-factor	560.50	329	1.70				.88	.91	.91	.05 (.04–.06)
Second-order factor	640.58	343	1.87	80.08	14	<.001	.87	.88	.89	.05 (.05–.06)

Note. On the basis of previous studies (Blais & Weber, 2006; Weber, Blais, & Betz, 2002), we combined gambling, financial risk, and natural/physical risk as one domain to construct the five-factor model, combined gambling with financial risk as one domain to construct the first six-factor model (six-factor 1), and combined gambling with natural/physical risk as one domain to construct the second six-factor model (six-factor 2). GFI = goodness of fit index; CFI = comparative fit index; IFI = incremental fit index; RMSEA = root mean square error of approximation.

models (Model 1: GFI = .84, CFI = .80, IFI = .80, RMSEA = .07, $\Delta\chi^2$ = 768.03, Δdf = 6, p < .001; Model 2: GFI = .85, CFI = .83, IFI = .83, RMSEA = .07, $\Delta\chi^2$ = 582.78, Δdf = 6, p < .001).

To facilitate the use of our scale in a broader range of applied settings, we further performed a CFA on the sample of respondents from the general public (n = 300) to determine whether a seven-factor solution fit the data better than the alternative factor solutions. Here, the hypothesized seven-factor model (GFI = .88, CFI = .91, IFI = .91, RMSEA = .05) again yielded a better fit than the one-factor model (GFI = .65, CFI = .44, IFI = .45, RMSEA = .12, $\Delta\chi^2$ = 1,259.59, Δdf = 21, p < .001), the five-factor model (GFI = .76, CFI = .69, IFI = .69, RMSEA = .09, $\Delta\chi^2$ = 608.84, Δdf = 11, p < .001), or the six-factor models (Model 1: GFI = .82, CFI = .80, IFI = .81, RMSEA = .07, $\Delta\chi^2$ = 294.81, Δdf = 6, p < .001; Model 2: GFI = .81, CFI = .83, IFI = .80, RMSEA = .07, $\Delta\chi^2$ = 315.63, Δdf = 6, p < .001; see Table 2).

Although the correlations among the seven factors were relatively low (.03–.49) in both the student and the general public samples, a few correlations were higher than .40. In factor analysis, when several factors are moderately or highly correlated with each other, it suggests the existence of a common higher order factor or factors that may affect all of these correlated factors (Chen, Sousa, & West, 2005). Therefore, we conducted a higher order (second-order) factor analysis to see if any higher order factor was affecting individual differences in risk-taking propensity across the domains (Thompson, 2004).

As shown in Table 2, for both the student and general public samples, all of the four indices of goodness of fit (i.e., GFI, CFI, IFI, and RMSEA) and the $\Delta\chi^2$ tests showed that the seven-factor model was the best fitting model, with one exception in which the χ^2 of the second-order factor model was lower than the seven-factor model for the student sample. Thus, overall, we consider the seven-factor model to be the best fitting model. However, the existence of a higher order factor suggests that some cross-domain differences in risk taking, such as men having a higher risk-taking propensity than women across multiple domains (Wang et al., 2009), may be regulated by this higher order factor.

Overall, the factor structure of all three samples consistently identified the same seven distinct domains of risk-taking propensity measured by the ratings of the subjective likelihood of engaging in a risky activity. The Cronbach's α coefficient (an internal consistency measure) for each of the domains was .61 for cooperation/competition, .76 for safety, .68 for reproduction, .75 for natural/physical risk, .72 for moral risk, .84 for financial risk, and .92 for gambling.

Our further analysis of criterion-related validity indicated that a scale of general risk attitude (Meertens & Lion, 2008) had a moderate, positive correlation with risk-taking propensity scores across all seven domains (correlational coefficients ranging from .11 to .46) whereas more specific measures of the sensation-seeking scale (Horvath & Zuckerman, 1993; Zuckerman & Kuhlman, 2000) and the Biological Activity Reaction Test (BART; Lejuez et al., 2002) were selectively correlated with the domains of natural/physical risk and gambling, respectively.

To summarize, we started with 58 items, reduced the number of items to 38 after performing EFA, and ended the analysis with 28 items (4 in each of the seven risk domains) after CFA. The result of this synthetic scale development is the DOSPERT-7 scale, the items of which are listed in the *Appendix*.

The DOSPERT-7 scale differs from many well-studied scales of personality traits such as impulsiveness. First, our measurement is specific to particular risk domains (situations) whereas personality traits are often assumed to be cross-situational and generalizable across task domains. Second, the classification framework used in our measurement of risk-taking propensity differs from that used in some widely used measures of impulsiveness, which also is viewed as consisting of multiple subcomponents. The risk domains in our scale were classified by functionally unique tasks and activities engaged in by human decision-makers (e.g., safety control, reproduction, financial investment, gambling, etc.). In contrast, impulsiveness as a personality trait is classified into subcategories in terms of psychometric properties and information-processing faculty or capacity, such as attentional impulsivity, cognitive instability, and nonplanning (Stanford et al., 2009), or urgency, lack of

premeditation, and sensation seeking (e.g., Whiteside, Lynam, Miller, & Reynolds, 2005).

Study 2: A Twin Study of Domain-Specific Risk Taking

Because most extant twin studies of risk taking have generally focused on only one type of risk, and because no integrative and comparative twin studies have been performed on a scale of domain-specific risk taking (Miles et al., 2001), Study 2 aimed to estimate genetic and environmental influences on individual differences in risk-taking propensity over the seven domains. In Study 2, we used univariate and multivariate models to examine genetic (A), common environmental (C), and nonshared environmental (E) effects for each domain and to determine whether domain specificity in the risk-taking propensity could be obtained at both phenotypic and genetic levels.

Method

Participants and procedure. A total of 240 same-sex twin pairs (108 female pairs and 132 male pairs) sampled from the Beijing Twin Study (BeTwiSt) registry were included in Study 2. The average age of the participants was 20.2 ± 1.83 years. Among these twin pairs, 151 pairs were monozygotic (MZ) and 89 were dizygotic (DZ). For all twin pairs who participated in our study, zygosity was assigned by DNA testing, with classification accuracy of nearly 100%.

Measurement. The twin participants rated their likelihood of engaging in various risky actions described on the DOSPERT-7, developed in Study 1. The Cronbach's α coefficient for the full scale was .86, and its values for the subscales were as follows: .63 for cooperation/competition, .73 for natural/physical risk, .64 for safety, .70 for reproduction, .69 for moral risk, .88 for financial risk, and .90 for gambling. The hypothesized seven-factor model (GFI = .92, CFI = .90, IFI = .90, RMSEA = .05) yielded a better fit than the one-factor model (GFI = .69, CFI = .44, IFI = .45, RMSEA = .12).

Analysis. The analysis was conducted using the different levels of genetic relatedness between MZ twins, who are genetically identical, and DZ twins, who share 50% of their segregating genes on average. By comparing the resemblance between MZ and DZ twin pairs on their risk propensity ratings in each domain, we can decompose the variance and covariance in risk-taking propensity into four components: additive genetic, dominant genetic, shared environmental, and residual environmental including measurement errors. Additive genetic variance (A) results from the sum of allelic effects within or across multiple genes affecting a trait (e.g., body height or risk-taking propensity). Dominant genetic variance (D) comes from interactions between alleles, in which one or more alleles are more dominant than others in determining a trait (e.g., eye color). Shared environmental variance (C) is due to environmental influences shared within twin pairs, such as family or school environment, prenatal influences, parental style, and socioeconomic status. Residual (nonshared) environmental variance (E) results from environmental factors that are unique to each twin in a pair (e.g., idiosyncratic events and experiences). For twins raised together, it is not possible to simultaneously model the C and D contributions because the former decreases and the latter increases

differences between MZ and DZ intrapair correlations (e.g., Neale & Cardon, 1992). Accordingly, the initial model to fit—which includes only one of these components, either C or D—is selected based on whether the MZ correlation is less or greater than twice the DZ correlation. The result is an ACE model in the former case or an ADE model in the latter case.

For all data submitted to the genetic modeling analysis, we controlled for age and gender through multivariable regression. We used the change in χ^2 and Akaike's information criterion (AIC) as the model fit indices in both univariate and bivariate modeling. A significant χ^2 difference suggests that the nested model fits significantly worse than the full model; thus, the full model should be chosen. Otherwise, a nested model with fewer parameters should be considered (Bollen, 1989; Santor, 1999). Likewise, AIC assesses model fit relative to the number of parameters; a lower or negative AIC value indicates a better model fit.

We used univariate and bivariate models implemented in the Mx package (Neale, Boker, Xie, & Maes, 2003) to estimate genetic and environmental effects on risk-taking propensity.

Univariate model analyses were used to partition the variance in risk propensity scores into genetic and environmental effects. For natural/physical risk, ADE was selected as the initial model because ICC_{MZ} was greater than two times ICC_{DZ}. For the other six domains, ACE was selected as the initial model because ICC_{MZ} was less than twice ICC_{DZ}. A full model with all parameters (e.g., ACE) and submodels (e.g., AE, CE, and E) was tested by systematically removing one or two components of variance and using χ^2 difference tests and AIC.

Similar analyses were also performed on the consistency measure of risk propensity (e.g., mean standard deviation of the propensity scores across the seven domains for each participant) to estimate genetic and environmental influences on individual differences in the consistency of risk-taking propensity.

Multivariate model analyses. Bivariate model analyses were used to examine the pairwise genetic and environmental correlations between the seven domains. The analyses were performed on the cross-twin, cross-trait (domain) covariance matrices to decompose the covariance in the risk propensity scores between MZ twins or DZ twins into genetic, shared, and nonshared environmental correlations (Neale et al., 2003). The higher a correlation, the more likely it is that the influences on individual risk propensity are from the same source (e.g., additive genetic) across the related domains. The relative genetic and environmental contributions were assessed by comparing the full model and submodels. Where risk-taking propensity was correlated across more than two domains at both behavioral and genetic levels, trivariate Cholesky decomposition modeling was used to estimate the possible underlying common genetic and/or environmental influences.

Results and Discussion

Univariate model fitting. Univariate models were used to examine the additive genetic effect (A), dominant genetic effect (D), common environmental effect (C), and nonshared environmental effect (E) for each domain. The intraclass correlation coefficients (ICCs) of the seven domains for the MZ and DZ pairs are given in Table 3. The results showed that the MZ correlations were higher than the DZ correlations in five risk domains (natural/physical risk, moral, financial risk, reproduction, and cooperation/

Table 3

Means, Phenotypic Correlations, and Twin Intraclass Correlations of Subjective Ratings of Engaging in Risky Activities in and Among Seven Risk Domains in Study 2

Domain	Mean	Twin correlation		r					
		ICC_{mz}	ICC_{dz}	1	2	3	4	5	6
1. Natural/physical risk	2.64	.64	.3						
2. Moral risk	2.19	.67	.38	.17*					
3. Financial risk	3.81	.57	.33	.26*	.32*				
4. Reproduction	2.4	.55	.4	.29*	.24*	.19*			
5. Cooperation/competition	4.8	.46	.36	.20*	.12	.22*	.03		
6. Safety	3.92	.5	.5	.18*	.45*	.23*	.23*	.34*	
7. Gambling	1.55	.32	.37	.27*	.32*	.23*	.19*	.08	.21*

Note. r = phenotypic correlation after controlling family factors for every twin pair. ICC_{mz} = intra-class correlation coefficient for monozygotic twins; ICC_{dz} = intra-class correlation coefficient for dizygotic twins.
* $p < .05$.

competition), indicating a significant genetic influence on risk propensity in these domains. However, in the gambling and safety domains, the MZ and DZ correlations were similar, suggesting strong environmental influences.

The results of model fitting analyses for each of the seven risk domains are shown in Table 4. AE models had the best fit for the domains of natural/physical risk, moral risk, financial risk, reproduction, and cooperation/competition. The heritability of risk-

Table 4

Univariate Genetic Model Fitting Estimates for Additive Genetic (A), Shared Environment (C), and Nonshared Environment (E) Components of Variance for Domain-Specific Risk-Taking Propensities in Study 2

Domain	Model	χ^2	p	$\Delta\chi^2$	AIC	a^2	d^2/c^2	e^2
Natural/physical risk	ADE	2.10	.55		−3.90	.13	.37	.50
	AE	3.05	.55	.00	−4.95	.48		.52
	E	43.62	.00	41.52	33.62			1
Moral risk	ACE	2.25	.52		−3.75	.45	.00	.55
	AE	2.25	.69	.00	−5.75	.45		.55
	CE	7.45	.11	5.20	−.56		.38	.62
	E	43.59	.01	41.34	33.59			1
Financial risk	ACE	1.69	.64		−4.31	.39	.00	.61
	AE	1.69	.79	.00	−6.31	.39		.61
	CE	5.12	.28	3.43	−2.88		.31	.69
	E	29.01	.00	27.32	19.01			1
Risk in reproduction	ACE	.86	.84		−5.14	.22	.15	.63
	AE	1.31	.86	.45	−6.69	.38		.62
	CE	1.70	.79	.84	−6.30		.33	.67
	E	29.45	.00	28.59	19.45			1
Cooperation/competition	ACE	2.11	.55		−3.89	.22	.07	.71
	AE	2.21	.70	.10	−5.79	.30		.70
	CE	2.97	.56	.86	−5.03		.25	.75
	E	18.12	.00	16.01	8.12			1
Safety	ACE	1.08	.78		−4.92	.16	.18	.65
	AE	1.94	.75	.86	−6.06	.37		.63
	CE	1.60	.81	.52	−6.40		.31	.69
	E	25.97	.00	24.89	15.97			1
Gambling	ACE	7.53	.06		1.53	.05	.16	.78
	AE	8.22	.08	.69	.22	.24		.76
	CE	7.58	.11	.05	−.42		.20	.80
	E	17.62	.00	10.09	7.62			1
Consistency in risk propensity	ACE	45.79	.00		39.79	.39	.01	.59
	AE	45.80	.00	.01	37.80	.41		.59
	CE	48.93	.00	3.14	40.93		.30	.70
	E	71.58	.00	25.79	61.58			1

Note. a^2 = additive genetic effects, d^2 = dominant genetic effects, c^2 = shared environmental effects, e^2 = nonshared environmental effects. ADE, ACE, AE, CE, and E models are based on different combinations of these parameters. The best-fitting model is in bold. Consistency in risk propensity refers to the consistency measure of participants' risk propensity across domains (i.e., the mean standard deviation of the propensity scores across the seven domains for each participant). AIC = Akaike's Information Criterion.

taking propensity ranged from 30% to 48% across these five domains. Nonshared environmental influences accounted for the remainder of the variances in risk-taking propensity whereas shared environmental contributions were close to zero. However, for the other two domains (i.e., gambling and safety), CE models had the best fit, suggesting strong shared and nonshared environmental influences.

With regard to the higher order factor identified in Study 1, the AE model fit as well as the full model ($\Delta\chi^2 = .00$; $a^2 = .44$; $e^2 = .56$) in accounting for its effects on risk-taking propensity across risk domains. Therefore, according to the parsimony principle, the AE model was selected (Bollen, 1989; Santor, 1999).

Individual consistency in risk-taking propensity was measured by the mean standard deviation of the propensity scores across the seven domains. The mean standard deviation scores were best described by an AE model (A = 41%, E = 59%), indicating that shared environmental factors had little influence on an individual's level of consistency in his or her risk-taking propensity across domains.

Caution should be exercised when interpreting the lack of dominant genetic effects in our results. Twin studies generally have low power to detect dominant genetic effects due to the low DZ correlation (Christian & Williams, 2000), especially when the sample size is relatively small.

Bivariate model fitting. On the basis of the covariance matrices of scores of risk-taking propensity between MZ and DZ twins, we examined possible paired correlations across domains in terms of shared genetic or environmental contributions. Of the 21 possible paired genetic correlations, only 5 were significant, showing that individual risk-taking propensity is also largely domain specific at genetic levels (see Table S1 in the online supplemental material for bivariate genetic model fitting estimates for additive genetic, shared environmental, and nonshared environmental components of variance in domain-specific risk-taking propensity). The relatively few genetic correlations were found between the moral and financial domains, between the moral and cooperation/competition domains, and

between natural/physical risk and three other domains (moral, financial, and reproduction). Similar to the results of univariate analyses, AE models had the best fit for the pairs. These AE models had modest genetic correlations (r_g ranging from .11 to .44) and small nonshared environmental correlations (r_e ranging from .01 to .20) between the domains.

Cholesky decomposition model fitting. Of all of the possible pairwise correlations between risk domains, three of them (i.e., financial, moral, and natural/physical risk) had both significant behavioral correlations in risk propensity scores (Figure 1A) and significant genetic correlations in the covariate matrix between MZ twins relative to DZ twins across the pairwise domains. Using trivariate Cholesky decomposition modeling, we explored the common genetic contributions between the three domains (see Figure 1B).

The phenotypic (behavioral) correlations, as shown in Figure 1A, suggest significant interactions among financial, moral, and natural/physical risk taking. Trivariate Cholesky decomposition modeling would help to identify any common genetic factors underlying these behavioral interactions.

The Cholesky decomposition model is shown in Figure 1B, $\chi^2(27) = 45.51$, $p = .014$, RMSEA = .064. A common genetic factor (A_1) was identified, and this factor accounted for a substantial portion of the variation in financial (.63), moral (.22), and natural/physical risk taking (.22). These results suggest that individual differences in risk-taking propensity in the financial, moral, and natural/physical domains are partially affected by a common genetic source.

Our results from urban Chinese samples are not necessarily generalizable to other populations. Additional and more diverse samples would increase the generalizability as well as the precision of the estimates. Thus, meta-analyses would help to integrate the findings of extant twin studies, including and beyond the present twin sample, and to assess genetic and environmental effects on risk taking across different task domains.
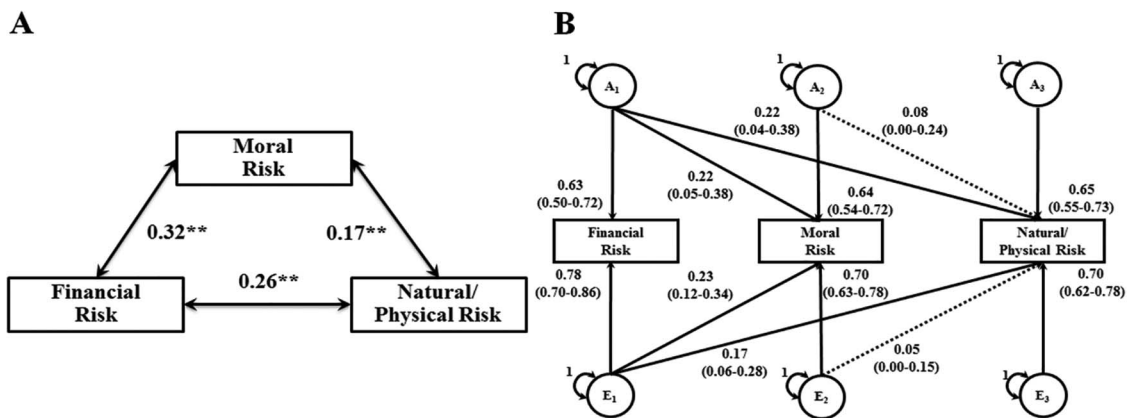

Figure 1. Behavioral and genetic correlations across the domains of financial, moral, and natural/physical risk taking. (A) The behavioral correlation coefficients between the three risk domains (** indicates $p < .01$). (B) the best-fit trivariate Cholesky decomposition modeling of common genetic effects between the three domains with the path estimates and the 95% confidence intervals in parentheses. Measured variables are in rectangles. Latent factors A (additive genetic factors) and E (nonshared environmental factors) are in circles.

Study 3: Meta-Analyses of Twin Studies Across Risk Domains

The extant twin studies of risk-taking behaviors span various domains and differ dramatically in their heritability estimates even within the same domain. To gain a better understanding of the heritability of risk taking in different domains and to disentangle the influences of nature and nurture, we completed meta-analyses of twin studies in each of the seven domains.

Method

We searched for twin studies on risk taking conducted from January 1, 1970 to December 1, 2015 in the following databases: PubMed, PsycINFO, Science Direct, Web of Science, and EBSCO. We limited the search for twin studies to 1970 and thereafter because most behavior-related genetic studies with comparable methods were conducted after 1970 (Bezdjian, Baker, & Tuvblad, 2011). The search terms used included *twin* together with phenotype search terms in each of the seven domains of risk taking. Table S2 in the online supplemental material lists the search terms used in each risk domain.

Inclusion criteria. Five criteria were applied in our meta-analyses. First, the risk-taking behavior examined in a twin study had to match a search term derived from each of the seven identified domains in Studies 1 and 2. Second, the studies must have provided MZ and DZ twin correlations in risk-taking measures. Third, they must have reported sample sizes of MZ and DZ twin pairs. Fourth, the measures of risk-taking behaviors had to be continuous variables. Finally, the meta-analyses included both adolescent (age 13 years and older) and adult samples of participants.

Exclusion criteria. Some studies were excluded from the meta-analyses because of repeated use of the same dataset but with a smaller sample size than in the study included in the analysis. Another reason for exclusion was the presence of nonindependent samples (e.g., using more than one dependent measure of risk taking in the same sample). On the basis of suggested methods of dealing with nonindependent samples (Lipsey & Wilson, 2001; Rhee & Waldman, 2002), we adopted the following strategy: if the sizes of nonindependent samples were identical, then we used weighted averages to compute the effect size; if the sample sizes were not identical, then the largest sample was chosen.

The search process identified 100 papers on twin studies for meta-analyses. Of these 100, there were 14 on cooperation/competition, 9 on financial risk, 16 on gambling, 14 on moral risk, 14 on reproductive risk (i.e., risk of reproductive failure), 11 on safety, and 22 on natural/physical risk. These twin studies are listed in Table S3 in the online supplemental material, sorted by risk domain. The following information is included in Table S3 in the online supplemental material for each study: the population from which the sample was drawn (e.g., general public or special clinical population), sample size, measure(s) of risk taking, assessment method (e.g., web based, questionnaires, clinical diagnosis), effect size, age, sex, and number of pairs by zygosity.

Data analysis. Structural equation modeling was used to perform the genetic model-fitting analyses (Neale et al., 2003).

Seven meta-analyses, one for each risk domain, were conducted on the results of relevant twin studies plus our own data from Study 2. Either intraclass or Pearson product–moment correlations were used to indicate the effect sizes (r). In each risk domain-specific meta-analysis, we compared the ACE, AE, CE, and ADE models for all data, and we identified the model with the lowest $\Delta\chi^2$ and lowest AIC value as the model with the best fit.

Results and Discussion

The parameter estimates of genetic and environmental effects and the fit of ADE, ACE, AE, and CE models for the seven meta-analyses are presented in Table 5. The ADE model was the best-fitting model for financial, natural/physical, and safety risks. The dominant genetic effect was minimal for financial risk taking (4%) and higher but still small for natural/physical risk (8%) and safety (20%). We further estimated broad-sense heritability with both additive and nonadditive genetic effects and found that it varied across the three domains (29% for financial risk, 49% for natural/physical risk, and 55% for safety). The ACE model provided the best fit to the risk-taking data in the domains of cooperation/competition and moral risk. The heritability estimates were 31% for cooperation/competition and 45% for moral risk. In addition, we found small but significant influences of shared environment in these two domains (9% and 13%, respectively). Finally, the AE model was the best-fitting model for gambling and reproduction, identifying 50% of individual differences in gambling and 51% in reproduction due to genetic influences.

As shown in Figure 2, the results regarding broad-sense heritability (A + D) from the meta-analyses were largely consistent with the results of Study 2 in five of the seven domains, with the exceptions of gambling and safety. The heritability estimates for safety and gambling in the present twin study were based on the results of the ACE models. We further divided gambling into two types, addictive (pathological) gambling and general (nonaddictive) gambling, and we compared the relative genetic and environmental influences between these two types. The $\Delta\chi^2$ was significant for type of gambling ($\Delta\chi^2(3) = 84.066$, $p < .01$). Additive genetic influences were larger for addictive gambling (55%), with little shared environmental influence. In contrast, for general gambling, the genetic influences were reduced to 31%, which was more consistent with the results of the meta-analyses.

To examine whether genetic and environmental influences differ by culture or nation, we estimated the moderating effects of this variable on the genetic and environmental influences in the meta-analyses. The samples included in our meta-analyses came from the United States ($n = 38$), Europe ($n = 41$), Australia ($n = 16$), Asia ($n = 3$), and Canada ($n = 2$). For our cultural analysis, we excluded the Asian samples because there were so few. We adopted a similar classification method used in the Tucker-Drob and Bates' study (2016) to divide the remaining samples into U.S. and non-U.S. groups.

We found enough papers for moderation-effect analyses in six domains, all but financial risk. We estimated the moderating effect of culture/nation on genetic and environmental influences in the meta-analyses across the six domains. The results indi-

Table 5
Best-Fitting Models for Twin Studies in the Meta-Analyses Across Seven Domains in Study 3

Model	Fit statistic				Parameter estimate			
	χ^2	df	p	AIC	a^2	c^2	e^2	d^2
Natural/physical risk								
ADE	**275.184**	**169**	**<.001**	**−62.816**	**.41**		**.51**	**.08**
ACE	279.062	169	<.001	−58.938	.48	0	.52	
AE	279.062	170	<.001	−60.938	.48		.52	
CE	751.191	170	<.001	411.191		.36	.64	
Moral risk								
ADE	1,207.192	103	<.001	1,001.192	.59	—	.41	0
ACE	**1,109.382**	**103**	**<.001**	**903.382**	**.45**	**.13**	**.42**	**—**
AE	1,207.192	104	<.001	999.192	.59	—	.41	—
CE	1,858.359	104	<.001	1,650.359	—	.43	.57	—
Financial risk								
ADE	**229.82**	**52**	**<.001**	**125.82**	**.25**	**—**	**.71**	**.04**
ACE	232.41	52	<.001	128.41	.29	0	.71	—
AE	232.41	53	<.001	126.41	.29	0	.71	—
CE	546.92	53	<.001	440.92	—	.19	.81	—
Risk in reproduction								
ADE	636.875	118	<.001	400.875	.51	—	.49	0
ACE	636.58	118	<.001	400.58	.5	.01	.49	—
AE	**636.875**	**119**	**<.001**	**398.875**	**.51**	**—**	**.49**	**—**
CE	1,212.028	119	<.001	974.028	—	.38	.62	—
Cooperation/competition								
ADE	454.023	91	<.001	272.023	.42	—	.58	0
ACE	**442.659**	**91**	**<.001**	**260.659**	**.31**	**.09**	**.6**	**—**
AE	454.023	92	<.001	270.023	.42	—	.58	—
CE	521.171	92	<.001	348.171	—	.33	.67	—
Safety								
ADE	**724.38**	**88**	**<.001**	**548.38**	**.35**	**—**	**.45**	**.2**
ACE	741.73	88	<.001	565.73	.53	0	.47	—
AE	741.73	89	<.001	563.73	.53	0	.47	—
CE	1,221.23	89	<.001	1,043.23	—	.37	.63	—
Gambling								
ADE	388.635	109	<.001	170.635	.5	—	.5	0
ACE	388.635	109	<.001	170.635	.5	0	.5	—
AE	**388.635**	**110**	**<.001**	**168.635**	**.5**	**—**	**.5**	**—**
CE	721.705	110	<.001	501.705	—	.38	.62	—

Note. a^2 = additive genetic effects, c^2 = shared environmental effects, e^2 = nonshared environmental effects, d^2 = nonadditive genetic effects. ADE, ACE, AE, and CE models are based on different combinations of these parameters. The best-fitting model is in bold. AIC = Akaike's Information Criterion.

cated that for safety and cooperation/competition risk, genetic influences played a more significant role in the U.S. samples than in the non-U.S. samples. In contrast, for natural/physical and reproductive risk, the genetic effects were larger in the non-U.S. samples. No significant moderating effects of culture/ nation were found for gambling and moral risk. It appears that this variable interacts with genetic and environmental factors to determine individual differences in risk-taking propensity in a
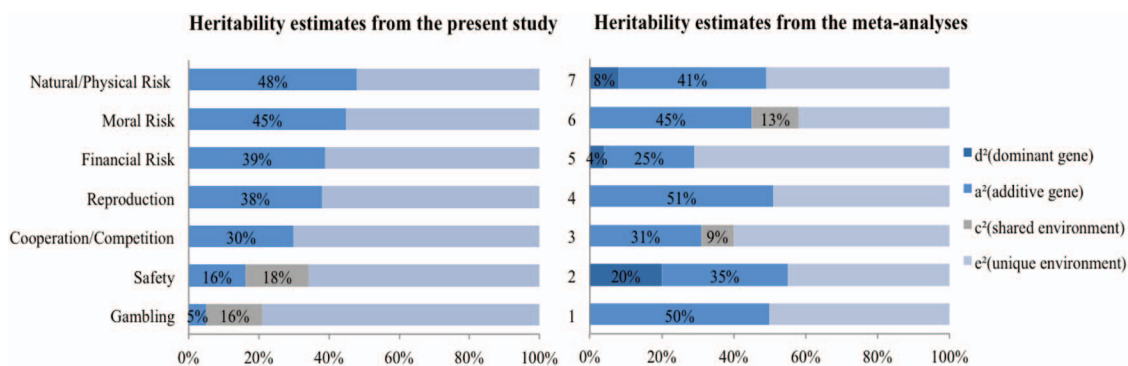

Figure 2. Comparison of heritability estimates from the present twin study and the meta-analyses of 100 twin studies across seven risk domains.

manner that is specific to each risk domain (for details, see Table S4 in the online supplemental material).

General Discussion

In the three studies, we conducted the following two partitioning processes: a partitioning by risk domain and a partitioning of genetic and environmental contributions to risk propensity in each risk domain. In the first partitioning process, we demonstrated that behaviorally distinct domains as measured by a synthetic risk propensity scale (the DOSPERT-7) are also largely independent of each other at the genetic level. Of the 21 possible pairwise correlations for the seven risk domains, only 5 (24%) had limited and significant genetic correlations. Thus, domain specificity in risk-taking propensity was identified at both phenotypic and genetic levels. For the limited correlations between domains, we focused on the three domains that had both significant phenotypic and genetic correlations, finding a common genetic influence underlying financial, natural/physical, and moral risk taking. This result indicated that individuals who are more predisposed to take financial risks may also be inclined to take greater moral and natural/physical risks.

In the second partitioning process, we found that the AE model in Study 2 best accounted for the risk-taking propensity data in five of the seven domains. Across all seven domains, risk-taking propensity was shaped mainly by additive genetic effects and by individually unique (nonshared environmental) experiences, with little contribution from shared environmental experiences. In Study 3, we synthesized the results from our own twin study (Study 2) with previous twin studies of risk-taking propensity by conducting a series of separate meta-analyses across the seven risk domains. The heritability estimates yielded from these meta-analyses varied from 29% to 55%. Again, shared environmental contributions were rather limited (0%–13%; see Figure 2). A practical implication of these results is that prevention and intervention of excessive risk taking should pay relatively greater attention to unique individual experiences and develop personalized approaches.

Our analysis suggests that people's risk-taking propensity is more likely to be regulated by additive genetic influences (i.e., a function of multiple genes summed over loci) rather than dominant genetic influences. Although we found little dominant genetic effect in our twin study, the meta-analyses indicated that dominant genes accounted for 20%, 8%, and 4% of the variance in the domains of safety, natural/physical, and financial risk, respectively, suggesting that some individuals are born to be more cautious or reckless than others. Both additive and dominant genetic effects contributed to the individual differences in risk-taking propensity.

With regard to environmental effects, both our twin study and the meta-analyses showed that individual differences in risk-taking propensity stems mainly from nonshared environmental and personally unique experiences. However, this does not mean that family, school, and other shared environmental factors play no role in shaping risk-taking propensity. The literature suggests that people, even identical twins living together, differently perceive and react to the same environments (Hanscombe, Haworth, Davis, Jaffee, & Plomin, 2010). Thus, it is likely that the impact of shared environmental influences would be idiosyncratic because of different individual perceptions and coping mechanisms.

It is of interest to compare the results of our meta-analyses of twin studies on risk-taking propensity to those from meta-analyses of twin studies on other psychological variables, including intelligence (Bouchard, 2004), personality (Vukasović & Bratko, 2015), social behavior (Li, Cheng, Ma, & Swan, 2003), and mental disorders (Burt, 2009b). Overall, the pattern of relative contributions of heredity and environment to risk-taking propensity was quite similar to those in twin studies on personality traits. Heritability estimates from the meta-analyses ranged from 29% to 55% across the seven domains. By comparison, the heritability estimates for personality traits, such as neuroticism (37%), openness (41%), and impulsivity (50%), and for some risky social behaviors such as smoking (46%) and rule breaking (48%), are in a similar range of moderate influence (Bezdjian et al., 2011; Burt, 2009a; Li et al., 2003; Vukasović & Bratko, 2015). In addition, our Studies 1 and 2 found little effects of shared environmental influence on individual risk-taking propensity across the seven domains. Likewise, shared environment has been found to play a negligible role in personality and intelligence (Bouchard, 2004). In contrast to these results on risk-taking propensity and personality, twin studies on adolescent psychopathology and aggression revealed significant influences of a shared environment (Burt, 2009b; Cleveland, 2003).

In addition to risk-domain specificity, the results of Studies 1 and 2 also revealed significant individual differences in consistency of risk-taking propensity across domains. If a person's risk-taking propensity is relatively consistent across domains, then his or her risk behavior becomes more predictable from one domain to another. Our results showed that individual differences in cross-domain consistency of risk-taking propensity depended on additive genetic (41%) and nonshared environmental (59%) factors, with little contribution from the shared environment (e.g., peer groups, family, or common education). As suggested by other researchers, such individual differences in risk-taking consistency across domains may be rooted in specific personality traits, such as neuroticism and agreeableness (Soane & Chmiel, 2005). Following this lead, future research could further explore the genetic basis underlying the correlations between certain personality traits and consistency in risk taking across domains.

The results of Studies 2 and 3 were largely consistent in five of the seven domains, except for gambling and safety. As one possible reason for this inconsistency, distinctions between trait and state risk taking may partially explain the lack of genetic effects on gambling propensity in Study 2. Previous twin studies have shown that state anxiety is largely environmentally influenced whereas trait anxiety is more genetically controlled (Lau, Eley, & Stevenson, 2006; Legrand, McGue, & Iacono, 1999). In Study 2, the items used on the DOSPERT-7 scale to assess gambling propensity were all state and situation dependent rather than trait and disposition related. When we separated state-dependent general gambling from addictive gambling, the environmental influences accounted for a majority (68%) of variance in general gambling propensity whereas genetic influences accounted for a majority (57%) of variance in addictive gambling.

Second, distinctions between proactive and reactive risk taking may partially explain the lack of genetic effects in the safety domain in Study 2. Proactive choices involve whether to take

precautionary action in anticipation of a risk (e.g., preventing fire hazards). In contrast, reactive choices determine what actions to take when a person is actually at risk (e.g., escaping from a burning house). The safety items on the DOSPERT-7 were all proactive whereas safety control behaviors measured in the other twin studies included in the meta-analysis were largely reactive. We propose that, when compared with reactive risk taking, proactive choices are more likely to be determined by environmental factors such as education, personal experiences, and conditioning.

The meta-analyses showed that the two risk domains with the greatest genetic influences were safety (55%) and reproductive risk taking (51%). Throughout hominid evolution, our ancestors have always managed to do two things: survive to reproductive age and reproduce. Thus, the biggest evolutionary risk is the risk of reproductive failure. Evolution by natural selection should have prioritized proximate mechanisms to deal with risks that directly jeopardize survival and reproductive fitness over those that indirectly affect survival and reproduction (e.g., social and relational risks).

The significant genetic contribution (45%) to moral risk taking was not expected. This finding suggests a strong evolutionary origin of morality and supports the proposition that moral risk taking (e.g., the likelihood of violating a collective moral code) is not only learned and sociocultural but also innate and heritable (Joyce, 2007). Our analysis comparing the U.S. and non-U.S. samples revealed that sociocultural factors indeed play a significant role in modulating risk-taking propensity in a domain-specific manner. These results call for more study of the cultural effects on individual differences in risk taking and their genetic and environmental mechanisms.

In summary, the present studies are the first to partition genetic and environmental influences on domain-specific risk taking, both comprehensively across risk domains and synthetically in a series of meta-analyses of twin studies within each domain. Overall, the results support the notion of domain specificity in risk-taking propensity; highlight the roles of additive genetic and nonshared environmental factors in shaping risk-taking propensity (and individual consistency in risk-taking propensity) across domains; and reveal limited genetic interactions among moral, financial, and recreational risk taking.

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(Appendix follows)

Appendix

Domain-Specific Scale of Risk-Taking Across Seven Domains (DOSPERT-7)

For each of the following statements, please indicate your **likelihood** of engaging in each activity or behavior. Provide a rating from **1 to 5**, using the following scale: **1** = *very unlikely*, **2** = *relatively unlikely*, **3** = *not sure*, **4** = *likely*, **5** = *very likely*

Natural/physical risk
   Climbing an unexplored and uninhabited plateau alone to take pictures of spectacular scenery.
   Going on a vacation in a third-world country without prearranged travel and hotel accommodations.
   Going down a ski run that is beyond your ability or closed.
   Periodically engaging in a dangerous sport (e.g., mountain climbing or sky diving).

Moral risk
   Having an affair with a married man or woman.
   Cheating on an exam.
   Revealing a friend's secret to someone else.
   Passing off somebody else's work as your own.

Financial risk
   Investing 10% of your annual income in a moderate growth mutual fund.
   Investing 5% of your annual income in a very speculative stock.
   Investing 10% of your annual income in a new business venture.
   Investing 10% of your annual income in government bonds (treasury bills).

Reproduction risk
   Getting sterilized so you cannot have children but have more leisure time and more financial flexibility.
   Exposing yourself to chemicals that might lead to birth defects for a high-paying job.
   Participating in a medical study that pays a large amount of money but has some chance of making you sterile.
   Postponing having your first child to an age over 35 to develop your career.

Cooperation and competition
   Physically intervening between two friends who are aggressively pushing each other to prevent a fight.
   Lending an amount of money equal to your monthly income to a friend in urgent need.
   Disagreeing with an authority figure on a major issue.
   Publicly refuting anyone who disparages your hometown.

Safety
   Riding a motorcycle without a helmet.
   Sunbathing without sunscreen.
   Regularly eating high-cholesterol foods.
   Walking home alone at night in an unsafe area of town.

Gambling
   Betting a day's income at the horse races.
   Betting a day's income at a high-stake poker game.
   Betting a day's income on the outcome of a sporting event.
   Spend a week's income at a casino.