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Genes Unite Executive Functions in Childhood

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Abstract

Individual differences in children's executive functions (EFs) are relevant for a wide range of normal and disordered psychological outcomes across the lifespan, but the origins of variation in child EFs are not well understood. We used a racially and socioeconomically diverse sample of 505 3rd-8th grade twins from the Texas Twin Project to estimate genetic and environmental influences on a common EF factor and on variance unique to four core EF domains: Inhibition, Switching, Working Memory, and Updating. As has been previously demonstrated in young adults, the common EF factor was 100% heritable, indicating that correlations among the four EF domains are entirely attributable to shared genetic etiology. Nonshared environmental influences were evident for variance unique to individual domains. General EF may thus serve as an early life marker of genetic propensity for a range of functions and pathologies later in life.

Executive functions (EFs) are supervisory cognitive processes that monitor, coordinate, and control the execution of other cognitive operations necessary for learning and everyday functioning. Across the lifespan, there exist marked individual differences in EF abilities, which include temporary storage of information simultaneous with cognitive processing (working memory), monitoring incoming stimuli and replacing old information with new information (updating), rapid shifting between cognitive operations (switching), and effortful inhibition of prepotent responses (inhibition). The neural bases for EFs are well studied, with early research implicating the prefrontal cortex as fundamental to EFs and more recent research implicating a complex and distributed network of brain regions as underlying executive processes (Carpenter, Just, & Reichle, 2000; Collette, Hogge, Salmon, & Van der Linden, 2006). EFs are commonly conceptualized as psychological intermediaries between neurobiology and complex thought and behavior, including both normal-range individual differences and clinical disorders (e.g., Best, Miller, & Naglieri, 2011; Elliott, 2003; Finn, Gunn, & Gerst, 2014; Young et al., 2009). Although much of the research on EFs has been based on adult samples, a growing body of developmental research implicates childhood EFs as related to a host of normative psychological outcomes, such as academic achievement and externalizing problem behaviors, as well as childhood onset

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psychiatric disorders such as attention deficit hyperactivity disorder and autism (Pennington & Ozonoff, 1996; Zelazo et al., 1997), both concurrently and prospectively.

Among adults, behavioral genetic studies of EFs have highlighted the importance of genetic influences on these abilities. Individual differences in performance on individual EF tasks are moderately heritable (e.g., Ando, Ono, & Wright, 2001; Kremen et al., 2009; Lee et al., 2012; Vasilopoulos et al., 2012). When individual tasks are combined to measure broader EFs, these abilities – including inhibition, switching, and updating – “are almost entirely genetic in origin” (Friedman, Miyake, Young, Defries, Corley, & Hewitt, 2008).

Additionally, the covariation among EF domains, as represented by a single higher-order EF factor, is also nearly 100% heritable. Thus, by adulthood, non-genetic variance in environmental experience accounts for variation in EF only narrowly, that is, at the level of performance on a specific task; at the level of the construct, identical twins' EFs are perfectly correlated. However, it is currently unclear whether the outstandingly high heritability of general EF extends downward to childhood EFs or whether adulthood represents a developmental apex for genetic influences.

Very few behavioral genetic studies of child EF exist, and those that do have focused on individual EF tasks in isolation, rather than broader EF factors (e.g., Kuntsi et al., 2006; Luciano et al., 2001; Polderman et al., 2006; Schachar, Forget-Dubois, Dionne, Boivin, & Robaey, 2011; Wang & Saudino, 2013; Wang, Deater-Deckard, Cutting, Thompson, & Petrill, 2012). Such task-level analyses are unable to differentiate genetic and environmental influences on nonexecutive demands of each task from those specific to EF. Other studies (e.g., Cuevas et al., 2014) have examined parent-child resemblance for more general EF composites but have been unable to distinguish the extent to which such resemblance derives from genetic versus shared environmental factors. We are aware of no studies in childhood that have both implemented genetically informative designs capable of distinguishing genetic from environmental effects and focused on broader EF factors representing variance common to multiple EF tasks as separate from unique, potentially non-executive, variance.

The heritability of EFs in childhood might be substantially lower than in adulthood, as developmental increases in genetic influence have been observed for multiple phenotypes. For instance, meta-analyses (Briley & Tucker-Drob, 2013; Haworth et al., 2009) have indicated that the heritability of cognitive ability increases continuously from less than 20% in early childhood to upwards of 70% by early adulthood. From middle childhood forward, these increases primarily result from the amplification of the same genetic factors over time (Briley & Tucker-Drob, 2013; Tucker-Drob & Briley, 2014), possibly as a result of dynamic processes whereby children select and evoke cognitively stimulating experiences on the basis of genetically influenced traits, which further serve to differentiate their cognitive ability by genotype over development (Tucker-Drob, Briley, & Harden, 2013). Should EFs show substantially lower heritability in childhood than has been reported in early adulthood, this may point to the sensitivity of EFs to similar dynamic processes over development.

Alternatively, it is possible that EFs are nearly entirely genetic in origin even in childhood. In such a situation, individual differences in EFs may act as genetically influenced aptitudes

that are expressed early and serve as foundations onto which higher-order cognitive processes are scaffolded. Should childhood EFs prove to be similarly high in heritability as adult EFs, they may serve as developmental endophenotypes: early life markers of genetic risk for a cross-cutting range of later life functions and pathologies (Gottesman & Gould, 2003). Researchers who are interested in understand the mechanisms of genetic risk for these complex, multidetermined outcomes would thus be able to study variables that are mechanistically more proximal to the genotype and less “diluted” by extraneous influences. Developmental endophenotypes could also be leveraged in applied settings to identify children who are at genetic risk for – but who have not yet expressed – maladaptive outcomes and who might therefore be the best candidates for preventative treatments or interventions.

The current paper represents the first comprehensive multivariate behavioral genetic analysis of executive functions in childhood. Using a population-based sample of 3rd through 8th grade twins, we investigate genetic and environmental effects on a multivariate battery of four EF domains: Inhibition, Switching, Working Memory, and Updating.

Method

Sample

Data were drawn from 505 individuals in grades 3-8 from the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013) who participated in an ongoing, IRB-approved, in-laboratory study at the University of Texas at Austin. For the current report, data were available for a total of 272 pairs (233 twin pairs and 39 pairs from triplet sets). Participants ranged in age from 7.89 to 15.25 years ($M = 10.97$, $SD = 1.74$) and were 52.1% female, 64.6% Non-Hispanic White, 18.6% Hispanic, 6.7% African American, 2.0% Asian, 1.2% other race/ethnicity, and 6.9% two or more races/ethnicities. 31.2% of participating families reported having received a form of means-tested public assistance such as food stamps. Thus, the current sample is comparable in size to and considerably more diverse than the sample that served as the basis for Friedman et al.'s (2008) finding of nearly 100% heritability of EF factors in young adulthood ($N = 293$ pairs, approximately 90% Non-Hispanic White; Rhea, Gross, Haberstick, & Corley, 2006). As data collection for our study is predominantly conducted each summer at a rate of ≈ 100 -150 pairs per year, we decided to proceed with the current analysis after the second summer of data collection, such that our sample size would approximate that of Friedman et al. (2008).

Zygosity of same-sex twins was assessed by a latent class analysis of parent and experimenter ratings of physical similarity. Latent class analyses of physical similarity ratings have been found to be over 99% accurate as compared to genotyping (Heath et al., 2003). This resulted in a final sample of 84 (30.8%) monozygotic pairs, 97 (35.6%) same-sex dizygotic pairs, and 89 (32.7%) opposite-sex dizygotic pairs. Behavioral genetic analyses that excluded opposite-sex twin pairs produced a very similar pattern of results to those reported here.

Measures

Twelve experimental tasks were selected to assess individual differences in four EF domains: Inhibition, Switching, Working Memory, and Updating (see Table 1). As EF tasks are generally known for suffering from poor reliability relative to cognitive ability measures (Miyake et al., 2000), we placed considerable emphasis on selecting tasks that previously have been reported to have strong psychometric properties in developmental samples. Tasks were administered either orally, by computer (E-Prime 2.0 and Inquisit 4 programs run on Dell Optiplex 3010 computers), or by paper and pencil.

To maintain consistency with the broader EF literature, timed responses were converted to a reaction time metric. Switch costs and inhibition costs were reflected so that higher scores indicated better performance. To correct for positive skew, Trail Making and Local-Global scores were log-transformed, and N-Back and Listening Recall scores were square rooted. Block-level Stop Signal scores were omitted for failure to stop less than 25% or more than 75% of the time, failure to respond to Go trials over 60% of the time, Go trial errors more than 10% of the time, and SSRTs less than 50ms (Congdon et al., 2010). SSRTs were averaged across blocks for the 91% of participants for whom block-level data remained. Plus-Minus scores falling more than 3 *SD* from the mean were Winsorized to next least extreme value. Additional omitted scores were due to administration error. All analyses use standardized scores. We controlled for age-related differences in performance by regressing latent factors onto age in all models.

Phenotypic Analyses

For all phenotypic analyses, the sample was treated as individual cases. Analyses were run using *Mplus* version 7.11 (Muthén & Muthén, 2012). We used the Complex Survey option in *Mplus* to correct for the nonindependence of observations that arises from having individuals embedded in the same family. Each of the twelve tasks was specified to load onto one of four latent variables representing Inhibition, Switching, Working Memory, and Updating ability. This latent variable approach allowed us to extract factors representing variance common across selected tasks as separate from task-specific (and potentially non-executive) variance.

We fit a series of confirmatory factor models to evaluate possible relationships among the EF tasks: (1) a four-factor model in which four distinct EFs sufficiently accounted for variation in task performance, (2) a three-factor model in which Updating and Working Memory tasks were modeled as indicators of a single latent variable, (3) a three-factor model in which Inhibition and Switching tasks served as indicators of a single latent variable (4) a two-factor model in which Updating and Working Memory were combined into one latent factor, and Switching and Inhibition were combined into a second factor, and (5) a one-factor model in which all tasks were regressed onto a single latent variable. In order to test the principle that distinct EFs tap a common executive component, Models 1 through 4 included a latent, Common EF factor for which all first-order latent factors served as indicators. Model fit was assessed by chi-square (χ^2), which measures badness of fit of the model to the data; Root Mean Square Error of Approximation (RMSEA), which indicates the overall degree of discrepancy between the observed covariance matrix and a model-

implied covariance matrix; Bentler Comparative Fit Index (CFI), which compares the model to a baseline model in which no variables are interrelated; and Akaike Information Criterion (AIC), which enables the comparison of non-nested models. To compare model fit, we computed scaled chi-square difference statistics.

Behavioral Genetic Analyses

Our primary behavioral genetic analyses modeled phenotypic variances as the sum of three factors: additive genetic influences (A) that serve to make individuals who are genetically more related (e.g., monozygotic twins compared to dizygotic twins) more similar on an outcome of interest; shared environmental influences (C) that serve to make children raised in the same family more similar, regardless of genetic relatedness; and nonshared environmental influences (E) that serve to differentiate children raised in the same family, even when genetically identical. We also fit models in which the C factors were dropped. One of these consisted of only A and E factors, while the other allowed for contributions from A and E factors, along with a factor representing dominance genetic effects (D), which are nonadditive. Using the best-fitting phenotypic model for guidance, we estimated the relative contributions of the genetic and environmental factors to variance at three levels of measurement: that of the Common EF factor, that unique to the domain-specific factors, and that unique to the individual tasks. All behavioral genetic analyses used the Complex Survey option in *Mplus* to correct for the nonindependence of observations that arose from having multiple “twin” pairs from each set of triplets.

Results

Table 2 reports descriptive statistics for the twelve executive function tasks. For each Inhibition and Switching task that compared RTs across non-executive and executive conditions, there was a mean RT cost associated with the respective executive skill. Reliabilities were generally moderate to high for individual conditions but occasionally somewhat lower for difference scores, representing person-specific Switching and Inhibition costs, as is typical for the literature. Reliabilities for the Updating and Working Memory tasks were also generally moderate to high. Age correlations, within-twin (phenotypic) correlations, and cross-twin correlations in task performance are provided in Tables S1-S3 of the online supplement.

Confirmatory Factor Models

We compared four factor structures to determine which model to enter into behavioral genetic analyses. Table 3 presents standardized factor loadings from these competing models. Our primary model was a hierarchical factor model consisting of four first-order EF domains and a higher-order Common EF factor (Model 1). The fit of this full model was excellent, $\chi^2(58) = 62.31$, $p = .326$, RMSEA = .01, CFI = .997 (see Table 4). Factor loadings of individual tasks on the first-order factors were all significant and generally in the moderate range, with the exception of lower – yet still significant – loadings for the Mickey, Stop Signal, and Plus Minus tasks. This overall pattern of loading magnitudes (median = .62, mean = .54) is comparable to previous EF research in adult samples: Miyake et al. (2000) reported a median loading of .60 and a mean loading of .50, while Friedman et al.

(2008) reported a median loading of .63 and mean loading of .59. Loadings of the first-order factors on the higher-order Common EF factor, when standardized relative to their total variances, were moderate in range (.33, .61, .75, and .78 for Inhibition, Switching, Working Memory, and Updating, respectively). However, because each of the first-order factors was also regressed on age (see final row of Table 3), such loadings are semi-partial with respect to age; the loadings are therefore attenuated relative to what they would be in an age-homogenous sample. When standardized relative to variance that was unique of age – that is, partial with respect to age and therefore more directly comparable to loadings from an age-homogeneous sample – these higher-order loadings were large (.66, .80, 1.00, and .92 for Inhibition, Switching, Working Memory, and Updating, respectively), as has often been found in child samples (e.g., Lee et al., 2013). Model-implied semi-partial correlations among the first-order factors were .20 (In-Sw), .25 (In-WM), .26 (In-Up), .46 (Sw-WM), .48 (Sw-Up), and .59 (WM-Up). Model-implied partial correlations among the first-order factors were .52 (In-Sw), .65 (In-WM), .60 (In-Up), .79 (Sw-WM), .73 (Sw-Up), and .91 (WM-Up).

We tested whether a number of more parsimonious models could account for the data as well as the full hierarchical four-factor model. Model 2 was a hierarchical three-factor model in which Working Memory and Updating tasks served as indicators for the same factor. Though model fit was good overall ($\chi^2(60) = 82.19$, $p = .030$, RMSEA = .03, CFI = .984), there was a significant decrease in fit compared to Model 1 ($p < .001$). In Model 3, Inhibition and Switching tasks were loaded onto the same factor, while Working Memory and Updating remained independent. The model fit the data well ($\chi^2(60) = 76.86$, $p = .07$, RMSEA = .02, AIC = 15139.49), though not as well as Model 1 ($p < .001$). Model 4 was a two-factor model that consisted of a combined Inhibition and Switching factor and a combined Working Memory and Updating factor. The decrement in model fit ($\chi^2(63) = 97.21$, $p = .004$, RMSEA = .03, CFI = .976), as compared to Model 1, was even more pronounced ($p < .001$). Lastly, we considered the possibility that the commonalities among the tasks and factors could be explained by a unitary dimension (Model 5). Although all factor loadings onto the Common EF remained significant and model fit was acceptable ($\chi^2(65) = 127.623$, $p < .001$, RMSEA = .044, CFI = .956), this model fit appreciably worse than all other models ($p < .001$). Additional model fit statistics and comparisons are provided in Table 4. Based on these comparisons, Model 1 was accepted as the best fitting model.

Age Invariance Models

Age-related differences in the measurement properties of the EF tasks could distort estimates of genetic and environmental influence. To address this concern, we divided the sample into relatively equally sized groups younger children (< 11 years) and older children/adolescents (≥ 11 years) and tested for measurement invariance. We first fit an unconstrained model in which the hierarchical factor structure identified in Model 1 was identical across age groups, but factor loadings were allowed to differ. Constraining the loadings of each task on its respective factor to be equal across groups did not result in an appreciable decrement in model fit ($p = .534$), thus indicating measurement invariance across age groups.

Behavioral Genetic Models

The best-fitting model (Model 1) from the confirmatory factor analyses specified a hierarchical structure with each task loading onto one of four broad EF domains (Inhibition, Switching, Working Memory, and Updating) that in turn loaded onto a higher-order Common EF factor. This structure served as the basis for our behavioral genetic analyses. We first fit a model that estimated A, C, and E influences operating on the Common EF factor, individual EFs, and specific tasks. As depicted in Figure 1 and Table 5, the standardized A coefficient for the Common EF factor equaled 1.00 ($p < .001$), indicating that genetic influences on the Common EF factor mediated 100% of the variance common to the domain-specific factors. Of the domain-specific factors, only Switching showed genetic influence independent of the Common EF factor ($a = .59$, $p < .001$). We also observed significant unique non-shared environmental influence on Working Memory ($e = .38$, $p = .003$) and Updating ($e = .24$, $p = .028$). Significant residual genetic effects were present for seven of the twelve tasks, and all tasks exhibited significant non-shared environmental effects. The shared environment significantly contributed to residual variance of only one task, Stop Signal ($c = .28$, $p = .021$).

We next fit an AE model, which yielded a pattern of results very similar to that of the ACE model: 100% additive genetic influence on the Common EF factor, unique genetic influence on the Switching domain and seven tasks, and unique non-shared environment influence on Working Memory, Updating, and all twelve tasks. A model fit comparison revealed that the AE and ACE models did not significantly differ in terms of chi-square ($p = .092$), meaning there was no loss in fit to the data when shared environment parameters were dropped completely.

Finally, we fit an ADE model representing the possibility that dominance genetic effects explained the observed task and factor correlations better than additive genetics alone. Genes continued to explain over 99% of the variation in Common EF performance; additive genetics contributed 77.4% ($p < .001$), while dominance genetics contributed the remaining 23.0% ($p = .177$). The non-shared environment accounted for less than 1% of variation in the Common EF factor. Dominance genetic effects significantly contributed to unique variance in Switching performance, as well as to residual variance of five tasks. After accounting for dominance effects, additive genetics contributed significantly to unique variance in only one task. Model fit, as indexed by chi-square, did not differ significantly from that of the AE model ($p = .248$). The AE model performed best in terms of the AIC fit index, which takes into account model parsimony.

Discussion

Despite widespread interest in EFs as explanatory mechanisms for the development of a host of psychological and social outcomes, there has been surprisingly very little behavioral genetic work on EFs in childhood. Motivated by provocative findings of substantial heritability of EF factors in young adults (Friedman et al., 2008), the current study applied behavioral genetic methods to estimate the magnitude of genetic and environmental influence on individual differences within a hierarchical factor structure of EFs in childhood.

Our results indicate that an exclusively genetic factor mediates 100% of the variance common to all four EF domains that we examined: Inhibition, Switching, Working Memory, and Updating. That we found this high level of heritability in a childhood sample is particularly striking in light of strong evidence that other phenotypes, such as general intelligence, are only modestly heritable in childhood and increase in heritability into adulthood (Haworth et al., 2009). The non-shared environment contributed significantly to variance specific to the Working Memory and Updating factors, as well as to potentially non-executive variance specific to each individual task, but not to the Common EF factor. No appreciable effects of the shared environment were apparent at any level of analysis. Together, these results indicate that EFs in childhood are united by shared genetic influences, yet distinguishable as a result of both genetic and non-shared environmental contributions to specific EF domains and tasks.

While our main findings are consistent with the genetic architecture uncovered for young adults by Friedman et al. (2008), there was one notable difference. In contrast to Friedman et al. (2008), we did not detect genetic effects specific to the latent Updating factor above and beyond those mediated by the Common EF factor. This may indicate that the genetic factors that distinguish EFs from one another are not fully expressed until later in development.

The finding that the Common EF factor is entirely heritable in middle childhood has important implications for our understanding of how EFs develop over time and the mechanisms through which they are associated with important psychosocial sequelae. In combination with accumulating evidence that childhood EFs predict a cross-cutting range of academic, economic, and mental health outcomes later in life, our results suggest that child EFs may act as developmental endophenotypes – or prodromal markers – for an array of genetically influenced psychological, social, and health outcomes. Not only does this suggest that EFs have the potential to provide researchers “simpler clues to [the] genetic underpinnings” (Gottesman & Gould, 2003, p. 636) of such outcomes, it also suggests that EFs might be used to identify children at genetic risk for as yet unexpressed maladaptive outcomes, and could therefore be targeted in early interventions.

Our findings also open exciting avenues for future work. First, in light of the strong theoretical and empirical link between EF and neurobiology, it will be important to test the extent to which the neural bases of EF are themselves genetically influenced and whether such genetic factors are fully captured by behavioral EF measures. Second, although our findings indicate a strong statistical link between the Common EF factor and genetic variation, it is well known that heritability may encompass variation resulting from gene $\times$ environment interactions in addition to more direct genetic main effects. Future work will be necessary to test for gene $\times$ environment interactions involving EFs. For instance, do gene-by-socioeconomic status interactions observed on intelligence and achievement (Tucker-Drob, Briley, & Harden, 2013) occur on EFs? Alternatively, are genetic influences on EFs equally expressed across the range of socioeconomic status but differentially related to intelligence and achievement across socioeconomic strata? Third, it will be important to test for gene-environment correlations with respect to EFs. If dynamic amplification processes involving gene-environment correlations serve as the basis for the strikingly high heritability of EF, as has been postulated to be the case for the heritability of cognitive

ability (Tucker-Drob, Briley, & Harden, 2013), such processes would need to primarily unfold very early in childhood, as our results indicate that heritability has already approached a maximum by middle childhood. Finally, future research will be necessary to test the extent to which interventions to boost EF attenuate or magnify genetic variation in EF. Investigating such questions has the potential to reveal key mechanisms underlying the development of a range of psychological and social outcomes, and doing so may better inform interventions and policies that promote psychological and social well-being.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Hierarchical multivariate twin model for additive genetic (A), shared environmental (C), and non-shared environmental (E) contributions to EF performance. Numbers on arrows represent standardized factor loadings. Our model controls for age-effects at the level of the first-order factors (Inhibition, Switching, Working Memory, and Updating). Because the purpose of this analysis was to understand the relative contributions of genetic and environmental influences to individual differences, as separate from age-related differences, the loadings of the first-order factors have been standardized relative to their age-independent variance. Bolded paths and estimates indicate significance at $p < .05$.

Table 1

Descriptions of Tasks and Measured Outcomes

Executive Function	Task	Source	Paradigm	Dependent Variable
Inhibition	Animal Stroop	after Wright, Waterman, Prescott, & Murdoch-Eaton (2003)	Verbally identify animal drawings Conditions Congruent: Animal face matches body Incongruent: Animal face does not match body; identify based on body Neutral: Face area blank; identify based on body	Inhibition Cost: Mean RT of incongruent trials minus mean RT of congruent and neutral trials
Inhibition	Mickey	Lee et al. (2013)	Press button indicating which side of a screen Mickey Mouse face appears; ignore squares that flash before Mickey appears Conditions Congruent: Square flashes on same side as Mickey Incongruent: Square flashes on opposite side of Mickey Neutral: Squares flash on both sides	Inhibition Cost: Mean RT of incongruent trials minus mean RT of congruent and neutral trials
Inhibition	Stop Signal	after Logan, Schachar, & Tannock (1997), Verbruggen, Logan, & Stevens (2008)	Indicate where an arrow points, but do not respond when tone sounds after arrow presentation	Stop Signal Reaction Time: k th RT for "go" trials minus mean stop signal delay, where k is the product of the probability of responding to a stop signal and the number of responses, and stop signal delay is the delay between arrow and stop signal presentation
Switching	Trail Making	Salthouse (2011)	Connect circles containing numbers and letters Conditions Numbers: Connect circles in numerical sequence Letters: Connect letters in alphabetical order Numbers-Letters: Connect numbers and letters in alternating fashion, still in numerical and alphabetical order Letters-Numbers: Connect letters and numbers in alternating fashion, still in numerical and alphabetical order	Switch Cost: Mean RT of alternating conditions minus mean RT of simple conditions
Switching	Plus-Minus	after Miyake et al. (2000)	Complete simple addition and subtraction problems Conditions Addition: Add 1 to each number Subtraction: Subtract 1 from each number	Switch Cost: Mean RT of alternating conditions minus mean RT of simple conditions

Executive Function	Task	Source	Paradigm	Dependent Variable
Alternating: Alternate between adding and subtracting 1				
Switching	Local-Global	after Miyake et al. (2000)	Verbally identify letters and shapes composed of smaller letters and shapes, respectively Conditions Local: Name the small, constituent letters or shapes Global: Name the large, outlined letter or shape Alternating: Alternating between naming local and global letters or shapes	Switch Cost: Mean RT of alternating conditions minus mean RT of simple conditions
Working Memory	Symmetry Span	after Kane et al. (2004)	View/encode square flashing on grid and, on alternating trials, indicate whether geometric display is symmetrical; recall order and location of increasing numbers of flashing squares	Total number of squares correctly recalled
Working Memory	Listening Recall	after Daneman & Carpenter (1980)	Listen to single letters and sentences presenting in alternating fashion, determine whether sentences make sense; recall order of increasing numbers of presented letters	Total number of letters correctly recalled
Working Memory	Digit Span Back	Wechsler (2008)	Repeat increasingly long strings of numbers backward	Total number of trials correctly recalled
Updating	Running Memory for Letters	after Broadway & Engle (2010)	View sequence of single letters; identify last <i>N</i> digits in order of initial presentation	Total number of letters correctly recalled
Updating	N-Back	after Jaeggi et al. (2010)	View sequence of individual shapes; indicate when current shape matches shape from two trials prior	Correct responses minus responses incorrectly identified as matching
Updating	Keeping Track	after Miyake et al. (2000)	Listen to words falling under four categories; recall most recent word from given category	Total number of words correctly recalled

Table 2
Descriptive Statistics

Task: Condition	<i>N</i>	<i>M</i>	<i>SD</i>	Reliability
Animal Stroop: Congruent	504	953.86	250.38	.83 ^c
Animal Stroop: Neutral	504	955.99	218.01	.81 ^c
Animal Stroop: Incongruent	504	1180.27	322.40	.86 ^c
Animal Stroop: Inhibition Cost	504	229.42	206.26	.86 ^b
Mickey: Congruent	472	419.52	100.04	.93 ^c
Mickey: Neutral	472	444.22	112.84	.82 ^c
Mickey: Incongruent	472	454.26	96.91	.94 ^c
Mickey: Inhibition Cost	472	22.39	44.30	.38 ^c
Stop Signal Reaction Time	422	326.44	82.41	.42 ^c
Trail Making: Numbers	505	1151.50	490.07	.88 ^c
Trail Making: Letters	505	1622.76	1999.89	.83 ^c
Trail Making: Numbers-Letters	505	2514.92	1653.57	.76 ^c
Trail Making: Letters-Numbers	503	3239.71	3476.84	.76 ^c
Trail Making: Switch Cost	505	1316.93	1051.60	.87 ^b
Local Global: Local	496	1089.30	344.03	.84 ^c
Local Global: Global	496	1021.05	386.25	.75 ^c
Local Global: Alternating	496	2473.43	973.49	.80 ^c
Local Global: Switch Cost	495	1432.36	788.49	.67 ^b
Plus-Minus: Addition	490	3223.41	3264.16	.94 ^c
Plus-Minus: Subtraction	491	3690.44	4556.96	.94 ^c
Plus-Minus: Alternating	491	4154.18	4069.63	.94 ^c
Plus-Minus: Switch Cost	491	703.71	1357.53	.69 ^b
Symmetry Span	501	20.17	8.60	.77 ^a
Listening Recall	498	23.83	7.85	.77 ^a
Digit Span Back	505	6.96	1.81	.57 ^a
Running Memory	490	19.13	8.23	.74 ^a
N-Back	497	2.59	8.27	.84 ^c
Keeping Track	494	6.71	2.28	.48 ^a

Note. Statistics based on untransformed data. All reliabilities calculated as Cronbach's alpha.

^a Reliability calculated among trials,

^b reliability calculated among all reaction time differences measured by switch blocks minus non-switch blocks (or inhibit blocks minus non-inhibit blocks) reaction time differences,

^c reliability calculated among blocks.

Table 3
Standardized Factor Loadings, Age Effects, and 95% Confidence Intervals from Hierarchical Factor Models

	1. Four EF domains				2. Three EF domains				3. Three EF domains				4. Two EF domains		5. One EF dimension
	In	Sw	WM	Up	In	Sw	WM/Up	In/Sw	WM	Up	In/Sw	WM/Up	In/Sw	WM/Up	EF
Animal Stroop	.42*** (.28, .56)				.42*** (.28, .57)			.43*** (.33, .53)			.43*** (.33, .53)		.43*** (.33, .53)		.39*** (.30, .48)
Mickey	.30*** (.17, .44)				.30*** (.17, .43)			.26*** (.16, .36)			.26*** (.16, .36)		.26*** (.16, .36)		.22*** (.12, .32)
Stop Signal	.15* (.02, .28)				.15* (.02, .28)			.13* (.02, .25)			.13* (.02, .25)		.13* (.02, .25)		.12* (.01, .23)
Trail Making		.68*** (.59, .76)				.67*** (.59, .76)		.64*** (.56, .72)			.64*** (.56, .72)		.64*** (.56, .72)		.62*** (.56, .69)
Plus-Minus		.32*** (.20, .45)				.33*** (.20, .45)		.33*** (.20, .45)			.33*** (.20, .45)		.33*** (.20, .45)		.30*** (.18, .42)
Local-Global		.60*** (.51, .69)				.60*** (.51, .69)		.59*** (.50, .68)			.59*** (.50, .68)		.59*** (.50, .68)		.54*** (.44, .64)
Symmetry Span			.64*** (.58, .71)				.63*** (.56, .70)		.64*** (.58, .71)			.63*** (.56, .69)	.63*** (.56, .69)		.63*** (.57, .70)
Listening Recall			.76*** (.71, .82)				.76*** (.71, .81)		.76*** (.71, .82)			.76*** (.71, .81)	.76*** (.71, .81)		.75*** (.71, .80)
Digit Span Back			.52*** (.45, .60)				.53*** (.46, .61)		.52*** (.45, .60)			.53*** (.46, .61)	.53*** (.46, .61)		.53*** (.45, .60)
Running Memory				.82*** (.77, .86)			.78*** (.73, .83)			.82*** (.77, .86)			.78*** (.73, .83)		.76*** (.71, .81)
N-Back				.67*** (.59, .74)			.64*** (.57, .71)			.67*** (.59, .74)			.64*** (.57, .71)		.63*** (.55, .70)
Keeping Track				.64*** (.58, .70)			.63*** (.56, .69)			.64*** (.58, .70)			.63*** (.56, .69)		.62*** (.55, .68)
Common EF	.33** (.13, .54)	.61*** (.49, .73)	.75*** (.65, .84)	.78*** (.68, .88)	.46** (.14, .63)	.74*** (.47, 1.01)	.64*** (.40, .87)	.57*** (.46, .68)	.75*** (.64, .86)	.78*** (.68, .88)	.81*** (.65, .97)	.55*** (.45, .65)			
Age Effect	.86*** (.59, 1.13)	.65*** (.56, .74)	.66*** (.56, .75)	.53*** (.43, .63)	.86*** (.59, 1.13)	.65*** (.56, .74)	.60*** (.51, .69)	.71*** (.63, .79)	.66*** (.58, .75)	.53*** (.43, .63)	.71*** (.63, .79)	.60*** (.51, .69)	.64*** (.57, .72)		

Note. EF = executive function, In = Inhibition, Sw = Switching, WM = Working Memory, Up = Updating. Age effects were calculated by regressing the latent EF score onto age. Common EF refers to loadings of the first order In, Sw, WM, and Up factors on the higher order EF factor.

* p < .05,
** p < .01,
*** p < .001.

Fit Indices and Scaled Chi-Square Difference Results for Confirmatory Factor Models of Executive Functions

Table 4

Model	Model Fit Indices					χ^2 difference p			
	χ^2	df	χ^2 p	χ^2 scaling factor	RMSEA (95% CI)	CFI	AIC	vs. Model 1	vs. Model 2 vs. Model 3 vs. Model 4
1. Four factors: In, Sw, WM, Up	62.31	58	.326	1.07	.012 (.00-.03)	.997	15128.45		
2. Three factors: In, Sw, WM-Up	82.19	60	.030	1.06	.027 (.02-.04)	.984	15144.89	2.05e-6	
3. Three factors: In-Sw, WM, Up	76.86	60	.070	1.06	.024 (.00-.04)	.988	15139.49	1.77e-4	N/A
4. Two factors: In-Sw, WM-Up	97.21	63	.004	1.06	.033 (.03-.05)	.976	15155.35	8.48e-7	2.93e-3 2.08e-4
5. One factor: Common EF	127.62	65	<.001	1.07	.044 (.03-.06)	.956	15184.86	3.45e-9	1.48e-7 1.06e-8 4.32e-6

Note. EF = executive function, In = Inhibition, Sw = Switching, WM = Working Memory, Up = Updating; χ^2 = chi-square; RMSEA = Root Mean Square Error of Approximation; CFI = Comparative Fit Index; AIC = Akaike Information Criterion.

Table 5
Standardized Factor Loadings and 95% Confidence Intervals of Multivariate ACE, AE, and ADE Analyses

Shared environmental effects included			Shared environmental effects omitted			Shared environmental effects omitted, dominance genetic effects included		
A	C	E	A	E		A	D	E
<i>Genetic and Environmental Contributions to the Higher Order EF Factor</i>								
Common EF	1.00*** (1.00, 1.00)	.00 (.00, .00)	.00 (-.05, .05)	1.00*** (1.00, 1.00)	.00 (-.01, .01)	.88*** (.52, 1.23)	.48 (-.21, 1.17)	.08 (-.61, .77)
<i>Genetic and Environmental Contributions Unique to Individual EF Domains</i>								
Inhibition	.00 (.00, .00)	.00 (.00, .00)	.75 (-.12, 1.61)	.00 (.00, .00)	.70 (-.46, 1.87)	.00 (.00, .00)	.00 (.00, .00)	.60 (-1.49, 2.70)
Switching	.59*** (.29, .89)	.00 (.00, .00)	.15 (-1.09, 1.39)	.59*** (.29, .89)	.15 (-1.08, 1.38)	.00 (.00, .00)	.62*** (.43, .81)	.00 (.00, .00)
Working Memory	.00 (.00, .00)	.00 (.00, .00)	.38** (.14, .62)	.00 (.00, .00)	.37** (.12, .61)	.00 (.00, .00)	.00 (.00, .00)	.36** (.09, .62)
Updating	.00 (.00, .00)	.00 (.00, .00)	.24* (.03, .46)	.00 (.00, .00)	.24* (.03, .46)	.00 (.00, .00)	.00 (.00, .00)	.26* (.05, .47)
<i>Genetic and Environmental Contributions Unique to Individual Tasks</i>								
Animal Stroop	.47*** (.25, .68)	.00 (.00, .00)	.78*** (.65, .92)	.47*** (.25, .68)	.79*** (.65, .92)	.00 (.00, .00)	.57*** (.35, .79)	.72*** (.55, .89)
Mickey	.00 (.00, .00)	.26 (-.04, .56)	.92*** (.81, 1.02)	.26 (-.15, .68)	.92*** (.79, 1.05)	.26 (-.16, .68)	.00 (.00, .00)	.92*** (.79, 1.05)
Stop Signal	.00 (.00, .00)	.28* (.04, .51)	.95*** (.88, 1.02)	.19 (-.25, .62)	.97*** (.88, 1.06)	.18 (-.26, .63)	.00 (.00, .00)	.97*** (.89, 1.06)
Trail Making	.33*** (.14, .52)	.00 (.00, .00)	.63*** (.52, .74)	.35*** (.14, .52)	.63*** (.52, .74)	.00 (.00, .00)	.38*** (.21, .55)	.60*** (.51, .70)
Plus-Minus	.00 (.00, .00)	.00 (.00, .00)	.95*** (.91, 1.00)	.00 (.00, .00)	.95*** (.91, 1.00)	.00 (.00, .00)	.32 (-.47, 1.11)	.90*** (.62, 1.18)
Local-Global	.26* (.02, .51)	.00 (.00, .00)	.76*** (.67, .85)	.26* (.02, .51)	.76*** (.67, .85)	.00 (.00, .00)	.34* (.04, .63)	.73*** (.60, .86)
Symmetry Span	.38* (.01, .76)	.15 (-.58, .87)	.63*** (.53, .73)	.42*** (.29, .54)	.62*** (.54, .71)	.41*** (.28, .54)	.00 (.00, .00)	.63*** (.54, .71)
Listening Recall	.00 (.00, .00)	.00 (.00, .00)	.64*** (.57, .70)	.00 (.00, .00)	.64*** (.57, .70)	.00 (.00, .00)	.00 (.00, .00)	.64*** (.57, .71)
Digit Span Back	.43*** (.28, .59)	.00 (.00, .00)	.72*** (.64, .81)	.43*** (.28, .59)	.72*** (.64, .81)	.33 (-.32, .98)	.31 (-.48, 1.09)	.72*** (.61, .82)
Running Memory	.17 (-.12, .46)	.00 (.00, .00)	.54*** (.45, .64)	.17 (-.12, .46)	.54*** (.45, .64)	.00 (.00, .00)	.28** (.07, .49)	.51*** (.40, .61)
N-Back	.44*** (.30, .57)	.00 (.00, .00)	.62*** (.52, .71)	.44*** (.30, .57)	.62*** (.52, .71)	.34 (-.18, .86)	.32 (-.34, .97)	.60*** (.48, .71)
Keeping Track	.30** (.10, .50)	.00 (.00, .00)	.69*** (.60, .78)	.30** (.10, .50)	.69*** (.60, .78)	.00 (.00, .00)	.35** (.12, .53)	.68*** (.57, .78)

Note. EF = executive function, A = additive genetics, C = shared environment, E = non-shared environment, D = dominance genetics. Our model controls for age-effects at the level of the first-order factors (Inhibition, Switching, Working Memory, and Updating). Because the purpose of this analysis was to understand the relative contributions of genetic and environmental influences to individual differences, as separate from age-related differences, the loadings of the first-order factors have been standardized relative to their age-independent variance.

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