



The heritability of self-control: A meta-analysis

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ABSTRACT

Self-control is the ability to control one's impulses when faced with challenges or temptations, and is robustly associated with physiological and psychological well-being. Twin studies show that self-control is heritable, but estimates range between 0% and 90%, making it difficult to draw firm conclusions. The aim of this study was to perform a meta-analysis to provide a quantitative overview of the heritability of self-control. A systematic search resulted in 31 included studies, 17 reporting on individual samples, based on a sample size of > 30,000 twins, published between 1997 and 2018. Our results revealed an overall monozygotic twin correlation of 0.58, and an overall dizygotic twin correlation of 0.28, resulting in a heritability estimate of 60%. The heritability of self-control did not vary across gender or age. The heritability did differ across informants, with stronger heritability estimates based on parent report versus self-report or observations. This finding provides evidence that when aiming to understand individual differences in self-control, one should take genetic factors into account. Recommendations for future research are discussed.

One factor that contributes to good adjustment across the lifespan is self-control. Yet, not all individuals develop the same levels of self-control, which begs the question: 'where do these individual differences come from?'. While the effects of the environment on such individual differences are well documented (Bridgett et al., 2015; Pallini et al., 2018; Willems et al., 2018b), the research on genetic influences on self-control is more nascent. The aim of this study is therefore to perform a meta-analysis to provide a quantitative overview of the heritability of self-control.

Self-control is defined as the capacity to alter unwanted impulses and behaviors in order to bring them into agreement with internal and external standards (Duckworth and Steinberg, 2015; Tangney et al., 2004). Multiple studies across disciplines emphasize the importance of self-control. On the one hand, individuals with high self-control are happier, healthier, and wealthier across adolescence and adulthood, compared to those with low self-control (de Ridder et al., 2012; Duckworth and Seligman, 2005; Finkenauer et al., 2005; Hofmann et al., 2014). On the other hand, low self-control has been associated with lack of success in school, relationships, and the labor market (Caspi et al., 2016; Moffitt et al., 2011; Vazsonyi et al., 2017). Because self-control is a powerful predictor of health, wealth, and public safety,

numerous studies examined why some individuals have higher self-control than others. Most of these studies focused on environmental effects, examining how parenting or peer involvement explains variation in self-control (Finkenauer et al., 2005; Gottfredson and Hirschi, 1990; Karreman et al., 2006; King et al., 2018; Pallini et al., 2018).

In the last decade, though, various studies have shown that almost all traits and behaviors are at least partly influenced by genetic factors (Polderman et al., 2015). For self-control, results are mixed. For example, some studies state that differences in self-control are not or weakly explained by genetic factors (e.g., Friedman et al., 2011), while others state that almost all variation in self-control is explained by genetic factors (e.g., Beaver et al., 2009; Wright et al., 2008), and again others state that about half of the variance in self-control is explained by genetic factors (e.g., Boisvert et al., 2013c; Willems et al., 2018a; Yamagata et al., 2005). To obtain a clearer picture from previously researched data of the genetic influence on self-control, we performed a meta-analysis including twin studies that address the heritability of self-control. By doing so, we aim to provide an encompassing and quantitative overview on the extent to which genetic factors play a role in explaining individual differences in self-control.

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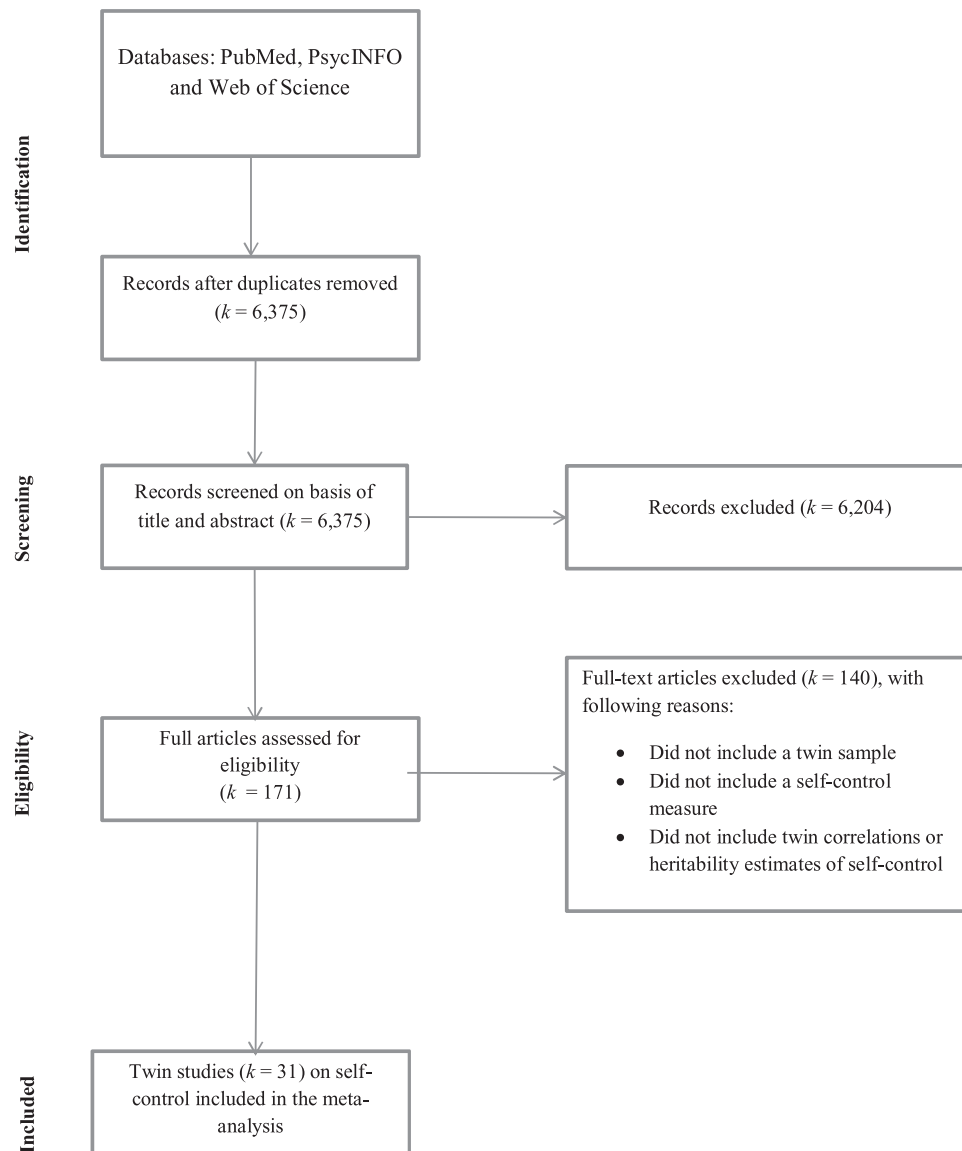


Fig. 1. PRISMA flowchart of selected twin studies.

1. Method

1.1. Twin design

The classical twin design is built on the premise that differences in the resemblance between monozygotic twins (sharing approximately 100% of their DNA) and dizygotic twins (sharing 50% of their segregating genes on average) can be used to parse phenotypic trait variance into genetic and environmental components. Genetic influences are implied if the correlation between monozygotic twin (MZ) pairs is higher than the correlation between dizygotic twin (DZ) pairs. An influence of the common environment – influences that are shared between family member– is implied when the DZ twin pair correlation is higher than half of the correlation between MZ twin pairs. Unique environmental factors are person specific and not shared between twins. Identical twin correlation's deviation from 1 provide a direct estimate for the non-shared environmental influences, since identical twins share both their genetic make-up as well as part of the environment (the shared environment).

More specifically, twin correlations can potentially be parsed into additive genetic (A), non-additive or dominance genetic (D), common environment (C), and non-shared environment (E). If MZ correlations

are larger than DZ correlations, A, C, and E effects are to be expected. If MZ correlations are more than twice the DZ correlations non-additive genetic effects are expected. In the classical twin design the non-additive genetic influences (D) and shared environmental influences (C) are confounded and cannot be estimated in the same model, and it is common for authors to estimate one or the other based on the twin correlations. Note that this distinction does not influence the results presented in our study as we speak of general genetic influence without specifically modelling the difference between additive or non-additive genetic influences.

Like any statistical model, the classical twin design is based on certain assumptions. One key assumption of twin models is that of the “equal environment”, assuming that the environment of monozygotic twins is no more similar than the environment of dizygotic twins. Critics of twin models state that the equal environment assumption does not hold, because MZ twins receive more similar treatment, and that heritability estimates are therefore not trustworthy (e.g., [Burt and Simons, 2014](#)). However, empirical evidence (systematic reviews, simulation studies and twin studies, for example with twins with misclassified zygosity) shows that this assumption is typically not violated, with heritability estimates garnered in twin models being relatively unbiased ([Barnes et al., 2014](#); [Conley et al., 2013](#)).

1.2. Search of studies

Articles were retrieved from various online databases through a computerized literature search. The databases included *PubMed* (<http://www.ncbi.nlm.nih.gov/pubmed>), *PsycINFO* (<http://www.apa.org/pubs/databases/psycinfo/index.aspx>) and *Web of Science* (<http://apps.whoebknowledge.com>). A literature search was conducted for studies published up to March 28th 2018. The following search terms (and their variations) were used: 1) *twin* OR *heritability* OR *genetics*, as well as 2) *self-control* OR *self-regulation* OR *effortful control* OR *self-discipline*.

1.3. Selection procedure

Studies were eligible for this meta-analysis when the following criteria were met. First, the study had to include twin correlations or standardized heritability estimates. This information is necessary to be able to extract information on the extent to which individual differences in self-control are explained by genetic factors. Second, the study had to assess self-control or a concept closely related to self-control, such as self-regulation, effortful control, self-discipline, or emotion regulation (Nigg, 2017). Third, only papers originally published in English and that were published in peer-reviewed journals were included. Fourth, we excluded papers that assessed individuals with clinical psychological problems (e.g. schizophrenia and autism), as well as papers primarily focusing on clinical physiological disorders (e.g. obesity and diabetes).

The initial search in the databases yielded a total of 6375 unique hits. Titles and abstracts of these hits were examined according to the inclusion criteria, resulting in 160 papers that were selected for in-depth reading. We also inspected possible missing publications by the main authors of the identified papers, resulting in the identification of 11 additional publications. Subsequently, all 171 articles were screened according to the inclusion criteria, resulting in 31 articles to be included in the present meta-analysis (see Fig. 1).

The main reasons for exclusion were that studies mentioned the heritability of self-control but did not contain a twin sample (60%), or did not empirically assess self-control (30%). Additionally, a substantial number of the excluded articles did not provide MZ/DZ correlations or other heritability measures needed to infer the genetic and environmental effects on self-control (10%). References included in this systematic review are preceded by * in the reference list.

1.4. Coding the studies

The first two authors coded all 31 articles, retrieving descriptive information (authors, article title, journal, year of publication), sample information (country, cohort, sample size, age), methodological information (measurement of self-control, informant of the measure), and heritability estimates (MZ correlation, DZ correlation, and standardized heritability estimate of the overall model if provided and otherwise estimates of the best fitting model). For every twin correlation, we coded age (1 = *early childhood*, 0–6 years; 2 = *middle childhood*, 7–12 years; 3 = *adolescence*, 13–18 years; 4 = *adulthood*, 18+ years), and informant (1 = *parent report*, 2 = *self-report*, 3 = *observation*). Some studies provided twin correlations separately for boys and girls. These studies were coded accordingly (1 = *girls* 2 = *boys*). See Table 1 for an overview and description of all the included papers.

1.5. Analyses

The meta-analysis was performed in the Metafor package in R version 3.5.1 (Viechtbauer, 2010; R Core Team, 2013). Our dataset and statistical scripts can be accessed from the [supplements](#), providing opportunities for other scholars to use, update or extend our data for future research purposes. Many studies reported multiple effect sizes, for

example reporting twin correlations for different self-control measures, for different age groups, separately for boys and girls, and separately for parent-report and self-report. Additionally, multiple studies used data from the same cohort, for example multiple studies used the AddHealth data (<http://www.cpc.unc.edu/projects/addhealth>). As a result, it is likely that effect sizes from the same sample are more similar than effect sizes from different studies, as they are part of the same sampling process, study group, and study population. Previous meta-analyses only included one effect size of each included study to deal with this dependency (Bartels, 2015; de Zeeuw et al., 2015). More recently, multi-level meta-analyses are suggested to be more preferable, because they take the dependency between studies into account while including all effect sizes. Thereby it increases statistical power and provides maximum information of your data (Assink and Wibbelink, 2016; Hendriks et al., 2018; Van den Noortgate et al., 2013). Accordingly, we applied a multi-level meta-analysis for the present study taking into account sampling variance (level 1), variance between effect sizes from the same sample (level 2), and variance between studies (level 3) (Van den Noortgate et al., 2013).

The analyses were conducted in multiple stages. First, we decided which effect size to meta-analyze. The included studies provide different parameters that allow to estimate the heritability of self-control, namely (1) MZ and DZ correlations or, (2) standardized genetic variance (cf. the heritability). Multiple studies only presented their best fitting model (dropping non-significant parameters), and report only the variance decomposition based on this best fitting model. This model choice and preference is sensitive to sample size, thereby possibly presenting a biased perspective (often an overestimation) of genetic influences on self-control (Posthuma and Boomsma, 2000). For the present study, we therefore decided to meta-analyze the twin correlations (MZ correlation and DZ correlation, respectively), rather than the standardized genetic variance.

Second, both the MZ correlations (r_{mz}) and the DZ correlations (r_{dz}) were transformed into Fisher's Z scores (ES_{Zmz} and ES_{Zdz} , respectively). This ES_Z scores is assumed to approach normality, which is necessary for the accurate determination of mean effect size estimates and for unbiased tests of statistical significance (Lipsey and Wilson, 2001). Subsequently, we meta-analyzed the ES_{Zmz} and the ES_{Zdz} separately, resulting in an overall ES_{Zmz} and an overall ES_{Zdz} . Dependency between effect-sizes was taken into account by categorizing all effect sizes based on the same sample within the same level, in line with the multi-level meta-analytic approach (Assink and Wibbelink, 2016; Viechtbauer, 2010). So effect sizes based on the same sample received the same 'identification number', to take into account the dependency between these effect sizes, which was used in the multilevel approach. Next, the Fisher's Z scores, ES_{Zmz} and ES_{Zdz} , were transformed back to MZ correlations (r_{mz}) and DZ correlations (r_{dz}), for interpretation purposes (Lipsey and Wilson, 2001)¹. Additionally, we calculated the heritability of self-control by applying Falconer's formula: $h^2 = 2(r_{mz} - r_{dz})$, with r_{mz} being the meta-analytic correlation of self-control among MZ twins and r_{dz} the meta-analytic correlation of self-control among DZ twins (Falconer, 1960). Third, we examined whether the ES_{Zmz} and ES_{Zdz} were potentially moderated by a number of factors such as gender, age, and informant.

2. Results

2.1. Descriptives

A total of 31 papers were included (see Table 1 for an overview). Of

¹ The Fisher's transformation of r was calculated in Excel (FisherZ function) using the following formula: $ES_{Zr} = \frac{1}{2} \log_e \left[\frac{1+r}{1-r} \right]$. For the back transformation, the function FisherInv was applied using the following formula: $r = \frac{e^{2ES_{Zr}} - 1}{e^{2ES_{Zr}} + 1}$ (see Lipsey and Wilson, 2001).

Table 1
Overview of the included heritability studies.

Author(s)	Country	Cohort	SC term	Measure	Age	Informant	Sex	MZ pairs	DZ pairs	rMZ	rDZ	A (95% CI)	C (95% CI)	E (95% CI)	D (95% CI)
Anokhin et al. (2011)	USA	LITGBFB	DD	DoG	12	Lab task	M	82	71	.45	.02	.30 (.15-.49)		.70(.51-.85)	
							F	87	49	.38	.29				
							OS	83	83	.34	.34				
					14	Lab task	M	63	50	.82	.14	.51 (.28-.71)		.49 (.29-.72)	
							F	66	31	.46	-.14				
							OS	56	56	.17	.17				
Beaver (2008)	USA	AH	SC	SoI	15	Self	All	245		.41	.41				
					16	Self	All	190		.32	.32				
Beaver et al. (2008a)	USA	ECLS-K	SC	Solssrs	4	Par/Teach	All	55	95	.72	.36	.72 (.58-.86)		.28 (.14-.42)	
					6	Par/Teach	All			.46	.23	.46 (.20-.73)		.54 (.27-.80)	
Beaver et al. (2008b)*	USA	AH	SC	SoI	15	Self	All	289	452	.64	.32	.64	0	.36	
					16	Self	All			.53	.27	.53	0	.47	
Beaver et al. (2009)	USA	AH	SC	SoI	15	Self	All	300	400	.41	.12				
					16	Self	All			.32	.12				
Beaver et al. (2013)	USA	CNLSY	SC	BPI	9	Par	All	13	1664	.94	.49	.90 (.79-1.00)		.06 (.02-.08)	
					11	Par	All			.82	.56	.53 (.35-.71)		.18 (.04-.26)	
					13	Par	All			.88	.53	.71 (.47-.94)		.12 (.00-.23)	
Boisvert et al. (2012a,b)	USA	AH	SC	SoI	15	Self	All	307	477	.45	.24	.42 (.19-.52)		.55 (.48-.65)	
					16	Self	All			.35	.14	.43 (.19-.52)		.55 (.47-.65)	
					18.5	Self	All			.50	.11	.43 (.18-.52)		.56 (.48-.65)	
Boisvert et al. (2013a)	USA	AH	SC	SoI	15	Self	All	289	452	.39	.23	.30 (.04-.45)		.62 (.52-.73)	
					16	Self	All			.44	.16	.40 (.20-.49)		.60 (.51-.70)	
					22	Self	All			.42	.12	.35 (.15-.46)		.64 (.54-.75)	
					29	Self	All			.39	.29	.37 (.07-.47)		.62 (.53-.74)	
Boisvert et al. (2013b)	USA	AH	SC	SoI	15	Self	M	155	124	.33	.29	.09 (.00-.46)		.69 (.54-.82)	
					16	Self	F	149	118	.49	.30	.44 (.01-.63)		.48 (.36-.64)	
						Self	M			.53	.23	.47 (.01-.59)		.52 (.41-.67)	
						Self	F			.50	.15	.47 (.13-.59)		.53 (.41-.68)	
					22	Self	M			.43	.18	.61 (.48-.78)		.59 (.45-.76)	
						Self	F			.42	.17	.39 (.00-.52)		.61 (.48-.78)	
Boutwell et al. (2013)	USA	AH	SC	SoI		Self	All	289	450	.38	.22	.38 (.13-.49)		.60 (.51-.71)	
Boutwell and Boisvert (2014)	USA	AH	SC	SoI	22	Self	All	285	247	.40	.17	.37 (.04-.48)		.62 (.52-.74)	
						Self	M	144	131	.41	.13	.38 (.00-.52)		.62 (.48-.79)	
						Self	F	145	116	.35	.18	.27 (.00-.47)		.66 (.52-.83)	
Coyne and Wright (2014)	USA	ECLS-K	SC	Solssrs	5.72	Par/Teach	All	59	121	.73	.56	.35 (.08-.62)		.27 (.18-.36)	
					7.26	Par/Teach	All			.64	.42	.44 (.01-.87)		.36 (.23-.49)	
					9.27	Par/Teach	All			.56	.32	.49 (.01-.97)		.44 (.24-.63)	
					11.26	Par/Teach	All			.67	.34	.67 (.53-.81)		.33 (.19-.48)	
Deater-Deckard et al. (2007)	USA	WRRP	EC	BBR	6.09	Obs	All	105	154	.51	.30	.28 (-.06-.56)		.50 (.39-.62)	
Fagnani et al. (2017)	IT	ITS	SC	SoIA	16.3	Par/Self	All	281	493	.59	.29	.58 (.47-.67)		.42 (.33-.53)	
Friedman et al. (2011)*	USA	CLTS	SRS	Stroop/stop signal	1.2	Obs	All	435	378	.50	.33	.36	.15	.49	
					1.7	Obs	All			.36	.33	.36	.15	.49	
					2	Obs	All			.23	.29	.00	.26	.74	
					3	Obs	All			.53	.33	.40	.13	.47	
Gagne and Saudino (2010)	USA	BUTP	IC	Lab-TAB	2.07	Obs	All	133	161	.38	.16	.38 (.06-.51)		.62 (.49-.77)	
Gagne and Hill Goldsmith (2011)	USA	WSBR	IC	Lab-TAB	3	Obs	All	173	338	.84	.55	.58 (.39-.82)		.16 (.12-.21)	
Gagne et al. (2011)	USA	BUTP	IC	CBQ	2.07	Obs	All	130	237	.67	.21	.39 (.25-.51)		.63 (.51-.72)	
						Obs	All	131	160	.41	.15	.39 (.25-.51)		.61 (.49-.75)	
Gagne and Saudino (2016)	USA	BUTP	IC	TBAQ-R	2	Par	All	145	168	.85	.55	.67 (.51-.85)		.17 (.13-.22)	
						Par	All			.87	.55	.61 (.78-.87)		.17 (.13-.22)	
						Obs	All			.38	.12	.38 (.14-.51)		.62 (.49-.76)	
					3	Par	All			.73	.42	.23 (.08-.45)		.01 (.00-.04)	
						Obs	All			.26	.35	.06 (.00-.25)		.01 (-.04-.00)	

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Table 1 (continued)

Author(s)	Country	Cohort	SC term	Measure	Age	Informant	Sex	MZ pairs	DZ pairs	rMZ	rDZ	A (95% CI)	C (95% CI)	E (95% CI)	D (95% CI)	
Gagne et al. (2017)	USA	WTP	AC	EATQ; HBQ	13.6	Par/Self	All	188	258	.59	.15	.73		.27		
Goldsmith et al. (1997)	USA		PER	TBAQ	2.21	Par	F	89	95	.61	.13					
			EC	CBQ	5.54	Par	All	55	64	.81	.33					
Guo et al. (2011)	CN		EC	EATQ-R	13.74	Self	All	585		.53	.24					
Gustavson et al. (2014)	USA	CLTS	IM	UPPS-P		Par	All	181	166	.71	-.03					
			SC	UPPS-P Self		Self	All			.47	.13					
			PR	GPS Self						.56	.07					
			VO	VCI Self						.39	.10					
Hahn et al. (2017)	DE	GTSIO	ACT	ACS Self			All	146	90	.32	.05					
			SD	TCI	33.3	Self	All			.59	.16	.54 (.00-.15)	.00 (.00-.15)	.41 (.32-.53)		
			LoC	GPIUS	33.3	Self	All			.37	.26	.21 (.00-.50)	.16 (.00-.43)	.63 (.50-.77)		
			MR	GPIUS	33.3	Self	All			.41	.28	.33 (.00-.54)	.09 (.00-.42)	.58 (.46-.73)		
			SR	GPIUS	33.3	Self	All			.26	.18	.21 (.00-.41)	.06 (.00-.33)	.73 (.59-.88)		
Lemery-Chalfant et al. (2008)*	USA	WTP	EC	CBQ	7.58	Par	M	214	198	.71	-.11	.00		.32	.68	
						F			.65	.27	.00		.32	.68		
Li et al. (2014)	CN	BTS	EC	CBQ	7.88	Par	OS		151		.04					
						M			.67	.03	.41		.21	.38		
						F				.75	.32					
						OS				.04		.83		.17	.00	
Wang and Saudino (2013) Willems et al. (2018a)	USA NL	BUTP NTR	ER	BSID II	15.47	Self	M	279	97	.58	.38					
			SC	AASCS	2.99	Par	M			.30	.45					
					7	Par	F			.59	.22					
						Par	F			.43	.25					
						Obs	M			.53	.27					
						Mot	M			.74	.34					
							F			.70	.32					
						Fa	OS			.31						
						Teach	M			.75	.39					
							F			.73	.36					
				OS			.75	.32								
				M			.68	.54								
				F			.56	.37								
				OS			.29	.14								
				M			.69	.49								
				F			.48	.37								
				OS			.67	.52								
				M			.70	.40								
				F			.31									
				OS			.65	.55								
				M			.35	.10								
				F			.57	.40								
				OS			.74	.66								
				M			.78	.41								
				F			.73	.40								
				OS			.35									
				M			.69	.65								
				F			.67	.35								
				OS			.31									
				M			.32	.16								
				F			.31	.27								
				OS			.26	.24								
				M			.26	.24								
				F			.22	.20								
				OS			.27	.26								
				M			.31	.27								
				F			.00	.00								
				OS			.00	.00								
				M			.69	.65								
				F			.34	.23								
				OS			.35	.07								
				M			.17	.00								
				F			.01	.00								
				OS			.00	.00								
				M			.31	.27								
				F			.00	.00								

(continued on next page)

Table 1 (continued)

Author(s)	Country	Cohort	SC term	Measure	Age	Informant	Sex	MZ pairs	DZ pairs	rMZ	rDZ	A (95% CI)	C (95% CI)	E (95% CI)	D (95% CI)	
Wright et al. (2008) *	USA	AH	SC	Sol	15	Self	All	798	560	.63	.31	.39 (.09 -.70)	.00	.34 (.31 -.36)	.27 (-.03 -.58)	
								OS	1135	.27						
								M	172	.57	.32	.39 (.23 -.54)			.43 (-.05 -.91)	.18 (-.18 -.54)
									197	.40	.32	.26 (.04 -.49)			.58 (.03-1.00)	.16 (-.18 -.49)
								OS	182	.03						
									M	739	.44	.19	.38 (.18 -.58)			.57 (.55 -.60)
Yamagata et al. (2005) *	USA	KTP	EC	EC scale	16	Self	All	1103	837	.52	.21	.32 (.06 -.58)		.47 (.43 -.52)	.21 (-.06 -.47)	
								OS	1661	.16						
								M	565	.45	.23	.40 (.04 -.76)			.50 (.47 -.53)	.10 (-.26 -.46)
									868	.44	.15	.20 (.00 -.41)			.52 (.47 -.58)	.28 (.05 -.50)
								OS	1223	.20						
									All	289	.44	.34	.40			.60
Yancey et al. (2013)	USA	UMTR	TI	ESI	29.4	Self	All	152	73	.45	.21	.49		.51		
								EC	.18	.07	.25	.01	.74			
								ACC	.38	.17	.39		.61			
								ATC	.42	.20	.45		.55			
								IC	.30	.12	.32		.68			
								TI	.60	.24	.59 (.46-.69)	.00	.41 (.31-.54)			

Abbreviations: **Countries:** CN – China, DE – Germany, IT – Italy, JP – Japan, NL – the Netherlands, USA – United States of America; **Cohorts:** AH – Add Health, BPI – the Behavioral Problems Index, BUJP – Boston University Twin Project, BTS – Beijing Twins Study, CNLSY – the Child and Young Adult Supplement of the National Longitudinal Survey of Youth 1979, CLTS – Colorado Longitudinal Twin Study, ECLS-K – the Early Childhood Longitudinal Survey, Kindergarten Class, GTSIO – German Twin Study on Internet and Online-game behavior, ITS – Italia Twin Register, KTP – Keio Twin Project, LTGBFB – Longitudinal Twin Study of the Genetics of Brain function and Behavior, NTR – the Netherlands Twin Register, WRRP – Western Reserve Reading Project, WSBR – Wisconsin State Birth Records, WTP – Wisconsin Twin Project, UMTR – University of Minnesota Twin Registry; **Self-control Term:** AC – Attentional Control, ACT – Action Control, DD – Delay Discounting, EC – Effortful-Control, ER – Emotion Regulation, IC – Inhibitory Control, IM – Impulsivity, LC – Locus of control, PR – procrastination, PER – Persistence, SC – Self-Control; SR – Self-Regulation, SRS – Self-Restraint, SD – Self-Directedness, TCI – Temperament and Character Inventory, Temp – Temperament, VO – Volitional behavior; **Measure:** ACS – Action Control Scale, ASCS – ASEBA Self-Control Scale, BBR – Bayley’s Behavior Records, BRS – Bayley’s Behavior Records, CBQ – Child Behavior Questionnaire, BSID-II – the Bayley Scales of Infant Development-Second Edition, DoG – Delay of Gratification procedure, EATQ(-R) – Early Adolescent Temperament Questionnaire (Revised), EC Scale – Effortful Control Scale, ESI – Externalizing Spectrum Inventory, GPS – General Procrastination Scale, GPIUS – Generalized Problematic Internet Use Scale, HBQ – the MacArthur Health and Behavior Questionnaire Lab-TAB – Laboratory Temperament Assessment Battery, Sol – Sum of Items (Add Health), SoIA – Sum of Items Achenbach scales, Solssrs – Sum of Items Social Skills Rating Scale, TBAQ(-R) – the Toddler Behavior Assessment Questionnaire (Revisited), VCI – Volitional Components Inventory; **Informant:** fa – father report, mot – mother report, par – parent-report, self – self-report, teach – teacher report; **Sex:** All – Males and Females, F – Females, M – Males, OS – Opposite sex. **Heritability Parameters:** MZ – monozygotic, DZ – Dizygotic, A – additive genetic, C – common environment, E – unique environment, D – dominant genetic influences. **Note:** Articles marked with an * did not include confidence intervals in article.

the 31 papers, 17 papers reported on independent samples. Multiple articles applied data from the Add Health project ($k = 9$), the Boston University Twin Project ($k = 4$), and the Colorado Longitudinal Twin Project ($k = 2$). Most studies were conducted in the United States of America ($k = 25$ twin studies). The other studies ($k = 6$) were based on non-American samples, with two studies from China (a population sample and the Beijing Twin study), one study from Germany (German Twin Study on Internet and Online Game Behavior), one study from Italy (Italia Twin Register), one study from Japan (Keio Twin Project), and one study from the Netherlands (the Netherlands Twin Registry), respectively. The total sample size, only counting sample size of independent studies, was 15,892 MZ individuals and 17,384 DZ individuals, with a total sample size of 33,276.

The earliest published paper was in 1997 (Goldsmith et al., 1997), while the most recent publication was in 2018 (Willems et al., 2018a). The papers were published in 20 different journals. Most studies used self-reports ($ES = 46$), or parent reports ($ES = 29$), and some studies included observations ($ES = 17$). In total, 20 different measures were used, such as the Children's Behavior Questionnaire (Rothbart et al., 2001), and the ASEBA Self-Control scale (Willems et al., 2018a). The smallest sample consisted of 119 twin pairs (Goldsmith et al., 1997), while the largest sample consisted of more than 4000 twin pairs (Willems et al., 2018a). The samples covered a wide age-range, from 1.20 years (Friedman et al., 2011) to 33.30 years (Hahn et al., 2017), with an average age of 13.04 years. Most studies reported on children in middle childhood (7–12 years, $ES = 33$) or adolescence (13–18 years, $ES = 33$), but there were also studies specifically investigating early childhood (0–6 years, $ES = 21$) and adulthood (> 19 years, $ES = 20$).

For the present study, we meta-analyzed the twin correlations (MZ correlation and DZ correlation, respectively), rather than the standardized genetic variance. Of the 31 included studies, 11 studies reported twin correlations of full models with correlations separately for males and females, 14 studies reported correlations for full models constraining correlations to be equal for males and females, 4 studies reported correlations for the best fitting model, and for 2 studies it was unclear whether the correlations were based on full or best fitting models.

2.2. Heritability of self-control

The 31 twin studies provided 108 MZ twin correlations and 104 DZ correlations (two studies only included MZ twins; Beaver, 2008; Guo et al., 2011). The MZ twin correlations ranged between .18 (Wright et al., 2008) and .94 (Beaver et al., 2013). The DZ correlations ranged between -.14 (Anokhin et al., 2011) and .56 (Coyne and Wright, 2014). An examination of the standardized heritability estimates showed that heritability ranged from 0% (Friedman et al., 2011; Gagne et al., 2011) to 90% (Beaver et al., 2013).

This heterogeneity in the heritability estimates is likely a result of the sample size of the studies. A scatterplot of the 31 studies, including the distribution of MZ and DZ correlations across sample sizes respectively, showed that there was less variance between studies with increasing sample size (see Fig. 2). Studies with a small sample size showed more variance in the MZ correlations (with correlations ranging between .28 to .94) than studies with larger sample sizes (with correlations ranging between .51 and .75). A similar pattern was found for the DZ correlations; studies with a small sample size showed more variance in the DZ correlations (with correlations ranging between -.14 to .54) than studies with larger sample sizes (with correlations ranging between .31 and .40).

2.3. Meta-analytic estimates

Meta-analyzing all data, applying multi-level analysis to take the dependency between effect sizes into account, resulted in an overall MZ correlation of 0.58 ($ES_{Z_{MZ}} = .67$, S.E. = .05, $t = 13.27$, $p < .001$, 95%

CI = [.57, .77]) and an overall DZ correlation of .28 ($ES_{Z_{DZ}} = .29$, S.E. = .03, $t = 9.48$, $p < .001$, 95% CI = [.23, .34]). Applying Falconer's formula to calculate the heritability based on the meta-analytic MZ and DZ correlations results in an overall heritability of 60%. In other words, 60% of individual differences in self-control were due to genetic differences between people. The MZ correlation was twice as large as the DZ correlation, indicating little to no evidence for shared environmental effects. Rather, these results suggest that environmental effects on self-control, that explain 40% of the variance, are unique to individuals. This is in line with the standardized variance estimates reported by the studies, where 76% of the studies reported no or very little influence of the shared environment on the variance in self-control.

Next, we assessed whether the magnitude of the MZ or DZ correlation was moderated by study variables such as gender, age, and informant (see Table 2). Eleven papers tested for heritability differences in self-control between males and females, but none found significant differences between gender. This is confirmed by our moderator analyses, where gender did neither moderate the MZ correlations ($F(1, 46) = .49$, $p = .49$) nor the DZ correlations ($F(1, 46) = .02$, $p = .90$). This indicates that both the MZ and the DZ correlations are similar for males and females, indicating no differences in the heritability of self-control for males and females. Important to note is that twin analyses examine differences in the variance, not differences in the mean. It could well be that males and females differ in their average self-control performance (see for example Duckworth et al., 2015), however, the relative contributions of genetic and environmental influences are equal across gender.

Initially, age did moderate both the MZ and DZ correlations, with higher MZ and higher DZ correlations in middle childhood compared to the other age groups. Informant also significantly moderated the twin correlations, with significantly higher MZ correlations for parent-report compared to self-report and observations. However, taking a closer look at the data illustrated that age and informant were not independent: in early and middle childhood, most assessments were based on parent-reports while assessments in adolescence and adulthood were mostly based on self-reports. Accordingly, we applied multiple-moderator models including both age and informant in the same model to take this dependency into account (see Table 3).

This multivariate analysis indicated that age did not significantly moderate the MZ correlation ($F(3, 85) = 1.79$, $p = .15$) nor the DZ correlation ($F(3, 81) = 1.70$, $p = .17$). Informant remained a significant moderator for the MZ correlations ($F(2, 85) = 17.00$, $p < .001$), but not for the DZ correlations ($F(2, 81) = 2.10$, $p = .13$). This indicates that differences in twin correlations were driven by differences in informants, rather than differences in age. More specifically, the MZ correlations were significantly higher when assessed by parent reports than self-reports and/or observations. The DZ correlations were similar across informants. MZ and DZ correlations did not significantly differ across self-reports and observations. Translating this to standardized heritability estimates using Falconer's formula, the heritability of self-control was significantly higher when assessed by parent-report (75%) as compared to self-report (53%) or observations (41%).

3. Discussion

In the present meta-analysis, we synthesized research on the heritability of self-control from different behavior genetic studies. The analyses included monozygotic and dizygotic twin correlations of 31 twin studies, reporting a total of 108 correlations, covering an age range from 1.20 to 33.30 years, with a total sample size of more than 30,000 individual twins. The results of the meta-analysis of the twin correlations indicate an overall monozygotic twin pair correlation of .58 and an overall dizygotic twin pair correlation of .28, resulting in a heritability of 60% for self-control. Thus, 60% of the variation in self-control is due to genetic variation between individuals in the

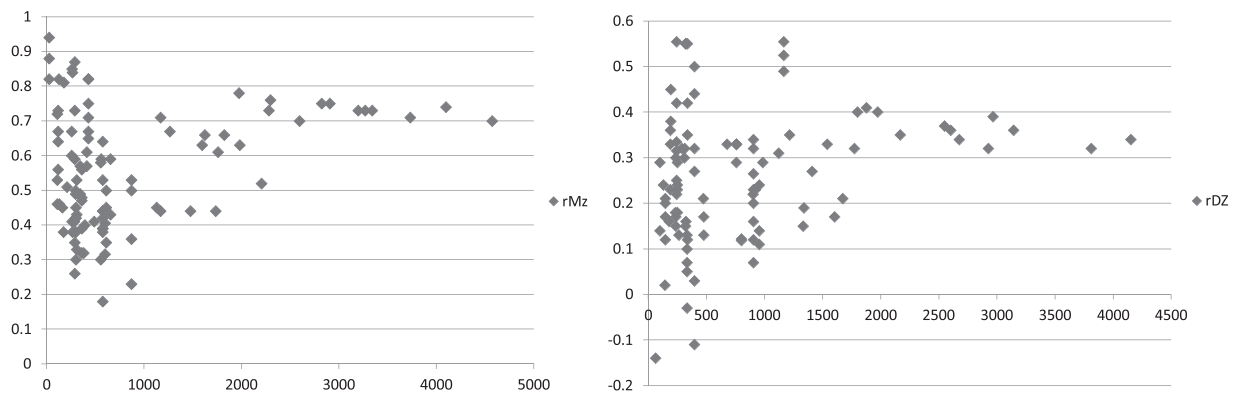


Fig. 2. MZ correlations (on the left, rMZ) and DZ correlations (on the right, rDZ) per sample size.

Table 2
Results for the univariate moderator analyses.

Moderator	Categories	MZ		DZ			
		ES _{Zmz}	95% CI	rMZ	ES _{Zdz}	95% CI	rDZ
Gender	Female	.65	[.47, .83]	.57	.25	[.16, .32]	.24
	Male	.69	[.51, .86]	.60	.25	[.16, .33]	.25
Age	Early childhood	.68	[.56, .79]	.59	.37	[.29, .46]	.36
	Middle childhood	.85	[.74, .95]	.69	.36	[.28, .44]	.34
	Adolescence	.58	[.48, .67]	.52	.23	[.16, .31]	.23
Informant	Adulthood	.51	[.40, .62]	.47	.17	[.09, .25]	.17
	Parent report	.91	[.83, .99]	.72	.36	[.29, .43]	.35
	Self-report	.48	[.41, .55]	.45	.18	[.11, .25]	.18
	Observation	.57	[.47, .67]	.52	.32	[.24, .40]	.31

Note: ES_Z = Fisher's Z score, MZ = monozygotic twins, DZ = dizygotic twins, and CI = confidence interval.

Table 3
Results for the multiple moderator analyses.

Moderator variables	ES _Z (SE)	95% CI	t-Statistic	p-Value
<i>MZ correlations</i>				
Intercept (ref.)	.86 (.06)	[.74, .99]	14.15	< .01
Slope Middle childhood	.10 (.07)	[-.04, .23]	1.50	.14
Slope Adolescence	-.05 (.09)	[-.22, .13]	-0.51	.61
Slope Adulthood	-.08 (.10)	[-.28, .13]	-0.81	.42
Slope Self-report	-.34 (.08)	[-.50, -.19]	-4.40	< .01
Slope Observation	-.31 (.07)	[-.44, -.18]	-4.74	< .01
<i>DZ correlations</i>				
Intercept (ref.)	.40 (.06)	[.29, .52]	6.89	< .01
Slope Middle childhood	-.03 (.07)	[-.16, .11]	-0.40	.69
Slope Adolescence	-.10 (.08)	[-.26, .05]	-1.32	.19
Slope Adulthood	-.17 (.08)	[-.33, -.00]	-2.04	.05
Slope Self-report	-.09 (.06)	[-.21, .02]	-1.63	.11
Slope Observation	-.07 (.05)	[-.16, .03]	-1.39	.17

Note: ES_Z = Fisher's Z score, MZ = monozygotic twins, DZ = dizygotic twins, and CI = confidence interval, ref. = reference category is early childhood and parent-report.

population. Overall, this indicates that there is indeed a robust genetic effect on self-control.

Moderator analyses revealed that monozygotic and dizygotic twin correlations did not differ for males and females, indicating no gender differences in the heritability of self-control. This is consistent with earlier research illustrating that gender differences in heritability for a broad range of behavioral, psychiatric, and health related phenotypes are rare (Vink et al., 2012). In the present study, age did not moderate the heritability estimates of self-control, and we found influences of the unique environment rather than influences of the shared environment. The absence of common environmental influences may be specific to

self-control. This is in line with traits closely related to self-control, such as ADHD and persistence, where research finds no influence of the shared environment across the lifespan (Chang et al., 2013; Kan et al., 2014; Keller et al., 2005). However, the absence of the common environment could also be a result of confounding informant effects. Most studies in childhood are limited to parent reports, and more research including multiple informants (i.e. parent-, self- and teacher-report) at the same age is necessary to distill whether the absence of C is specific to self-control or the result of reporter effects (Bartels et al., 2007; Wesseldijk et al., 2017).

The heritability of self-control implies that individual differences between persons in their self-control performance is partly explained by genetic differences between these individuals. This means that an individual with a predisposition for low self-control may struggle with the regulation of thoughts, behaviors, and impulses while an individual with a genetic predisposition for high self-control may excel in dealing with self-control challenges, although both individuals are exposed to the same environment. These findings imply that the environment – peers, parents, teachers – should take into account such innate individual differences in people's self-control capacities (Pandey et al., 2018).

While some researchers advocated for the ban of twin studies (e.g., Burt and Simons, 2014), the present study underscores the importance of a multidisciplinary approach, including a genetic perspective, to comprehensively understand the etiology of self-control. In contrast to the article by Burt and Simons (2014), an increasing line of work emphasizes the importance of genetic sensitivity in the development of self-control (Beaver et al., 2013; Bolger et al., 2018), embracing twin models as complementary models (Barnes et al., 2014). With the present meta-analysis, we demonstrate the potential of twin studies and a genetic perspective to stimulate future research on the etiology of self-control. Incorporating a genetic perspective in addition to an environmental perspective paves the way for a better understanding of the causes and consequences of self-control and provides new opportunities to improve self-control prevention and intervention efforts (Boisvert et al., 2012a,b; Finkenauer et al., 2018; Harold et al., 2017).

3.1. Future studies

While the current study provides evidence for a robust influence of genetic factors, it does not provide specific information about which sets of genes explain individual differences in self-control. Future work may employ Genome Wide Association Studies (GWAS) in order to unravel the specific genes that are linked to self-control. Considering the wide variety of research groups assessing self-control, and the worldwide increasing collection of DNA, applying a GWAS is feasible.

Monozygotic twin correlations are twice as large as the dizygotic twin correlations, suggesting evidence for a role of the unique environment and little influence of the shared environment. The absence

of shared environmental influences does not mean that parents do not play a role in the development of self-control of their children (Ayoub et al., 2018; Engelhardt et al., 2018). Rather, it implies that environmental factors that make individuals dissimilar play a more important role, such as individual perceptions of parenting that make twins dissimilar even though they are raised within the same family (Cecil et al., 2012; Hannigan et al., 2016). However, more research is needed in order to specifically distill what unique environmental effects create individual differences in children growing up in the same family (Plomin and Daniels, 2011).

Genetic and environmental influences are not mutually exclusive or additive per se, and future research is recommended to investigate how gene-environment correlations (rGE) and gene-environment interaction (G x E) influence individual differences in self-control (Krueger et al., 2008; Plomin et al., 1977; Saxe and Matzel, 2018). Some recent work suggest the presence of passive rGE, where parents create a home environment that is influenced by their own self-control which, in turn, correlates with the (genetic predisposition of) self-control of their child (Bolger, Meldrum, Barnes., 2018; Bridgett et al., 2015). Additionally, adoption studies suggest evidence for evocative rGE, where children with inherited regulation problems evoke more parental harshness (Fearon et al., 2015; Harold et al., 2017; Klahr et al., 2017).

Distinct from rGE, G x E entails that the genotypes vary in their sensitivity to the environment. In twin studies, genotypic influences are estimated conditional upon environmental exposure: when there is no interaction, the influence of genetic factors should not differ in the different environmental exposures. For example, recent work shows that the heritability of ADHD was the same across socio-economic strata indicating an absence of G x E, yet more research is needed to replicate these findings (Gould et al., 2018). On a molecular level, attempts to unravel G x E effects mostly concern candidate gene studies, yet the reliability of such methods is strongly debated (Dick et al., 2015). However, recently developed methodological techniques to take into account both environmental and genetic factors (e.g., genomic SEM or G x E with polygenic risk scores, Grotzinger et al., 2018; Peyrot et al., 2014), provide interesting avenues for future research on gene-environment interplay.

The age of participants in the included twin studies ranged between 1.2 and 33 years, with most studies reporting on middle childhood (7–12 years) or adolescence (13–18 years). Extending this line of work to middle and late adulthood is an important issue for future work. First, genetic research shows that heritability estimates for traits closely related to self-control (i.e., ADHD and emotion regulation) decreases over the course of adulthood (Kan et al., 2014; Nivard et al., 2015). Future work could explore whether a similar trend appears when investigating the heritability of self-control across the lifespan. Second, recent work in the social sciences illustrates how life events more typical to adulthood, such as marriage, children and loss of a loved one, explain individual differences in self-control (Bleidorn, 2015; Pronk et al., 2019). To expand our knowledge on the etiology of self-control, applying classical twin models and gene-environment interplay models in adulthood, would be of particular interest to gain the necessary insights in the underlying mechanisms explaining individual differences in self-control across the lifespan.

3.2. Limitations

There are several limitations of this study that should be noted. First, a heritability estimate depends on the included sample as it is based on the variance of that specific population (Boomsma et al., 2002). Considering that most of the included studies were conducted in the USA reporting on non-clinical samples, we should be cautious when generalizing our findings to other populations. Further work needs to be done to establish heritability estimates in more diverse populations. Second, a wide variety of measures were used to assess self-control, and it would be interesting to assess heritability separately for each

measure. However, the number of studies using the same measure was low. Considering that parameter estimates are poor when the number of studies is below five, we could not assess the influence of specific measurement on the heritability of self-control. Duckworth and Kern (2011) analyzed the phenotypic correlations between different self-control measures, indicating that self-control is a coherent but multi-dimensional construct. For future research, it would be interesting to investigate the genetic correlations between these measures, allowing to examine to what extent all these measures tap into the same underlying construct.

Third, not all studies reported twin correlations of the full model possibly generating bias in the overall heritability estimates. However, we assume this bias is small because only few studies reported correlations based on best fitting models. Fourth, not modelling gene by environment interaction (G x E) might bias heritability estimates of the included twin studies (Purcell, 2002). More specifically, an interaction between A and C would result in an upward bias of A, while an interaction between A and E would result in an upward bias of E. However, despite the increasing interest in genetic factors explaining self-control, there is little work thus far investigating the contribution of G x E to self-control using twin designs. Further work modelling G x E needs to be done in order to gain insights whether such mechanisms explain individual differences in self-control, and whether they bias its heritability estimates.

3.3. Concluding remark

The current study suggests that genes significantly contribute to individual differences in self-control: the heritability of self-control is 60%. This finding provides further evidence for the importance of considering genetic influences when aiming to understand the underlying mechanisms contributing to the development of self-control across the lifespan.

Conflict of interest

Y. E. Willems, N. Boesen, J. Li, C. Finkenauer & M. Bartels declare they have no conflict of interest.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.neubiorev.2019.02.012>.

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