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Review

Individual differences in adolescent self-control: The role of gene-environment interplay

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Self-control – the ability to alter unwanted impulses and behavior to bring them into agreement with goal-driven responses – is key during adolescence. It helps young people navigate through the myriad challenges they encounter while transitioning into adulthood. We review empirical milestones in our understanding of how individual differences in adolescent self-control exist and develop. We show how the use of molecular genetic measures allows us to move beyond nature versus nurture, and actually investigate how both nature *and* nurture explain individual differences in self-control. By highlighting the role of gene-environment correlations and gene-environment interactions, this paper aims to enthuse fellow researchers, with or without a background in genetics, to apply genetically sensitive designs too.

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Introduction

Adolescence presents a variety of self-control challenges. For instance, adolescents must complete their homework while resisting the lure of social media. They need to adhere to rules set by parents, schools, and society, even as they seek greater autonomy. They might face temptations to experiment with alcohol or cigarettes, despite understanding the risks of addiction. Additionally, they must manage their emotions while navigating uncertainties about their future, including concerns about climate change [1–4]. Self-control helps adolescents navigate such challenges. Self-control is an umbrella construct that contains many facets, such as the ability to regulate thoughts, emotions, and behavior to reach a certain goal [5].

Adolescents differ in their self-control capacities. These individual differences, in turn, have been associated with myriad behavioral and relational outcomes throughout adolescence and beyond. For example, studies investigating between- and within-person differences in self-control across adolescence show that lower self-control is associated with deviance [6], mental health problems [7], educational attainment [8], and problematic social media and phone use [4,9]. The association between self-control and (mental)health extends into middle and later adulthood, with adolescents with lower self-control being less able to cope with the range of later-life health, financial, and social demands of adulthood [10–12].

Across disciplines, researchers focus on identifying factors and mechanisms that explain how individual differences in self-control exist and develop during childhood and adolescence. On the one hand, individual differences in self-control arise through interactions with adolescents' proximal environments such as with their families, peers and teachers at school [13]. Positive parenting strategies (e.g. warmth, autonomy, consistent discipline) help adolescents internalize social rules and develop self-control. In contrast, negative parenting strategies (e.g. inconsistent discipline, conflict, negative control) deprive youths of the opportunity to develop self-control independently and may create stressful environments that hinder their ability to self-regulate. A meta-analysis of 191 studies showed that a wide array of parenting dimensions are associated with adolescents' self-control [see Table 1 in Ref. 13]. Chen and colleagues applied cross-lagged models to longitudinal meta-analytic data and found that parenting is longitudinally associated with adolescent self-control, and adolescent self-control is also longitudinally associated with parenting, showing that adolescents are active agents in their own self-control development as they also steer the way they are being parented [14].

On the other hand, individual differences in adolescent self-control are explained by their genetic make-up. Recently, a longitudinal study showed that the heritability of self-control increases from 48% in early adolescence to 76% in young adulthood [15]. This indicates that genetic factors increasingly play a role in explaining the individual differences in adolescent's self-control.

Individual differences in self-control arise from the intricate interplay *between* genetic and environmental factors rather than solely attributed to one *or* the other. Failure to account for gene-environment interplay results in misestimation and misinterpretation of any main effect, whether conceptualized as "genetic" or "environmental" [16]. Until recently, studying gene-environment interplay was limited to family studies. While these designs are powerful, they require access to specific data (e.g., twin studies). Rapid innovations in molecular genetic techniques and the ever-decreasing cost of genotyping are enabling various social scientists interested in self-control (e.g., psychologists, economists, criminologists) to study gene-environment interplay by incorporating molecular genetic measures into their studies [17].

As a result of these technical developments, we are at a new crossroads in science, moving beyond the "nature *versus* nurture" debate by integrating genetic data into social sciences. In this paper, we therefore focus on gene-environment interplay perspectives as they allow us to investigate how "nature *and* nurture" influence individual differences in behavior, transcending many developmental models (e.g., ecological systems theory, general theory of crime, social learning theory [5,16]). In Box 1 we provide an overview of key terms used in this paper.

Molecular genetic studies of self-control

While twin studies offer insights into estimates of genetic contributions to phenotypic variance (heritability), molecular studies examine the specific genetic variants associated with an outcome of interest. Genome-wide association studies (GWAS) have become the preferred method for scrutinizing the genetic landscape because they look at associations between a multitude of genes and outcomes across the genome, considering the complexity of the trait [17].

Recently, numerous GWAS have been published for traits, behaviors, or abilities that underlie self-control capacities such as self-regulation, impulsive personality, attention-deficit/hyperactivity disorder (ADHD), risk-taking, delay discounting, non-cognitive skills, and executive functioning [18–24]. The nomenclature and interest in specific self-control capacities vary across disciplines, which is also reflected in the inclusion of different or overlapping traits in GWAS. Despite these variations, there is genetic overlap among divergent measures, suggesting that albeit conceptually distinct, genetic scores based on GWASs of self-control traits are broadly applicable across different conceptualizations and measures of self-control [25–27].

These GWAS can in turn be used to create polygenic scores which summarize the association across a multitude of genetic variants into a single genetic score for each individual. This score is not something innate or unchangeable, it rather says something about someone's genetic propensity for a certain trait. The strong push for open access data within genomic research and initiatives such as the Polygenic Index Repository have made these polygenic scores available in a wide range of publicly available datasets [28]. Accordingly, more and more social scientists are investigating gene-environment interplay in adolescence including such polygenic scores. This particularly opens up broad opportunities to understand adolescent self-control, as adolescence is a crucial period where self-control differences become increasingly apparent and potentially have a lasting impact on later life.

Gene–environment interplay: gene–environment correlation

One way to understand the interplay between genes and environment is through gene-environment correlations (rGE; often categorized as passive, evocative and active gene-environment correlation). Passive gene-environment correlation occurs when parental genotype correlates with the genotype of their child and the environment they create. For example, parental genetic propensity for self-control partly explains the way they parent (path b, Figure 1) *and* individual differences in their adolescent self-control because of the genetic propensity they transmit to their children (path a, Figure 1). As such, the observed association between certain parenting styles and self-control (path d, Figure 1) during adolescence is not 'purely environmental', as it is influenced by the genetic make-up of parents which influences their environment and their children's genetic make-up. Via this 'double (dis) advantage' the genotype and environment are correlated [29].

Agnew-Blais and colleagues found evidence for such passive-gene environment correlations in a longitudinal adolescent cohort including polygenic scores [30]. They found that mothers' polygenic score for ADHD was

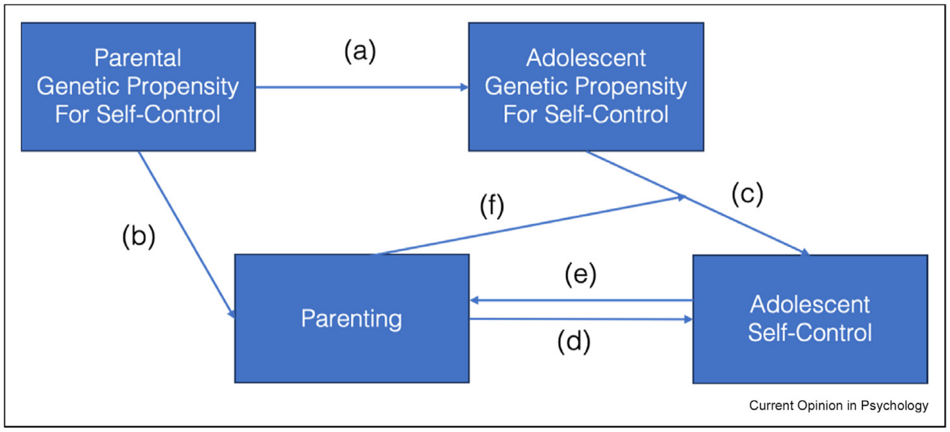
Box 1. Overview of some of the key terms used in this paper

Concept	Explanation
Gene-environment correlation	Gene-environment correlation occurs when an individual's genetic predispositions is correlated to certain environments. This is often categorized as: *Passive gene-environment correlation* occurs when a child's environment is influenced by their parents' own genetic characteristics, which the child also inherits. Since the parents provide both the genes and the environment, the child passively receives environments that are correlated with their genetic predispositions. For example, parents who are genetically disposed to low self-control may create a more chaotic household. The child inherits the genes that predispose them to have low self-control, and they also grow up in an environment that hampers their self-control development, leading to a correlation between their genetic disposition and their environment. *Evocative gene-environment correlation* occurs when an individual's genetic disposed trait elicits specific responses or reactions from their environment. For example, an adolescent with a genetic predisposition for low self-control might evoke more family conflict, which can further influence their self-control development. *Active gene-environment correlation* occurs when individuals seek out or create environments that match their genetic predispositions. For example, an adolescent with a genetic predisposition for low self-control might actively choose to engage with people who have the tendency for risk taking behavior, thereby shaping their own environment in a way that reinforces their self-control abilities.
Gene-environment interaction	Gene-environment interaction refers to the phenomenon where the effect of a person's genetic make-up on a trait is influenced by their environmental context. In other words, the impact of genetic factors on a trait can vary depending on environmental conditions, and the influence of the environment can depend on an individual's genetic predisposition.
GWAS	A GWAS, or Genome-Wide Association Study, is a research method used to find genetic variants linked to specific traits (such as self-control). Researchers compare genetic differences between people with and without the trait or condition, aiming to find variations like single nucleotide polymorphisms (SNPs) related to this trait. GWAS have become the preferred method for scrutinizing the genetic landscape, because they look at associations between genes and outcome across the genome, taking into account the complexity of the trait (cf. a multitude of genes associated with a trait rather than a specific number of genes in candidate gene studies).
Genotype	A genotype refers to the genetic makeup of a person.
Phenotype	A phenotype is a trait or characteristic, like self-control. It's not something that comes directly from your genes or from your environment alone. Instead, it results from how your genes and environment interact with each other.
Polygenic score	A polygenic score summarizes the association across a multitude of genetic variants with a trait based on a GWAS into a single genetic score for each individual.

significantly associated with more chaos in the household (see path b, Figure 1), which in turn was associated with more self-control problems in their adolescents (path d, Figure 1). This suggests that the environment associated with adolescent self-control problems (household chaos) is partly influenced by parents' own genetic propensity for lower self-control, which they also transmit to their children because they share half of their genes (path a, Figure 1). The association between certain parenting styles and adolescent self-control is thereby not necessarily causal, as both can be influenced by parent and children sharing the same genes that influence both the home environment (e.g., household chaos) and behavior (e.g., low self-control). Claims of causal effects of parenting on adolescent outcomes are clouded by failure to account for this genetic transmission [29].

Evocative gene-environment correlation occurs when adolescent genetically predisposed self-control evokes certain environmental influences. For example, Ksinan and colleagues showed that genetic scores for risk taking

Figure 1



Simplified schematic overview of possible pathways of gene-environment correlations (rGE) and gene-environment interactions (G × E).
Passive gene-environment correlation occurs when a parent's genetic propensity is transmitted to their adolescent child (*path **a***) and influences the way they parent (*path **b***) which simultaneously influences the adolescent's actual level of self-control (*path **c*** and ***d***).
Evocative gene-environment correlation occurs when an adolescent's genetic propensity for self-control influences their actual level of self-control (*path **c***) which in turn evokes specific parenting responses (*path **e***).
Gene × Environment interaction occurs when adolescent genetic risk is associated with lower self-control, and this effect is moderated by home environment (e.g., parenting, see *path **f***).
Note: This is a simplified and schematic model which, for illustration purposes, does not include active rGE nor other possible gene-environment interplay mechanisms such as dynastic effects [45] or genetic nurture effects [46].

and impulsivity were associated with more self-control problems in adolescents (path c, Figure 1) which were also associated with less maternal closeness (path e, Figure 1) [31]. Similarly, de la Paz and Agnew-Blais and colleagues showed that polygenic risks for ADHD were associated with lower self-control (path c, Figure 1) and predicted lower levels of parental involvement and higher levels of poor supervision, inconsistent discipline and household chaos across adolescence (path e, Figure 1) [30,32]. Additionally, Kretschmer and colleagues found that polygenic risk for self-regulation problems was associated with adolescent self-control problems (path c, Figure 1), which was associated with family dysfunction (path e, Figure 1) [33]. Together, this suggests that adolescents with a genetic propensity of lower self-control are more likely to elicit certain environments, which in turn could exacerbate individual differences in self-control. For example, an adolescent who is genetically predisposed to show lower self-control is more likely to elicit less parental warmth, which in turn may hamper the possibility to further learn how to regulate impulses, emotions, and thoughts. This illustrates how genetic and environmental factors can buffer or amplify each other's effects, influencing the development of self-control over time [16].

Active gene-environment correlation describes the association between a person's genetically influenced trait and the environment they select. Adolescents with higher genetic scores for impulsivity or risk-taking are potentially more likely to select certain jobs over others (e.g., become entrepreneurs, [34]), choose specific friends or partners, which may shape their levels of impulsivity and risk-taking later in life [35,36], or choose certain life experiences to develop themselves (e.g., going on a world trip or moving to a different city, instead of staying in their hometown). More research is needed to investigate this in genetically sensitive designs, especially for more positive life trajectories as most studies focus on negative outcomes and trajectories.

Gene–environment interplay: gene–environment interaction

Another way to understand the intrinsic interplay between genes and environment is through gene-environment interaction (G x E). This describes the process where certain people with different genotypes vary in their sensitivity to the environment (e.g., differential susceptibility model [37]). It is hypothesized that adolescents with genetic propensity for lower self-control who experienced a high number of environmental risks (e.g., family violence or maltreatment) may be at the highest risk of actually developing lower self-control (see path f in Figure 1, and Figure 2).

Leffa and colleagues found evidence for G x E. They showed that a composite of environmental risks (family socio-economic conditions and prenatal stressors) and polygenic score for ADHD are predictive of lower self-control, respectively [38]. Additionally, they found a significant interaction effect, suggesting that those who

Figure 2

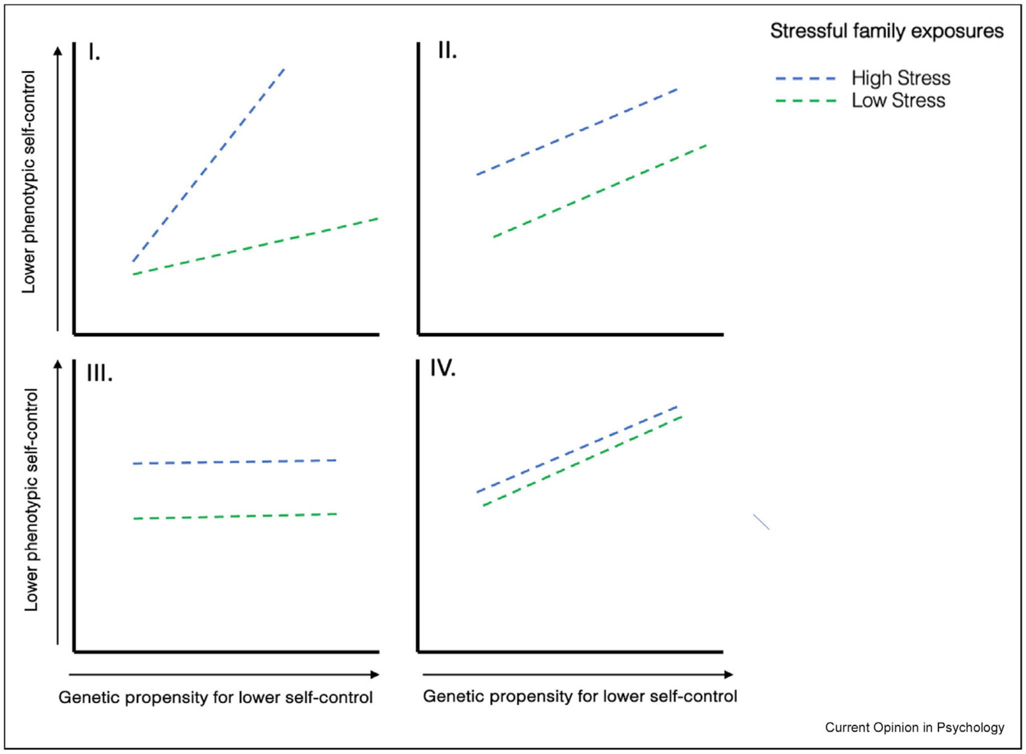


Illustration of different patterns on the relationship between genetic propensity for self-control problems and stressful family exposures on individual differences in adolescent self-control [47].
Panel I shows one possible type of gene-environment interaction as posited differential susceptibility model [37], where those who both experienced stressful family exposure and have a higher polygenic score show elevated self-control problems. **Panel II** shows a significant effect of both stressful family exposure and genetic propensity on self-control. Those who experienced more stressful family exposure show lower self-control, and those who have higher polygenic scores show lower self-control. This does not indicate an interaction effect, as the joint effects of genetic and environmental risk factors are not significantly greater than the sum of the separate effects. **Panel III** shows a significant effect of stressful family exposure on adolescent self-control, with individuals who experienced more stress showing lower self-control. There are no effects of genetic propensity for low self-control on actual self-control. **Panel IV** shows a significant effect of genetic propensity on self-control, with those with a higher polygenic score having lower self-control. There are no effects of stressful family exposure on self-control.
Note: Other gene-environment interactions are possible, e.g. buffering effect which are not illustrated here.

have genetic propensity for low self-control *and* experienced a high number of environmental risks are at an amplified risk of actually developing low self-control in adolescence (support for panel I, Figure 2).

Other studies focusing on G x E on self-control in adolescents do find main effects of family stress and genetic propensity, but do not find evidence for G x E effects (support for panel II, Figure 2). For example, He and Li, and Ksinan showed that maltreatment and polygenic score for impulsivity independently predicted lower self-control in adolescents [31,39]. However, no interaction effect between maltreatment and genetic risk was found. Similarly, Mooney and colleagues looked at the effect of family conflict and polygenic scores of ADHD on self-control problems in adolescents, not finding consistent G x E effects across cohorts [40]. Østergaard and colleagues looked at parental unemployment, finding main effects but no significant G x E effects when including polygenic scores [41]. Together, these findings show that genetic risk and environmental risk explain low self-control, but there is no amplifying effect of the environment on this genetic risk. This mirrors the null effects for G x E on self-control in adults [42].

Opportunities for future research

More social scientists are encouraged to incorporate molecular measures to study how nature and nurture shape behavior. So far, most studies focus on cross-sectional data or data over short developmental periods, with little consideration of how polygenic scores may longitudinally contribute to self-control via accumulating mechanisms [16]. One way forward would be to investigate how polygenic scores predict self-control from early to late adolescence, something that is more commonly applied in educational sciences [43].

Research on rGE and G x E has typically focused on family-level environments, but broader sociocultural interactions are also important for shaping self-control. The null findings of G x E could perhaps be explained by its effect being dependent on larger societal processes, such as economic shocks or the COVID-19 pandemic, rather than just family-level effects. Recent research shows G x E effects on polygenic scores for educational attainment in individuals growing up in former East versus West Germany [44]. Applying similar studies to self-control would be interesting and feasible.

Integrating gene-environment designs into social science research offers the exciting opportunity to 1) understand how genetic and environmental factors shape self-control over the lifespan, and 2) investigate causality by identifying true environmental impacts on self-control. Gene-environment research is developing rapidly which will benefit researchers, with or without a genetic background, in understanding of individual differences in self-control.

Conclusion

Gene-environment interplay is a challenging yet stimulating direction to facilitate nuanced understanding of the etiological sources of adolescent self-control. Examining the causes of self-control differences while taking gene-environment interplay into account remains an intriguing yet challenging area of research, which we expect to blossom in the years to come.

Credit author statement

YW conceptualized, prepared and wrote the original draft. JL, MB and CW reviewed and edited the original draft. YW, JL, MB and CW reviewed and agreed on the submitted manuscript.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

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