



Polygenic Influences on Personality Traits and Everyday Decision Making in Relation to Self-Regulation and Risk Behavior

Khilola Ruzmetova, Khusan Marupov, Ulugbek Jumayev, Norjon Shamsiyeva, Alijon Sultanov, Qunduz Alimova, Gayrat Abdullakhodjaev, Dildora Yulchiyeva,

Department of Pedagogy, Psychology, and Inclusive Education, National Pedagogical University of Uzbekistan named after Nizami, Uzbekistan ORCID: <https://orcid.org/0009-0008-5534-1351> E-mail: hilolaruzmetova9@gmail.com

Lecturer, Fergana State University, Fergana, Uzbekistan ORCID: <https://orcid.org/0009-0004-0242-1279> , E-mail: husanboy-marupov149@gmail.com

Lecturer, Bukhara State University, Bukhara, Uzbekistan ORCID: <https://orcid.org/0000-0002-4287-0018> , E-mail: u.s.jumayev@buxdu.uz

Pediatric Infectious Disease Physician, Bukhara State Medical Institute, Bukhara, Uzbekistan , ORCID: <https://orcid.org/0009-0004-0418-0667> E-mail: norjonshamsiyeva02@gmail.com

Independent Researcher, Bukhara State University, Bukhara, Uzbekistan ORCID: <https://orcid.org/0009-0009-8053-4275> , E-mail: ALI.JON@gmail.uz

Lecturer, Department of Educational Management, Jizzakh State Pedagogical University. Jizzakh, Uzbekistan , ORCID: <https://orcid.org/0000-0001-5919-1540> E-mail: qunduzalimova2025@gmail.com

Associate Professor, Department of Social Disciplines, Doctor of Philosophy (PhD) in Law. Tashkent State Medical University , Tashkent, Uzbekistan ORCID: <https://orcid.org/0000-0001-6534-2595> E-mail: gavratxodja1952@gmail.com

Doctor of Philosophy (PhD) in Pedagogical Sciences, Senior Researcher, National Institute of Fine Art and Design named after Kamoliddin Behzod. Tashkent, Uzbekistan ORCID: <https://orcid.org/0009-0001-7805-2121> E-mail: dildora.yulchiyeva@gmail.com

ABSTRACT

Personality traits show moderate to high heritability and play a central role in shaping everyday decision making, particularly in domains involving self-regulation and risk behavior. Advances in genomic research indicate that these traits are influenced by a highly polygenic architecture, with numerous common genetic variants contributing small additive effects. This review examines how polygenic influences on personality traits relate to individual differences in self-regulation, impulsivity, sensation seeking, and real-world risk-taking behaviors. Drawing on evidence from twin studies, genome-wide association studies, and polygenic score analyses, it highlights genetic correlations between personality dimensions and self-regulatory processes that guide decision making across contexts and developmental stages. Mechanistic pathways linking polygenic variation to cognition, affect, and motivational control are discussed, alongside the moderating role of environmental context and gene–environment interplay. Understanding these pathways provides insight into the biological and contextual foundations of decision making and has implications for translational research, prevention strategies, and ethical considerations surrounding genetic information.

Keywords: *personality traits, polygenic risk, self-regulation, risk behavior, decision making, impulsivity, sensation seeking, genetic correlation, gene–environment interaction, behavioral genetics.*

INTRODUCTION

The heritability of human personality, as estimated through twin and adoption studies, is approximately 30–60% (Zwir et al., 2020). The remaining variance is expected to arise from environmental factors and

unaccounted influence of additional polygenic variation on the observed end-phenotypes. Over a number of decades various approaches have been developed to quantify the genetic architecture of personality. Under these models, one or more purely genetic contribution(s), referred to as “temperament”, influences fundamental biological processes connected to cognition, motivation, or affect (Sallis et al., 2018).

Polygenic influences on personality traits therefore imply that self-control faculties, reflected in measures of sensation-seeking, risk-taking, and impulsivity, are similarly affected by common genetic variation (Boutwell et al., 2017). The available evidence indicates that the genetic relationship between personality and self-regulation has a meaningful role in real-world decisions across diverse systems, ages, and life-stages. Such genetic architecture typifies the temporal dynamics governing personality and decision making at the molecular, circuit, and cognitive levels. It highlights also the translational potential of genetic information to inform approaches for screening, intervention, and policy. Ethical complexities remain (Azimova, S., et al).

Theoretical foundations: polygenic architecture of personality

Personality traits are important determinants of everyday choices. Two robustly interrelated constructs figure prominently in decision processes: self-regulation governs the expression of ideas and impulses, whereas risk behavior refers to the likelihood of engagement in risky or dangerous activities (Zwir et al., 2020). Self-regulation and risk behavior are implicated in real-world issues like substance abuse, gambling, and occupational injuries (V. Hibbing, 2011). Genetic contributions to personality have been extensively studied. Population-averaged heritability estimates for personality dimensions range from 40% to 60%, with up to 80% for specific traits. Recent models explore polygenic architectures and supply tools for estimating genetic influences on correlates of personality such as self-regulation and risk behavior. These contributions may operate through cognition, affect, motivation, or executive functions [table 1].

Table 1: Polygenic Architecture of Personality, Self-Regulation, and Risk Behavior

Domain	Key Constructs	Genetic Basis	Behavioral Implications
Personality Traits	Extraversion, conscientiousness, neuroticism, sensation-seeking	Moderately to highly heritable (30–60%), influenced by many common variants	Shape preferences, emotional reactivity, and baseline decision tendencies
Temperament	Biologically rooted predispositions affecting affect and motivation	Polygenic contributions influencing neural and hormonal systems	Provides stable foundations for personality and behavior across the lifespan
Self-Regulation	Effortful control, impulse inhibition, delay of gratification	Polygenic overlap with conscientiousness and executive function traits	Governs the expression or suppression of impulses during decision making
Risk Behavior	Risk-taking, impulsivity, sensation-seeking	Shared polygenic architecture with personality traits	Increases likelihood of engaging in potentially harmful behaviors

Heritability Estimates	Personality-related traits	40–60% on average, up to 80% for specific traits	Indicates substantial genetic contribution alongside environmental effects
Developmental Stability	Trait consistency across time	Stable polygenic influences interacting with environment	Explains continuity and change in behavior across life stages

Measurement of personality, self-regulation, and risk behavior

Risky decision-making depends on individual differences. Among the many factors influencing these differences, personality traits and self-regulation play an essential role. Personality affects everyday context-specific decisions, while self-regulation moderates the influence of other traits supportive of risk behavior at the moment of choice (Van Dyke, 2012).

Three multi-item, psychometrically validated, and widely accepted self-report instruments suited to measure traits and associated behaviors across diverse contexts are available now. Scales on personality and self-regulation rely upon the NEO-FFI-3 Model measure, and Zuckerman's Sensation-Seeking (SSS; Zuckerman, 1979). The complete versions of these two tools, along with the Delay Discounting Inventory (DDI; such as Shoham et al., 2021; Vuchinich & Simpson, 1998), accommodate easy access to short forms without losing modeling capacity and predictive power. Each scale is temporally orthogonalised to allow environmental assessments without interactions or confounding time-sampling constraints deriving from longitudinal assessments, thereby linking well with high-throughput genomic and epigenomic data collections for large populations (Sasmakov, S. A., et al).

Genetic correlations between personality traits and self-regulatory processes

Individuals vary considerably in their personality characteristics, self-regulation, and risk behavior. Genetic correlations exist between individuals' personality traits (such as extraversion and conscientiousness) and their self-regulatory processes (including effortful control). Estimating these genetic correlations can shed light on the nature, extent, and direction of polygenic influences among these behavioral domains. Such estimates can also advance understanding of how genetic predispositions shape complex behaviors and can aid in the development of translational research applications.

Substantial evidence indicates that polygenic influences contribute to individual differences in personality traits (Zwir et al., 2020) and self-regulatory processes (Boutwell et al., 2017). Polygenic scores (or genetic risk scores) based on publicly available summary statistics from large-scale genome-wide association studies provide a means of inferring the average effect of genome-wide common variation on these traits. These scores can then be used to examine genetic correlations, summarized as the proportion of variance in a trait that is jointly explained by polygenic scores for another trait-between personality and self-regulatory processes (Azimova, S., et al). Methods for estimating genetic correlations include twin models, which exploit the greater phenotypic similarity of monozygotic twins relative to dizygotic twins, and univariate and multivariate restricted maximum likelihood (GREML) approaches, which utilize genome-wide genotypic data and family-relationship information (van der Bijl et al., 2019) [table 2].

Table 2: Mechanisms Linking Polygenic Variation to Everyday Decision Making

Level of Influence	Mechanism	Neural and Cognitive Pathways	Impact on Decision Making
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Molecular Level	Polygenic variation across multiple loci	Distributed effects on neurotransmission and synaptic plasticity	Biases valuation, reward sensitivity, and behavioral thresholds
Neural Circuit Level	Integration of cost–benefit and probability signals	Prefrontal cortex, mesolimbic dopamine system, executive control networks	Shapes prioritization and selection of competing actions
Cognitive Level	Evaluation of desirability, feasibility, and expected value	Information weighting and policy formation	Influences everyday choices under uncertainty
Self-Regulatory Mediation	Control over impulses and goal-directed behavior	Executive function and effortful control mechanisms	Moderates risk-taking at the moment of choice
Gene–Environment Interplay	Environmental context modulates genetic expression	Peer presence, social norms, and situational cues	Alters magnitude and direction of genetic effects on behavior
Real-World Outcomes	Health and social decisions	Substance use, gambling, occupational risk	Genetic predispositions translate into observable life outcomes

Mechanisms linking polygenic variation to decision making

Polygenic variation in personality influences diverse cognitive, affective, and motivational processes that characterize everyday decision making. Characteristically polymorphic risk alleles associate with neural systems that shape the categorization, weighting, and prioritization of proposed actions across multiple contexts, conditions, and stages of development (Azimova, S., et al). Such pathways construct synthetic policies based on previously available information and alter the specification of desirability, feasibility, and expected value estimates of prospective goals and courses of action. Comprehensive gene-by-environment interaction models determine whether these parameters increase or decrease the propensity to accelerate or inhibit instrumentally contingent behaviors (J. Strawbridge et al., 2018). Gene-related deviations primarily affect temperamental inclinations yet can also activate strategies acquired from others via specialized neurobiological mechanisms (Boutwell et al., 2017).

The transactions that transfer polygenic variation into decision making are believed to proceed through a small number of specialized routes. Such medians transmit information concerning benefits, costs, delays, and probabilities and are communicated to different nodes within a collection of circuits that participate in the elaboration and execution of alternative behavioral programs. This distribution allows a diverse set of normative actions to compete for selection in parallel, supplying the opportunity for deviations at various points in the sequence to generate widely distinct profiles of comportment across individuals. Outcomes from pre-execution policy formation can modulate the utilization of other kinds of information during lower-level planning and even determine which endeavors remain targetable at the outset. Such effects can propagate variations in an amplifying manner across successive levels of the cognitive hierarchy and through the range of traits associated with the preliminary overall input, implicating the architecture of personality as theoretical guidance into an assemblage of global decision-making forms (Sasmakov, S. A., et al).

Environmental context and gene-environment interplay

Models of temperament emphasize the interplay between biological predispositions and environmental influences, with genetic endowment shaping environmental exposure, vulnerability, or resilience (A. Wiebe et al., 2009). Gene-environment-decision-making models broaden this perspective by recognizing that environmental factors can actively influence decision making and behavioral regulation, thereby informing the gene-decision-making pathway. These models highlight the role of context, such as the presence or

absence of peers, in determining appropriate decision-making strategies. The distinction between context as a moderator of gene-decision-making associations and context as a feature of the gene-decision-making process remains unresolved. Depending on the emphasis placed on differential susceptibility, gene-environment interactions may unfold across distinct life stages or across the lifespan ((Judith B. M.) Ensink et al., 2019) , with age at first risk behavior varying by setting, such as home versus away from home. Further development of systems models to capture these principles is essential (Sasmakov, S. A., et al).

Methodological considerations and evidence synthesis

Adopting a broad definition, polygenic refers to the influence of many genes, each with a small cumulative effect (J Lewis et al., 2014). Polygenic architecture underlies most behavioral and psychological changes, including personality traits ranging from impulsivity and emotional stability to novelty seeking. Personality consists of relatively stable and distinct patterns of thinking, feeling, and behaving, usually summarised by five dimensions: extraversion, agreeableness, neuroticism, conscientiousness, and openness to experience. Polygenic influences on basic personality traits are assumed to constrain individual differences in fundamental aspects of self-regulatory processes relevant for everyday decision making. Self-regulation is defined as the ability to monitor and modulate thoughts, emotions, and behaviours. Daily life decisions concerning financial commitments, health maintenance, substance consumption, and social interactions differ with respect to impulsivity, sensation seeking, and information processing, all of which are linked to both personality dimensions and risk behaviour (Zwir et al., 2020). Risk behaviour refers to choices that can lead to undesirable consequences for individuals and others, such as substance or alcohol abuse, gambling, reckless driving, or unsafe sex. The genetic architecture underlying basic personality traits is hypothesised to govern inter-individual variability in these self-regulation aspects and their practical implications (Abdurakhmanov, J., et al).

Implications for risk behavior across the lifespan

The longstanding expectation that polygenic risk influences personality traits such as self-control and sensation seeking is supported by multiple lines of evidence. Consistent with this hypothesis, many human characteristics exhibit a polygenic architecture, including neuroticism and general cognitive ability, which is in turn related to impulsivity, sensation seeking, and risk-taking behavior. Genetic correlations of polygenic scores across diverse studies are also significant. Furthermore, genetically influenced aspects of personality and cognition map onto value-based decision-making processes linked to these traits, revealing focal neural circuits that mediate the effects of polygenic variation.

At the behavioral level, the evidence indicates that polygenic influences on risk behavior unfold largely across the second and third decades of life, with specific genetic contributions to impulsivity and sensation seeking also evident in late adolescence. Early adolescent self-report measures of impulsive behavior predict adolescent sensation seeking, which in turn relates to risk-taking in young adulthood (Azimova, S., et al). Temporal ordering of genetic effects similarly emerges, although the models involve differential genetic variance estimates to signify availability rather than cumulative change. At the personality level, the modest levels of polygenic influence detected across the lifespan reflect both multiple and high-dimensional characterizations of risk-related traits, augmented by the availability of genotypic data. Theoretical models, therefore, identify a neotropical scheme of organization, consistent with broader life history parameters, through which polygenic influence operates on risk behavior over the life course (J. Strawbridge et al., 2018).

Practical applications and policy considerations

Proposals for targeted screening, personalized interventions, and policy guidance merit consideration in light of genetic contributions to self-regulation and risk behavior although careful attention to ethical issues and implications for equity is essential. Polygenic scores offer a promising avenue for improving population mental health and reducing risk behavior related to self-regulation (Zwir et al., 2020). Research highlights the potential for large-scale screening of self-regulation polygenic scores in the context of pharmacological

and non-pharmacological interventions that might be adapted to individuals' decision-making patterns and regulation abilities (Bleidorn et al., 2019). Such advances stand to inform policies that promote self-regulation and reduce risky behaviors (V. Hibbing, 2011). The ecological model of self-regulation posits that motivations, capabilities, and opportunities are influenced jointly by environmental contexts and individual characteristics. Interventions that adapt to individuals' propensity to rapid decision-making-linked to self-regulation and impulsivity polygenic scores-can have significant effects on consumer behavior. Since genetic data may be less emphasizee than broader information about individual preferences over time, population-wide screening could also be envisioned. Different life stages support specific strategies for the longitudinal regulation of self-regulation and risk behavior (Sasmakov, S. A., et al).

Conclusion

Individual differences in personality, self-regulation, and risk behaviour can affect many key life outcomes. Legal, financial, educational, and health-related choices-like obeying speed limits, eating healthily, exercising regularly, or saving for retirement-often require trade-offs between short- and long-term consequences. Psychological research indicates that temperament and personality define predispositions for specific decision rules and outcomes (Zwir et al., 2020). Genetic studies therefore have much to contribute to understanding, predicting, and potentially influencing a person's risk behaviour across their lifetime. Non-genetic environmental factors-such as family, peers, education, traumatic events, or an individual's own choices-are typically overwhelming in explaining personality differences. Nonetheless, substantial genetic contributions remain. A full understanding of genetic influences should consequently incorporate the person's polygenic background, defined as the specific genetic variants, and the associated polygenic risk, the cumulative level of those variants. Multiplex gene-environment interplay-whereby polygenic influences on personality develop, manifest, and modify an individual's response to the environment over successive life stages-must also be integrated. The central premise is therefore polygenic architecture influences personality, temperament, and self-regulation, and those influences in turn shape vulnerable decision-making traits, including self-regulation, impulsivity, and risk-related choices. Despite a relatively small literature, the available data broadly support the theory. Polygenic risk and gene-environment interplay indeed engage ostensibly distinct lifetime decisions. Individuals repaying student loans versus mortgages, entering occupations demanding high social interaction versus solitude, or pairing with high-agreeable versus low-agreeable partners select very different long-term decision policies and, by implication, different genetic profiles.

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