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Article

# Compensated Structural Savantism: Neurogenomic Characterization of High Cognitive Ability in a Phenotype at Risk for Autism Spectrum Disorder

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## Abstract

This case study (N=1) investigates the neurobiological and genomic architecture of a male individual with a history of exceptional cognitive performance (profound giftedness), contrasted with behavioral traits of neurodivergence. Integrated analysis of polygenic risk scores (PRS), structural neuroimaging, and cognitive assessment reveals a rare phenotype termed here as "Compensated Structural Savantism". The results demonstrate a paradoxical coexistence: a high polygenic risk for Autism Spectrum Disorder (81.6th percentile), typically associated with functional deficits, superimposed on elite markers for Executive Function (99.7th percentile) and Hippocampal CA1 Volume (99.8th percentile). It is concluded that high executive efficiency and hyperplasticity act as compensatory mechanisms, converting the predisposition to autism into productive hyperfocus and exceptional memory, preventing the clinical manifestation of impairments.

**Keywords:** giftedness; neurogenomics; savant syndrome; autism spectrum; executive function; case study

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## 1. Introduction

The intersection between high cognitive ability (giftedness) and neurodevelopmental singularities presents significant diagnostic and theoretical challenges. While the literature describes 'Savant Syndrome' as a condition of islands of genius coexisting with deficits, there is a gap in the understanding of high-functioning phenotypes that touch the spectrum of neurodivergence without becoming fixed within it.

This article presents the neurogenomic analysis of an individual with an estimated IQ in the range of profound giftedness (>145 SD15), multiple records of creativity and leadership in high-IQ societies, who manifested, predominantly during childhood, subtle behavioral peculiarities restricted to the dynamics of social interaction. These characteristics, while not constituting a formal clinical diagnosis of Autism Spectrum Disorder (ASD), correlate with a genetic predisposition for low extroversion, suggesting a profile where traits of early social withdrawal are mitigated and reconfigured by high-performance compensatory neural mechanisms.

## 2. Methodology (Revised for N=1 with Self-Analysis)

### 2.1. Study Design and Subject

This study adopts a single case study design (N=1) focused on the integration of genomic, neuroimaging, and phenotypic data. The study subject is a 44-year-old adult male identified as carrying genetic variants of interest for high cognitive ability and neurodivergence. **Declaration of Identity:** For methodological transparency, it is declared that the study subject corresponds to the principal author (F.A.A.R.). Biological data collection was performed by independent third-party

laboratories (sequencing via *Nebula* and clinical neuroimaging), ensuring blindness in the generation of raw data, while clinical interpretation was reviewed by the second author to mitigate self-reporting bias.

## 2.2. Data Collection

- **Genomics:** DNA was extracted and sequenced targeting single nucleotide variants (SNPs). Standardized Polygenic Risk Scores (PRS) were calculated based on European cohorts (EUR), analyzing markers for General Intelligence (96.7th percentile - GIP Score), Executive Function (98.8th percentile - Efficiency), and Risk for Autism Spectrum Disorder (71.4th to 81.6th percentile).

- **Neuroimaging:** Magnetic Resonance Imaging (MRI) and Angiography (MRA) reports (2017-2025) were reviewed. The analyses confirm anatomical normality and the absence of expansive or acute vascular lesions, validating structural functionality.

### 2.2.1. Score Interpretation Criteria

It is imperative to distinguish that the percentiles presented in this study refer strictly to genetic predisposition (**Polygenic Risk Scores - PRS**) and do not correspond to percentiles of direct phenotypic performance (such as the result of a standardized IQ test or clinical psychometric assessment), although there is a strong statistical correlation between the two. The PRS indicates the subject's position on the genetic risk distribution curve compared to the base population (European cohort). For the categorization of predisposition levels in this study, the following classification scale was adopted:

- **Low Predisposition:** Percentile  $\leq 35\%$
- **Population Average:** Percentile between 36% and 64%
- **High Predisposition:** Percentile  $\geq 65\%$

### 2.2.2. Reference Population and Ancestry

For the calculation and normalization of Polygenic Risk Scores (PRS), the European cohort (EUR) was used as the population reference. This methodological selection is based on the analysis of the subject's global genomic ancestry, which demonstrates a composition of over 95% European origin. Ancestry matching is an imperative statistical prerequisite in post-GWAS analyses to mitigate population stratification biases, ensuring that risk percentiles reflect real biological variations and not artifacts of ancestral divergence.

## 3. Statement of Ethics and Conflict of Interests (Required)

**Ethics and Consent Statement:** As the principal investigator is also the research subject, free and informed consent for the publication of sensitive genetic and clinical data is considered full and explicit. All analyses followed the principles of the Declaration of Helsinki.

**Conflict of Interest:** Author F.A.A.R. declares that he is the subject of the case study. Author Adriel Pereira da Silva declares no conflict of interest and acted in the independent review of the data and in the clinical structuring of the manuscript.

## 4. Results

### 4.1. Architecture of Intelligence and Neural Efficiency

- Integrated genomic analysis revealed a cognitive architecture situated at the upper end of the population distribution (percentile  $> 95\%$ ), characterized by the coexistence of high raw processing power (*hardware*) and elite executive efficiency (*software*).

#### 4.1.1. General Intelligence (g Factor) and Development

The subject presents a GIP Intelligence Score at the 96.7th percentile, an index composed of elite markers associated with superior cognitive ability. Corroborating this data, the potential for Early Developmental Intelligence reaches the 99.6th percentile, indicating an exceptional cognitive "start-up" speed during neural maturation, which served as the basis for the consolidation of crystallized intelligence in adulthood. The General Cognitive Potential (based on Savage et al.) is at the 94.0th percentile, confirming a generalized capacity to deal with complexity.

#### 4.1.2. Executive Function: The Compensation Mechanism

The most distinctive marker of the profile, acting as the main compensatory mechanism for neurodivergent traits, is Executive Function with 99.8th percentile.

- **Mental Agility (TMT-B):** The subject is positioned in the 99.7th percentile, indicating cognitive flexibility and task switching speed (multitasking) at the absolute top of the population.
- **IQ Management Efficiency:** The executive efficiency score reaches the 98.8th percentile, suggesting that the prefrontal cortex exerts rigorous "Top-Down" control, allowing for the productive direction of hyperfocus.

#### 4.1.3. Memory and Information Processing ("High Mental RAM")

The memory architecture presents characteristics compatible with a *savant profile*, demonstrating a massive capacity for data retention and manipulation:

- **Hippocampal Integrity (CA1):** The genetic structural volume of the CA1 subfield of the hippocampus, critical for the consolidation of new episodic memories, is in the 99.8th percentile.
- **Working Memory:** The capacity of "Mental RAM" (Digital Span Backward) is located at the 92.5th percentile, allowing for the manipulation of multiple complex variables simultaneously.
- **Rapid Semantic Access:** The integrity of the Left Uncinate Fascicle, the pathway that connects semantic memory (temporal lobe) to Broca's area, is at the 98.6th percentile, facilitating instant lexical access and verbal fluency.

#### 4.1.4. Neuroplasticity and Structural Connectivity

Biomarkers of neural infrastructure reveal a system optimized for speed and adaptation:

- **Logical Superconductivity:** The integrity of the white matter in the left hemisphere (responsible for logic and language) is at the 86.9th percentile, suggesting high-speed signal transmission.
- **Neuroplastic Reserve (BDNF):** Genetic levels of Brain-Derived Neurotrophic Factor (BDNF) are at the 93.4th percentile, conferring a high capacity for synaptic regeneration and continuous learning.
- **Cortical Efficiency (Synaptic Pruning):** A rostral frontocortical thickness (RFPT) is observed at the 14.5th percentile (low thickness). In contexts of high intelligence, this marker is interpreted as a sign of neural efficiency (optimized synaptic pruning), where the brain prioritizes connection speed over the volume of unused gray matter ("aerodynamic brain").

#### 4.2. The Autism Paradox and Neurodivergence

- Genomic data reveal a complex biological basis that confirms neurodivergence through a "Broad Autism Phenotype" (BAP) architecture. The analysis challenges the absence of a classic clinical diagnosis by demonstrating that the genetic predisposition to the spectrum is converted into functionality through highly efficient compensatory mechanisms.

#### 4.2.1. The Genetic Basis of the Spectrum (Synaptic Signaling)

The subject presents a HIGH genetic susceptibility to Autism Spectrum Disorder (81.6th percentile), reinforced by a developmental bias that shifts the neurodevelopmental architecture by 77.6% towards Autism (to the detriment of conditions such as Tourette's).

- **High-Impact Sentinel Variant (GRIN2A):** Analysis identified the presence of the TT risk genotype in the GRIN2A gene (rs145791381), classified as "High Predisposition." This gene regulates the GluN2A subunit of NMDA glutamatergic receptors, crucial for excitatory synaptic plasticity and memory formation. Alteration in this pathway is a robust biomarker frequently associated with the hyper-systematization and rigid attentional focus typical of the spectrum.

#### 4.2.2. The Isolation Phenotype: Sensitivity vs. Schizoid Trait

Unlike the hypothesis of a schizoid personality disorder (whose genetic risk has proven to be LOW, at the 37.5th percentile), the social isolation and introspection observed in the subject's history are biologically explained by the interaction between Low Extraversion (12.9th percentile) and High Biological Sensitivity (98th percentile).

- **"Sensory Guarding" Mechanism:** The nervous system exhibits high basal reactivity to stimuli (genetic neuroticism), which makes prolonged social interaction energetically costly. Solitary behavior, therefore, does not reflect social anhedonia (since Agreeableness is 98.6th percentile), but a neurobiological strategy for preserving cognitive resources for hyperfocus activities.

#### 4.2.3. Selective Hyperconnectivity and Systematization

The brain architecture does not present the global pattern of disconnectivity seen in classic autism, but rather a "selective hyperconnectivity" in pathways that support crystallized intelligence and semantic memory, compensating for deficits in intuitive social processing:

- **The Emotion-Reason "Bridge" (Uncinate Fascicle):** Contrary to the pattern of low connectivity in the spectrum, the subject presents an axonal density (ICVF) in the Uncinate Fascicle situated at the 91.5th percentile and a fast access integrity in the left hemisphere at the 98.6th percentile. This allows logic (frontal) to access emotional content (temporal) with "broadband" speed, intellectualizing feelings that would be difficult to process intuitively.
- **Frontal Integration (Forceps) Minor:** Frontal interhemispheric connectivity reaches the 85.7th percentile, facilitating complex executive coordination.
- **Memory Systematization (CA1):** The structural volume of the CA1 subfield of the hippocampus (99.8th percentile) provides the necessary *hardware* for the massive retention of details, a central characteristic of the *Savant phenotype* that allows the systematization of large volumes of information.

#### 4.3. Compensatory Mechanisms (The Neural "Shield")

- The absence of severe functional impairment and high social adaptation, despite vulnerabilities to autism spectrum traits and low extroversion, are explained by a robust genetic compensation architecture that acts as an elite "operating system" over neurodivergent hardware.

##### 4.3.1. Limbic Stability and "Cold Blood" (Amygdala)

Unlike the emotional hypersensitivity common in classic autism, the subject presents a low reactivity profile in the amygdala (4th percentile). Biologically, this confers exceptional "top-down" emotional stability: the brain is not "hijacked" by immediate social fear or anxiety responses. Combined with high agreeableness, the subject navigates complex social interactions not through gregarious intuition (since Extraversion is low, 12.9th percentile), but through rational emotion regulation, allowing for an analytical "diplomat" stance.

#### 4.3.2. The Reason-Emotion Bridge (Uncinate Fasciculus)

The typical social disconnection of the spectrum is mitigated by extraordinary structural integrity in the Left Hemisphere's Uncinate Fasciculus (98.6th percentile). This white matter pathway connects the limbic system (emotion) to the frontal cortex (reason/language), allowing "Rapid Access" to semantic memory and the intellectualization of feelings. This compensates for deficits in intuitive empathy with high-speed cognitive empathy.

#### 4.3.3. The Neurochemical Engine: Dopamine vs. Motivation

The profile reveals a paradox that explains the history of disinterest in school contrasted with professional success:

- **Low Motivation for the "Generic":** The Motivation score on the Imposed Cognitive Task is extremely low (6.3rd percentile), explaining the aversion to standardized school curricula.
- **High Dopamine Synthesis Capacity:** Conversely, the enzymatic capacity for dopamine synthesis (DDC) is HIGH (85.8th percentile), supported by robust levels of Tyrosine (85.3rd percentile).

**Conclusion:** The individual possesses a "Ferrari engine" (high dopamine levels) that refuses to engage with mundane tasks, but when fueled by self-interest (hyperfocus), provides massive fuel for sustained productivity.

#### 4.3.4. Functional Hyperfocus ("Anti-ADHD")

Crucial to the high-performance phenotype is the genetic risk for ADHD, which has proven to be LOW (8.2nd percentile). Unlike many cases of autism or giftedness that suffer from scattered attention, this biological profile allows hyperfocus (a characteristic of the spectrum) to be directed, sustained, and productive. The absence of attentional chaos, combined with High Conscientiousness, allows the individual to systematize their intellectual obsessions into concrete achievements and records.

### 4.4. Biological and Metabolic Vulnerabilities: The Cost of High Performance

Maintaining an elite cognitive architecture imposes a significant metabolic and homeostatic cost. The analysis identified specific biological bottlenecks that act as limiting factors for sustained performance.

#### 4.4.1. Iron Metabolism and Neural Homeostasis

Genomic analysis revealed an Iron Overload Risk profile situated at the (80.8th percentile), characterized by the presence of the H63D variant (heterozygous CG genotype) in the HFE gene and polymorphisms in the TMPRSS6 gene associated with increased absorption.

- **Pathophysiological Mechanism:** Although iron is an indispensable cofactor for dopamine synthesis and myelination, hepcidin dysregulation in this profile favors the accumulation of free iron not bound to transferrin. In the central nervous system, excess iron catalyzes the Fenton reaction, generating free radicals that result in oxidative stress and ferroptosis (iron-programmed cell death).

- **Clinical Impact:** This mechanism correlates physiologically with symptoms of "brain fog" and fluctuations in cognitive performance observed by the subject, demonstrating that neurotoxicity from metals can transiently compromise neural efficiency, even in high-capacity brains.

#### 4.4.2. Neurobiology of High Sensitivity: The Cost of "Sentinel Intelligence"

The neurogenetic architecture of the subject presents a High Biological Sensitivity at the (98.0th percentile). This trait does not represent psychological fragility, but rather a nervous system genetically "tuned" for the detection of environmental discrepancies and risks, a phenotype described in the literature as "Sentinel Intelligence".

- **Energy Cost:** The deep processing of sensory and emotional stimuli requires a high basal energy consumption, making the individual more susceptible to sensory fatigue and exhaustion in chaotic environments.

#### 4.4.3. Vulnerability to Anxiety (F41) vs. Performance

The subject exhibits a high clinical predisposition for anxiety dysregulation (86.5th percentile, *Risk F41*), coexisting with extreme biological sensitivity (98.0th percentile). This architecture suggests an "Executive Hijacking" mechanism under conditions of acute stress.

- **The Neurobiological Mechanism:** Under stress, hyperactivation of the limbic system and the HPA axis competes for the metabolic resources of the dorsolateral prefrontal cortex (DLPFC), a region where the subject exhibits high neural efficiency (14.5th percentile) of thickness, indicating optimized synaptic pruning.

- **Hypothetical Performance Modeling:** Although cognitive "hardware" is intact and validated with a ceiling of **160 points** ( Wechsler Scale ) under homeostatic conditions, the high sensitivity profile allows for the schematization of a **hypothetical functional variation** for clinical illustration purposes. In a scenario of extreme pressure, the "noise" of anxiety could block access to working memory, resulting in momentarily lower performance (e.g., , a hypothetical score of 135), not due to a lack of logical ability, but due to the temporary unavailability of executive resources.

- **Conclusion:** The system operates under an "all or nothing" dynamic: maximum capacity (160 IQ) is always present, but access to it is conditioned by the regulation of the anxiety state.

#### 4.4.4. Neural Hyper-Excitability ("Synaptic Noise")

The genetic risk for neural excitability and hypersynchronous discharges is at the 86.1st percentile. This suggests that the subject's neurons have a lower activation threshold (membrane potential closer to firing).

- **Efficiency Paradox:** While favoring quick reactions and rapid creative associations, this characteristic generates excessive neural "noise" under stress. Without adequate GABAergic inhibition, the excess signaling impairs clarity of logical thought and executive efficiency, requiring a controlled environment for the manifestation of maximum potential.

#### 4.4.5. Specific Markers of Sensory Processing and Stress

The analysis of single nucleotide variants (SNPs) corroborates the physiology of a *Highly Sensitive Person* (HSP) with alterations in neurotransmitter and cortisol modulation:

- **SLC6A3 (Dopamine Transporter):** The CC genotype in the rs27072 marker is associated with increased sensory sensitivity, indicating dopaminergic modulation that prioritizes stimulus detection over filtering.

- **CRHBP (Corticotropin-Releasing Hormone Binding Protein):** The presence of the risk allele A (genotype AG in rs10062367) is linked to increased sensitivity to stress and acute sensory processing, especially in adverse developmental contexts.

- **NR3C1 (Glucocorticoid Receptor):** The rs41423247 variant is associated with hypersensitivity to glucocorticoids, indicating that the body takes longer to return to homeostasis after a stressful event (slow cortisol recovery).

#### 4.5. Phenotype Conclusion

The subject operates with massive processing "hardware," characterized by a genetic predisposition for General Cognitive Potential at the 94.0th percentile, phenotypically validated by reaching a ceiling of 160 points in a standardized neuropsychological assessment (Wechsler Scale). This elite processing system is, however, coupled with an extremely rigid neurobiological "safety sensor," evidenced by a Biological Sensitivity at the 98.0th percentile. This architectural configuration establishes a model of Conditional High Performance: in homeostatic and safe environments, the

capacity for logical integration and memory operates at a functional *savant level*; under conditions of psychosocial pressure or metabolic dysregulation, exacerbated by the genetic risk of iron toxicity (80.9th percentile), the limbic alarm system prioritizes biological defense, temporarily restricting the allocation of energy resources to higher executive functions.

## 5. Discussion

### 5.1. High-Performance Functional Neurodivergence

#### 5.1.1. The Functional Savant Phenotype Equation ( $\Phi_{SF}$ )

Based on the genetic report, we can synthesize this profile into an equation that describes the phenomenon of high-performance functional neurodivergence.

$$\Phi_{SF} = \frac{[(G_{at} \cdot K_{sav} \cdot OC) + (IQ \cdot EF \cdot A)] \cdot M_{neuro}}{\Delta_{clin}}$$

#### 5.1.2. Definition of Terms:

**$G_{at}$  (Atypical Neurogenetic Potential):** Represents the raw biological basis. It is the product of Polygenic Risk for Autism (81.6th percentile) and Neurodevelopmental Bias (77.6%). It is the "engine" of differentiated processing.

**$K_{sav}$  (Savantism Coefficient):** Represents the "Islands of Genius". It is the weighted average between Working Memory (99.8th percentile) and Logical Systematization (88.1st percentile). This coefficient defines the technical depth of hyperfocus.

**$OC$  (Obsessive-Compulsive Component):** The obsessive-compulsive component ( $OC$ ) in the equation represents the genetic and neurobiological factor of cognitive rigidity and directed hyperfocus, derived from its elevated risk for Obsessive-Compulsive Disorder (90.8th percentile).

The component  $OC$  is defined by the equation below, where  $PRS_{TOC}$  = Polygenic Risk Score for OCD (specific genetic predisposition) multiplied by the intensity of monotropic hyperfocus  $\sigma_{foco}$  (measured via *Digital Span* and *Perseverance* tests).

$$OC = \log(PRS_{TOC}) \cdot \sigma_{foco}$$

OCD is not noise, but a mechanism for cognitive acceleration, keeping the system in a high-performance "loop", blocking dispersive stimuli, as evidenced by the low risk of ADHD, and directing neural resources exclusively to tasks aligned with specific interests. Without the  $OC$  component, the  $K_{sav}$  would be chaotic, like a supercomputer without an operating system, with the component  $OC$  the brain operates like a *cognitive laser*: focused, coherent, and thermodynamically efficient.

**$(IQ \cdot EF \cdot A)$  (The Social Operating System):** This is the competitive differentiator as it represents General Intelligence (96.7th percentile), Executive Efficiency (99.7th percentile), and agreeableness (98.6th percentile). In the report, these factors act as "Cognitive Masking" that transforms autistic traits into social and professional competencies.

**$M_{neuro}$  (Neurochemical module):** (dopamine, norepinephrine, GABA, serotonin, melatonin, oxytocin, adrenaline, and endorphins). This module is essential because the analyzed profile has characteristics that are dependent on the neurochemical state, such as:

- Functional variation in IQ (135 → 160)
- Iron-induced brain fog/metabolic lability
- Neural hyperexcitability (86.1%)
- Physiological anxiety (86.5%)
- Biological hypersensitivity (98%)
- Dopamine-dependent hyperfocus

The module  $M_{neuro}$  It acts as a biological filter of performance where:

- Dopamine = motivation and hyperfocus

- Norepinephrine = alertness and speed
- Serotonin = emotional stability
- GABA = inhibition and clarity
- Melatonin = circadian rhythm
- Oxytocin = socialization
- Adrenaline = stress response
- Endorphins = resilience

When these neurotransmitters are in homeostasis, the system operates at its maximum level (*high-end savantism*). When dysregulated, temporary "executive collapse" occurs. In the equation, the module  $M_{neuro}$  is multiplied the equation because the neurobiological system works through modulation, not linear addition; neurotransmitters don't add capacity, they modulate access.

$\Delta_{clin}$  (**Clinical Risk Differential**): This is the denominator that reduces the chance of a pathological diagnosis. It is composed of the low risk of ADHD (8.2nd percentile) and low risk of schizoid isolation (37.5th percentile). The lower this value, the greater the functionality of the system.

### 5.1.3. Interpretation of the Condition

The equation demonstrates that, in this specific case, the high genetic load associated with autism spectrum disorder  $G_{at}$  does not manifest as a deficit because it is potentiated by two crucial factors:

1. the presence of a functional obsessive-compulsive component (**OC**), which transforms cognitive rigidity into productive focus, and
  2. The combined action of General Intelligence ( $IQ$ ), Executive Function ( $EF$ ) and Agreeableness ( $A$ ), which direct hyperfocus towards concrete and socially adapted goals.
- **The Elevated Numerator:** formed by  $[(G_{at} \cdot K_{sav} \cdot OC) + (IQ \cdot EF \cdot A)]$  represents a system with enormous processing capacity, logical precision, repetition oriented towards excellence, and great social adaptability. Here, the autistic-savant "engine" is coupled to a highly efficient executive "steering wheel."
  - **Neurochemical Module ( $M_{neuro}$ ):** acts as the dynamic regulator of this system. Dopamine, norepinephrine, serotonin, GABA, oxytocin, melatonin, adrenaline, and endorphins modulate, in real time, access to full cognitive potential. When the neurochemical state is in balance, the system operates at its peak; when there is dysregulation (e.g., physiological anxiety, high sensitivity), performance may fluctuate.
  - **The Low Denominator:** the clinical differential  $\Delta_{clin}$  demonstrates the absence of disorganizing factors such as ADHD (8.2nd percentile) and schizoid isolation (37.5th percentile). Because this denominator is small, it hardly reduces the numerator, allowing the genetic makeup to operate with maximum focus and minimal interference from cognitive noise.

Thus, the final value,  $\Phi_{SF}$ , does not describe a disorder, but rather Profound Giftedness, a case in which autism does not act as a barrier, but rather as a precision architecture, converted into high performance thanks to the compensatory system and neurochemical control .

### 5.1.4. The Equation Is Applicable to All Scenarios.

- a) The Multiplier Effect of Compensation:** In this case, intelligence and executive function not only add up, but they also multiply the usefulness of autistic and obsessive traits. While a Classic Savant has the "engine ( $G_{at} \cdot K_{sav}$ )", he lacks the "navigation system ( $IQ \cdot EF \cdot A$ )", which prevents cognitive rigidity from becoming unproductive stereotypy.
- b) The Reduced Clinical Barrier ( $\Delta_{clin}$ ):** In traditional clinical profiles, ADHD, impulsivity, and isolation act as a "power divider" that dramatically reduces performance. In this case, the ( $\Delta_{clin}$ )

It is very low (8.2nd percentile and 37.5th percentile), the system operates in a nearly lossless manner, a clean circuit, without distractions and without dispersion.

- c) **The Difference Between Being Intelligent and Being Structurally Savant:** Neurotypical individuals with high IQs may be efficient, but they lack the term ( $G_{at} \cdot K_{sav} \cdot OC$ ) extreme memory, which stands for deep systematization and monotropic focus. They are quick, but they don't possess the architecture of an "extreme specialist."
- d) The equation presented mathematically isolates what the genetic report describes: the rare combination where neurodivergence ceases to be a "disorder" (due to the low denominator) and becomes a "superpower" (due to the high compensating multiplier).

### 5.2. Deconstructing the Binary: Compensated Structural Savantism

The findings suggest that the subject does not fit into the binary clinical categories of "Neurotypical" or "Classic Autism," but rather into a rare phenotype that we define as Compensated Structural Savantism. Genomic analysis reveals a brain architecture where the highly specific hardware (typical of the spectrum) is governed by elite executive software.

### 5.3. Superconductivity and "Islands of Genius"

The basis for the subject's exceptional abilities lies in the superior structural integrity of specific neural pathways.

- **Logical-Verbal Processing:** The "superconductivity" observed in the white matter of the left hemisphere (86.9th percentile) facilitates ultra-fast transmission between Broca's and Wernicke's areas, supporting verbal fluency and accelerated analytical reasoning.

- **Direct Access Memory:** The mnemonic "islands of genius" are biologically supported by a structural volume in the CA1 subfield of the hippocampus located at the 99.8th percentile and a Working Memory at the 92.5th percentile. This confers a capacity for "Mental RAM" that allows the manipulation of multiple complex variables simultaneously, a central characteristic of functional savants.

### 5.4. The Compensating Factor: The Executive "Manager"

What distinguishes this phenotype from maladaptive autism is the Executive Function (TMT-B) situated at the top of the population curve (99.7th percentile). The subject's prefrontal cortex acts as a "strict manager," utilizing the cognitive rigidity and monotropic focus of autism (ASD risk: 81.6th percentile) for discipline and systematization, instead of allowing them to become limiting stereotyped behaviors. Additionally, high agreeableness (98.6th percentile) and the integrity of the Uncinate Fascicle (98.6th percentile) function as social adapters, allowing for the cognitive simulation of empathy and gregarious behavior.

### 5.5. IQ Fluctuation and the Cost of Sensitivity

The variation in IQ test scores (ranging from 135 to 160+) does not reflect instability in competence, but rather the interference of High Biological Sensitivity and Neuroticism (98th percentile).

- **Neural Noise:** The subject exhibits a predisposition to High Neural Excitability (86.1st percentile). Under stress or evaluation pressure, this low firing threshold generates synaptic "noise" (physiological anxiety, F41 risk at the 86.5th percentile) that competes for the resources of the prefrontal cortex.

- **Flow State:** In a state of rest or hyperfocus, when the amygdala's threat system is silenced, full processing capacity and fluid intelligence (88.1st percentile) manifest freely, reaching the cognitive ceiling.

## 6. Conclusion

This case study demonstrates that high-risk genetics for Autism Spectrum Disorder (77.6% bias), when co-inherited with elite variants for fluid intelligence, hippocampal memory, and executive function, can result in a Profoundly Gifted phenotype rather than a disabling disorder.

The subject exemplifies adaptive neurodiversity: traits that in isolation would be deficits (such as sensory hypersensitivity and restricted focus) become competitive advantages of precision and specialization when integrated into a higher executive control system.

**Clinical and Translational Recommendations:** To preserve the cognitive longevity of this unique neurobiological profile, rigorous monitoring of its specific vulnerabilities is imperative.

1. **Iron Metabolism:** Due to the genetic risk of overload (80.9th percentile) and HFE variants, neurotoxicity from metal accumulation should be avoided.

2. **Systemic Inflammation:** Controlling elevated C-Reactive Protein and susceptibility to neuroinflammation (95.6th percentile) is crucial to prevent "brain fog" and protect the integrity of white matter.

**Conflicts of Interest Statement:** The lead author (F.A.A.R.) is the subject of the case study. The analyses were generated by independent algorithms from the *Genetic Intelligence Project (GIP)*, *NeuroVitalis*, and third-party radiological reports, ensuring blindness in the production of raw data. Clinical interpretation follows evidence-based precision medicine protocols.

## Bibliographic References and Associated Studies

### 1. Intelligence, Cognition, and Neurodevelopment

- **Child Intelligence and Development:** Benyamin , B., et al. (2014). "Childhood intelligence is heritable, highly polygenic and associated with FBNP1L." *Molecular Psychiatry*, 19(2), 253-258.

- **General Cognitive Performance and Years of Education:** Lee, JJ, et al. (2018). "Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals." *Nature Genetics*, 50(8), 1112-1121.

- **Fluid Intelligence and g Factor:** Hatoum, AS, et al. (2023). "Genome-wide association study of cognitive performance in the UK Biobank." *Nature Genetics/Molecular Psychiatry*.

- **General Intelligence (Meta-analysis):** Savage , J.E., et al. (2018). "Genome-wide association meta-analysis in 269,867 individuals identifies new genetic and functional links to intelligence." *Nature Genetics*, 50(7), 912-919.

- **Processing Speed :** Davies, G. , et al. (2018). "Study of 300,486 individuals identifies 148 independent genetic loci influencing general cognitive function." *Nature Communications*, 9(1), 2098.

### 2. Neuroanatomy and Brain Connectivity (Genetic Neuroimaging)

- **Morphometry and Connectivity of White Matter (Fasciculi, Corpus Callosum):** Tanigawa , Y., et al. (2022). "Significant sparse polygenic traits in the UK Biobank." *PLoS Genetics*. (Base study for most structural connectivity scores and fascicles).

- **Architecture of the Hippocampus and Subcampuses (CA1, CA3, Dentate Gyrus):** Liu, N., et al. (2023). "Genetic architecture of subcortical brain structures in 40,000 individuals." *Nature Genetics*.

- **Accumbens Nucleus Volume :** Hibar , DP, et al. (2015). "Common genetic variants influence human subcortical brain structures." *Nature (ENIGMA Consortium)*.

### 3. Mental Health, Personality, and Behavior

- **Major Depression:** Howard, DM, et al. (2019). "Genome-wide meta-analysis of depression identifies 102 independent variants and highlights the importance of the prefrontal brain regions." *Nature Neuroscience*.

- **Bipolar Disorder:** Mullins, N., et al. (2021). "Genome-wide association study of more than 40,000 bipolar disorder cases provides new insights into the underlying biology." *Nature Genetics*, 53(6), 817-829.

- **Schizophrenia** : Ripke, S., et al. (2014). "Biological insights from 108 schizophrenia-associated genetic loci." *Nature* (Psychiatric Genomics Consortium).

- **Autism (ASD)**: Grove, J., et al. (2019). "Identification of common genetic risk variants for autism spectrum disorder." *Nature Genetics*.

- **Neuroticism and Personality Traits**: de Moor, MH, et al. (2012). "Meta-analysis of Genome-wide Association Studies for Personality." *Molecular Psychiatry*.

- **Risk Behavior and Dependency**: Hatoum, AS, et al. (2023). "Multivariate genome-wide association meta-analysis of addiction-related traits." *Nature Mental Health*.

#### 4. Cardiovascular, Metabolic, and Liver Health

- **Coronary Artery Disease (CAD)**: Tcheandjieu, C., et al. (2022). "Large-scale genome-wide association study of coronary artery disease in genetically diverse populations." *Nature Genetics*.

- **Atrial Fibrillation** : Gunn, et al. (2024) / Nielsen, JB, et al. (2018). "Biobank-driven genomic discovery yields new insight into atrial fibrillation biology." *Nature Genetics*.

- **Syndrome Metabolic** : Park, S., et al. (2024). "Genetic architecture of metabolic syndrome and its components." *Nature Genetics*.

- **Liver Health and Fibrosis (Cirrhosis)**: Ghose, J., et al. (2024). "Genome-wide association study of liver stiffness and fibrosis." *Nature Genetics*.

- **Steatosis Hepatic (NAFLD)**: Haas, ME, et al. (2021). "Genetic association of PNPLA3 with NAFLD." *Cell Genomics*.

- **Cholesterol and Lipids** : Ma, Y., et al. (2022). "Global biobank meta-analysis of lipid traits." *American Journal of Human Genetics*.

#### 5. Other Domains (Immunity, Longevity, Nutrition)

- **Inflammation and Autoimmune Diseases (Arthritis, Migraine)**: Truong, B., et al. (2024). "Genetic drivers of immune-mediated inflammatory diseases." *Cell Genomics*.

- **Vitamin metabolism ( B12 , D)**: Revez, JA, et al. (2020). "Genome-wide association study identifies 143 loci associated with 25-hydroxyvitamin D concentration." *Nature Communications*.

- **Microbiota (Methanobacteria)** : Kurilshikov, A., et al. (2021). "Large-scale association analyzes identify host factors influencing human gut microbiome composition." *Nature Genetics* (MiBioGen Consortium).

#### Sources of Primary Genomic Data:

- **PGS Catalog** : EMBL-EBI Polygenic Score Catalog ([www.pgscatalog.org](http://www.pgscatalog.org)).

- **Open GWAS**: MRC Integrative Epidemiology Unit, University of Bristol ([gwas.mrcieu.ac.uk](http://gwas.mrcieu.ac.uk)).

- **GWAS Catalog** : NHGRI-EBI ([www.ebi.ac.uk/gwas](http://www.ebi.ac.uk/gwas)) .

Note: This bibliography refers to genome-wide association studies (GWAS) and polygenic scores (PGS) used to calculate the percentiles presented in the technical report.

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