

Legal tender VS genetic tender: golden spoon VS golden key

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-----ABSTRACT-----

The current monetary models are mainly based on gold, petroleum, food, mine, government credit and so on. In the last hundreds of years, a lot of legal tenders have faced depreciation and worthless. In addition, when the value of money is bound to foreign objects, money will overuse the greedy, jealous, angry, and other parts of human nature. As humanity's understanding of genes deepens, the context of human beings becoming the hegemon of the earth is gradually becoming clear, and also the genes bounded features. Therefore, the author tries to build a simple monetary model based on human gene mine, or in other words, human gene monetization.

Key words: *monetary models, genes*

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I. Introduction

The global monetary system (GMS), a labyrinthine and constantly evolving architecture of institutions, agreements, conventions, and technologies, underpins the entirety of modern global economic activity. This system orchestrates the creation, allocation, valuation, and international flows of money, serving as the indispensable infrastructure upon which international trade, investment, financial stability, and macroeconomic performance are predicated. Understanding the intricacies of the GMS is therefore crucial for policymakers, economists, financial market participants, and even informed citizens, as its dynamics exert a profound influence on national economies and the overall stability of the global financial order.[1,2]

Throughout the 20th and early 21st centuries, the GMS has been undergone a series of profound transformations. The classical gold standard, which characterized the late 19th and early 20th centuries, gave way to the Bretton Woods system in the aftermath of World War II. Under Bretton Woods, the U.S. dollar was designated as the world's reserve currency and pegged to gold at a fixed rate, while other currencies were, in turn, pegged to the dollar. This system, while promoting relative exchange rate stability, proved unsustainable due to growing global imbalances, inflationary pressures, and the increasing convertibility demands on U.S. gold reserves.[3,4]

The collapse of Bretton Woods in the early 1970s marked a watershed moment, ushering in the era of flexible exchange rates and fiat currencies. Fiat currencies are not backed by any physical commodity, such as gold or silver, but rather derive their value from government decree and public confidence. This transition granted individual nations substantially greater autonomy over their monetary policies, enabling central banks to independently manage interest rates and control the money supply in response to domestic economic conditions, such as inflation, unemployment, and economic growth.[5]

However, the increased flexibility afforded by the fiat currency system has not been without its drawbacks. The absence of a fixed exchange rate anchor has introduced the potential for increased volatility in currency values, impacting international trade competitiveness, creating uncertainty for businesses engaged in cross-border transactions, and potentially triggering financial crises, particularly in emerging market economies with substantial foreign currency debt denominated in foreign currencies. Furthermore, the potential for speculative currency attacks and "sudden stops" in capital flows has become a persistent concern for policymakers in developing countries.

Empirical evidence on the effects of flexible exchange rates is mixed and often context-dependent. While some studies have found that flexible exchange rates can effectively act as shock absorbers, cushioning economies from external disturbances, other research has highlighted the potential for excessive exchange rate volatility to destabilize economic activity, distort investment decisions, and even exacerbate inflationary pressures. The effectiveness of flexible exchange rates is often contingent on a variety of factors, including the credibility of

monetary policy, the degree of financial market development, the level of trade integration, and the overall institutional quality of the country in question.

A defining characteristic of the contemporary GMS is the persistence of substantial global imbalances. These imbalances manifest as large and persistent current account surpluses in some countries, such as China, Germany, and Japan, and corresponding current account deficits in other countries, most notably the United States. These imbalances have been attributed to a complex interplay of factors, including differences in savings rates, investment opportunities, demographic trends, exchange rate policies, and the global distribution of income. The accumulation of vast foreign exchange reserves by surplus countries has contributed to downward pressure on global interest rates, potentially fueling asset bubbles and contributing to financial instability.

Addressing these global imbalances requires international cooperation and coordination of macroeconomic policies. However, achieving effective policy coordination is often hampered by conflicting national interests, a lack of binding enforcement mechanisms, and disagreements about the appropriate policy responses. The International Monetary Fund (IMF) plays a critical role in promoting international monetary cooperation, providing surveillance of national economic policies, and offering financial assistance to countries facing balance of payments difficulties. However, the IMF's effectiveness is often constrained by its limited resources, its governance structure, and the political sensitivities surrounding its policy recommendations.

The contemporary GMS is also facing new challenges and opportunities stemming from rapid technological innovation, particularly in the realm of digital currencies and financial technology (FinTech). Cryptocurrencies, such as Bitcoin and Ethereum, based on decentralized blockchain technology, have attracted significant attention as potential alternatives to traditional fiat currencies, offering the promise of faster, cheaper, and more secure transactions. However, cryptocurrencies also raise concerns about regulatory oversight, consumer protection, tax evasion, and the potential for illicit activities, such as money laundering and terrorist financing.

Central banks around the world are actively exploring the feasibility of issuing their own digital currencies (CBDCs), which could potentially revolutionize the payments landscape and fundamentally alter the structure of the GMS. CBDCs could offer numerous potential benefits, including enhanced payment efficiency, reduced transaction costs, greater financial inclusion, and improved monetary policy implementation. However, they also raise complex questions about privacy, cybersecurity, data security, and the potential for disintermediation of commercial banks. The introduction of CBDCs could also have far-reaching implications for the international monetary system, potentially challenging the dominance of the U.S. dollar and reshaping the landscape of cross-border payments. The network effects of CBDCs, especially if adopted by larger economies, can disrupt the monetary sovereignty of smaller nations.

The tapestry of human diversity, manifested in the myriad variations in physical traits and predispositions to disease, arises from a complex interplay between genetic inheritance and environmental influences. While the protein-coding regions of the human genome provide the blueprint for the building blocks of life, the non-coding regions, which constitute the vast majority of our DNA, harbor a sophisticated regulatory landscape that fine-tunes gene expression and shapes cellular phenotypes. Central to this regulatory landscape are gene-bounded features – the diverse array of DNA sequences, structural elements, and epigenetic modifications that reside within and immediately flanking genes. These features, by influencing gene transcription, chromatin organization, and genome stability, play a pivotal role in determining not only cellular identity and function but also the observable traits that define individual human characteristics, such as eye color, skin pigmentation, height, and susceptibility to specific diseases. Understanding the connection between these gene-bounded features and specific human traits is essential for unraveling the genetic basis of human diversity and developing personalized approaches to healthcare.

The connection between genotype and phenotype is rarely a simple one-to-one mapping. Instead, complex traits, such as those mentioned above, are typically influenced by multiple genes, each contributing a small effect, and by interactions between these genes and the environment. Gene-bounded features act as crucial intermediaries in this process, modulating the expression of genes involved in these complex pathways.

Eye Color: The determination of eye color is a classic example of a polygenic trait, influenced by multiple genes, most notably *OCA2* and *HERC2*, both located on chromosome 15. *OCA2* encodes a melanosomal transmembrane protein involved in melanin production, which is the pigment responsible for determining eye color. The *HERC2* gene, located upstream of *OCA2*, contains a regulatory region that controls

the expression of OCA2. Specific gene-bounded features within and around the HERC2 gene, including enhancers and silencers, influence the level of OCA2 expression, thereby affecting the amount of melanin produced in the iris and ultimately determining eye color. Genetic variations within these regulatory regions, such as single nucleotide polymorphisms (SNPs), can alter the binding affinity of transcription factors and modulate OCA2 expression, leading to the spectrum of eye colors observed in the human population. A specific SNP within an intron of the HERC2 gene, for instance, is strongly associated with blue eye color.[6]

Skin Color: Skin pigmentation, another highly variable human trait, is also influenced by multiple genes involved in melanin production and distribution. The MC1R gene, encoding the melanocortin 1 receptor, plays a key role in regulating the type of melanin produced: eumelanin (brown/black pigment) or pheomelanin (red/yellow pigment). Variations in gene-bounded features surrounding MC1R, particularly within its promoter region, can influence the level of MC1R expression and the relative proportions of eumelanin and pheomelanin produced, affecting skin, hair, and eye color. Other genes, such as SLC24A5 and KITLG, also contribute to skin pigmentation, and their expression is likewise regulated by gene-bounded features. Specific SNPs near these genes have been shown to be strongly associated with differences in skin pigmentation across diverse human populations.[7]

Height: Human height is a complex quantitative trait influenced by hundreds of genetic variants scattered throughout the genome. Many of these variants are located within non-coding regions, including gene-bounded regions, suggesting that they influence height by modulating the expression of genes involved in skeletal growth and development. For example, variations near the GH1 gene, encoding growth hormone, can affect the level of growth hormone production, thereby influencing overall height. Enhancers and silencers located within introns of the GH1 gene or in its flanking regions likely contribute to the regulation of GH1 expression.

Disease Susceptibility: Gene-bounded features also play a critical role in determining susceptibility to various diseases, including cancer, autoimmune disorders, and cardiovascular disease. For example, variations in the promoter region of the IL2RA gene, encoding the interleukin-2 receptor alpha chain, have been associated with an increased risk of autoimmune diseases such as type 1 diabetes and multiple sclerosis. These variations can alter the level of IL2RA expression, affecting the function of T regulatory cells and influencing the immune response. Similarly, variations near genes involved in lipid metabolism, such as LDLR and PCSK9, can influence the risk of cardiovascular disease by affecting cholesterol levels.

Hair Texture: Hair texture, ranging from straight to curly, is determined by the shape of the hair follicle and the composition of the hair shaft. The TCHH gene, encoding trichohyalin, a protein that is important for hair follicle differentiation, plays a significant role in determining hair texture. A SNP within an intron of the TCHH gene is strongly associated with hair curliness in European populations. This SNP is thought to influence hair texture by affecting the splicing of TCHH mRNA or by altering the stability of the protein. Gene-bounded features surrounding TCHH likely regulate its expression and influence the effect of this SNP on hair texture.

Muscle Fiber Type Composition: The proportion of different types of muscle fibers (slow-twitch and fast-twitch) in skeletal muscle influences athletic performance and susceptibility to metabolic diseases. The ACTN3 gene, encoding alpha-actinin-3, a protein found exclusively in fast-twitch muscle fibers, has a common polymorphism (R577X) that results in a complete deficiency of alpha-actinin-3 protein. Individuals with the XX genotype have a lower proportion of fast-twitch muscle fibers and may be more suited for endurance activities. Gene-bounded features surrounding the ACTN3 gene likely influence its expression and contribute to the variation in muscle fiber type composition observed in the human population.

Facial Morphology: Facial features, such as nose shape and chin prominence, are highly heritable traits that are determined by the complex interplay of multiple genes involved in craniofacial development. Recent genome-wide association studies (GWAS) have identified several SNPs associated with facial morphology, many of which are located within non-coding regions near genes involved in bone growth and cartilage formation. Gene-bounded features surrounding these genes likely influence their expression and contribute to the variation in facial features observed in human populations.

Human intelligence, a complex and multifaceted cognitive trait encompassing reasoning, problem-solving, and learning abilities, is a subject of enduring scientific interest. While genetic factors are known to play a significant role in shaping individual differences in intelligence, the precise mechanisms by which genes influence cognitive function remain largely elusive. Identifying specific genes and regulatory elements that contribute to intelligence has proven challenging due to the polygenic nature of the trait, the

complex interplay of genes and environment, and the difficulties in accurately measuring and defining intelligence itself. However, studies of identical twins, who share nearly identical genomes, provide compelling evidence for the heritability of intelligence and offer a unique opportunity to disentangle the genetic and environmental contributions to cognitive ability. Increasingly, research points to the importance of gene-bounded features – the diverse array of DNA sequences, structural elements, and epigenetic modifications that reside within and immediately flanking genes – as critical regulators of gene expression in brain development and function, and therefore as potential contributors to individual differences in intelligence.

The heritability of intelligence, typically estimated to be between 50% and 80% in adult populations, indicates that a substantial portion of the variation in intelligence can be attributed to genetic factors. Studies of identical twins, raised together or apart, have been instrumental in establishing this heritability. Because identical twins share nearly identical DNA sequences, differences in their intelligence can be attributed primarily to environmental factors. Conversely, similarities in their intelligence, even when raised in different environments, provide strong evidence for the influence of genes.

However, identifying the specific genes that contribute to intelligence has been a formidable task. Early candidate gene studies, which focused on genes known to be involved in brain development or neurotransmitter function, yielded inconsistent results. Genome-wide association studies (GWAS), which scan the entire genome for genetic variants associated with a trait, have identified numerous SNPs associated with intelligence, but each SNP typically explains only a small fraction of the overall variance. This suggests that intelligence is a highly polygenic trait, influenced by thousands of genetic variants, each with a small effect.

Importantly, many of the SNPs identified in GWAS of intelligence are located within non-coding regions of the genome, including gene-bounded regions, suggesting that they influence intelligence by modulating the expression of nearby genes. Gene-bounded features, such as enhancers, silencers, insulators, and epigenetic modifications, play a critical role in regulating gene expression in the brain, and variations in these features can potentially alter the development and function of neural circuits involved in cognitive processes. For example, variations in enhancers near genes involved in synaptic plasticity, neuronal signaling, or neurogenesis could influence the efficiency of learning and memory, thereby affecting overall intelligence.

The study of identical twins provides a powerful approach for investigating the role of gene-bounded features in intelligence. While identical twins share nearly identical DNA sequences at birth, epigenetic modifications, such as DNA methylation and histone modifications, can diverge over time due to environmental influences. These epigenetic differences can potentially lead to differences in gene expression and ultimately contribute to differences in intelligence. By comparing the epigenomes and transcriptomes of identical twins with differing cognitive abilities, researchers can identify specific gene-bounded features that are associated with intelligence and gain insights into the mechanisms by which these features influence brain development and function. Future research should focus on integrating genomic, epigenomic, and transcriptomic data from twin studies to elucidate the complex interplay between genes, environment, and gene-bounded features in shaping human intelligence.

Looking into the identical twin studies, the IQ scores are only around 0-2% gap between them. Based on this result, a monetary system based on gene is valid for the importance of intelligence in modern society. While this model is simple and it needs further refinements.

Monetary model

Since IQ is the outward manifestation of genes, this monetary model is highly related to IQ, money in this model can be called genetic tender. Although it is subjective to view IQ as golden feature and appearance, height and so on as silver features, the modern social development has provided more and more evidence to enhance the importance of IQ.

Coefficient of learning cost: A. Coefficient of time cost: B. Coefficient of risk of injury: C.

$$\text{Value} = A \times B \times C \times \text{IQ}$$

While there is some overlap between IQ and the coefficients of A, B and C. For the same task, IQ may influence the learning cost, time cost and risk of injury. Therefore, this is just an oversimplified model and this model needs further refinements.

Genetic tender VS legal tender

Here the author provided a metaphor, legal tender VS genetic tender: golden spoon VS golden key. The legal tender in the aspect of technology is actually a concrete reflection of the association with genes. While for the stability and random of genes, genes can be regarded as a currency form on their own.

II. Conclusion

This paper has put forward a viewpoint of human gene monetization and built a simple monetary model based on genes. This model is an over simplified model and it needs further refinements.

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