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An Extended Evolutionary Perspective on Evolutionary Bet Hedging

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ABSTRACT

Evolutionary bet hedging reduces the risk of catastrophic failure by maintaining alternatives suited to changing environments. Classical treatments generally focus on genotypes that produce phenotypic variation, while treating mutation mainly as undirected novelty. More recent research on mutational architecture shows that mutational spaces can be parameterized—constrained, reversible, and following biologically meaningful paths. Such systems can reliably generate alternative functional genetic states which act as a hedge against plausible future environmental states. Experimental evidence shows not only instances of this type of hedging occurring in nature, but even the real time development of such systems in response to rapidly changing environments.

KEYWORDS: Evolutionary bet hedging, Mutational architecture, Mutation, Phenotypic variation, Environmental change, Genetic states, Experimental evolution.

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INTRODUCTION

In the investment arena, "hedging" is the concept of making small investments that are counter to your main investment thesis that will protect your overall financial position from ruin in case your main investment thesis fails. As an example, if I were an investor who believed strongly in the future of solar, I might put the majority of my investment into various solar companies. However, as an investor, I realize that this is a *prediction* about the future, and, like many predictions, no matter how much I may think it is true, it might turn out to be wrong. Therefore, if I had a large amount of money, I might make smaller investments in oil companies as well, or perhaps even outside of the energy sector altogether.

The idea is that, if the market conditions change, and my prediction of the future is incorrect, these other companies are more likely to be in a position to take advantage of this different landscape. I am sacrificing some amount of future profitability in order to keep my overall investment safe. If I fail to do this, any sufficiently wrong investment thesis will drive the portfolio to zero, which is effectively unrecoverable.

Now, it is important to note that hedging does *not* involve blindly investing in different companies. Even as a hedge, one would not want to invest in a company that was being

incompetently governed or inefficiently run. Giving money to a beggar on the street is not a hedge, nor is lighting it on fire. A hedge only works as a hedge if the investment is sound overall, but the investment is not optimized for current market conditions. For a counterexample, if a company made no sense under any reasonable market condition, or if it was poorly run and unable to make a profit even in favorable conditions, it is not a hedge, as the investment will still lose money even if the market conditions change. The critical feature of a hedge is not merely diversity but diversity constrained to alternatives that could reasonably become viable under plausible future conditions.

A similar situation presents itself to evolving organisms. For the most part, organisms adapt to their present environment. However, over-adaptation can lead organisms to fail if the environment changes suddenly. By placing small bets on alternate strategies, an evolving population can insulate itself from complete catastrophe if sudden environmental changes occur.

THE DEVELOPMENT OF EVOLUTIONARY BET HEDGING UNDER THE MODERN SYNTHESIS

Hedging in general is not a new concept in evolutionary biology. However, historically, the application of the idea of hedging to evolution has been overly constrained by theory.

Under the modern synthesis, mutations were thought to arise entirely due to chance, not being constrained by function or fitness in any way.[1] As such, the modern synthesis could not tolerate the idea that a mutation may itself be a hedge. As we have noted, for a hedge to operate as a hedge, it has to be constrained to be a valid investment, just not for present market conditions. The modern synthesis could not model an evolutionary process which imparted such a high degree of teleonomic direction to evolution.[2] As such, the concept of bet hedging under the modern synthesis largely revolved around other things.

The first concept of bet hedging was described by Slatkin[3] when commenting on the results of Gillespie.[4] Slatkin characterized Gillespie's suggestion of selection acting on clutch size as an instance of bet-hedging—by having the same number of offspring but with smaller clutch sizes, the total risk to offspring was reduced.

This usage was expanded and generalized by Philippi and Seger[5] to describe situations where a genotype's fitness effect varies across different generations. They suggest that the relevant metric to judge the organism by in these scenarios is the geometric mean fitness (measured by relative growth rates) of organisms with the trait rather than the arithmetic mean fitness. The reason for this is that preventing devastation is much more important for driving traits to fixation than average fitness.

Philippi and Seger also noted the usage of bet hedging for phenotypic variance for the same genotype and within-lifetime variance for the same genotype. For instance, having a genotype that causes an organism to lay eggs of varying sizes can count as bet hedging. Additionally, having a genotype which can stochastically produce different phenotypic outcomes can also be considered bet hedging.

By proposing bet hedging in these ways, biologists following the modern synthesis were able to avoid imputing any amount of teleonomy to the mutational process. All of the methods of bet hedging proposed within the modern synthesis are based on a single genotype being selected for by the environment.

THE EXTENDED EVOLUTIONARY SYNTHESIS

The extended evolutionary synthesis, however, permits biologists to expand their notions of causation in evolution. Specifically, the most common theme in the developments of the extended synthesis has been the recognition of organisms as participating in their own evolution, rather than merely passive recipients of it.[6] The umbrella term for this is "evolutionary teleonomy," and in recent years many biologists have been working under this sort of paradigm.[7,8] While there is some dispute over what technically qualifies under the banner of evolutionary teleonomy[9], the fact is that the developments within the extended synthesis clearly demonstrate that organisms play an active role in their own evolution, and our theoretical models need to be updated to account for this. As demonstrated in Bartlett[10], this theoretical update has important mathematical, experimental, and inferential implications for evolutionary theory.

Contrary to the assumptions of the modern synthesis, organisms can constrain and direct mutations to beneficial locations through a variety of mechanisms. These mechanisms can vary from protecting important segments of DNA from mutation[11] to actively promoting DNA mutation in highly specific circumstances.[12]

Additionally, the mutational space can be sufficiently constrained to make large portions of it cyclical or even parameterized.[13] In a parameterized mutation system, an organism's genome has an organization that not only allows specific genotypes to recur at certain loci, these variations match different environmental parameters. One example of a parameterized mutation system is that of *Neisseria*, in which the outer coat protein gene *pilE* cycles through different configurations that are kept in pseudogenes.[14] By having multiple complete functional sequences, the organism does not have to wait for a workable gene to evolve, but rather recombination continually provides fully-functional alternatives that function well in different environments. The point is not that the system necessarily chooses a sequence to match the environment, but that all of the genes are broadly functional, so the search is merely of *which one* performs the task best in the current environment.

Another mechanism that can enable parameterized evolution is the mutation of Simple Sequence Repeats (SSRs). SSRs are short, repetitive DNA segments, usually 1–6 bases long. SSRs mutate almost entirely by copy number variation, the mechanism of which is generally thought to be replication slippage.[15] Although the number of SSR sites is small compared to the overall number of base pairs in the genome, their high mutation rate means that copy number variation of SSRs is actually the dominant type of mutations in humans.[16] While the search for the specific parameter spaces controlled by SSRs is new, there are already both morphological and biochemical parameters that are known to be shaped by repeat expansion.[17,18] Because the mutable sites in these cases can provide repeatable, reversible, and continuous modification of traits whose

functional range roughly match the demands of an environmental parameter, they can serve to parameterize evolution in such a way as to maximize the searchability of environmentally-relevant phenotypic space while minimizing pleiotropic effects.[13,19]

Mutational processes can also be directed in response to specific stresses. Combinations of binding sites, DNA secondary structure, and mutational targets can provide a mechanism by which organisms under stress can preferentially generate specific mutations that are needed by the organism.[20] While these explicitly directed mutations are not directly important to our present study, the point is that the actual mutational architecture of genomes is significantly more sophisticated and teleonomic than the modern synthesis allowed for.[6,9,10,21]

MUTATIONAL BET HEDGING

The existence of parameterized mutations provides a new way of conceiving evolutionary bet hedging. Because these mutations are parameterized according to environmentally-relevant dimensions, they match the profile of bet hedging in a number of ways.

Like typical mutations, parameterized mutations are stochastic. But they are stochastic *within* a constrained space. And not only a constrained space, but a space whose constraints cause the mutation to track environmental or biological parameters. A mutation that was not constrained in this way could not be considered a hedge in the same way that giving money to a beggar on the street is not a proper investment hedge. However, parameterized mutations allow for the deployment of *novel but sensical* mutations which are not adaptive for the moment but also are not intrinsically problematic from a structural perspective. While the specific mutational outcomes may be random within a highly constrained set, the parameterization means that even if the mutation is not the most fit for the external environment, the mutation does not break the internal biochemical stability of the organism.

Parameterized mutations are also more likely to have revertants. This means that for a given set of mutation rates, a population can achieve a steady-state ratio of alternate configurations that can be successful in alternate future environments. Because this occurs through stochastic processes, it means that populations can maintain these ratios without having to coordinate or communicate among the members of the population.

Thus, an organism's genome structure can provide *mutational hedges*—alternate configurations for possible future environments—to exist and be continually replenished as subpopulations in the current environment. This dynamic is a significant driver of the reduced beneficial/deleterious ratio of mutations in stable environments. As shown in Bartlett[13], as a parameterized trait approaches an environmental maximum, most mutations *of* that trait will be *away* from that maximum point, thus increasing the rate of deleterious mutations. However, most of these are only deleterious in the context of the current environment, not necessarily deleterious in the wider sense of biophysical integrity. Thus, they are introducing hedges into the population against potential environmental changes.

Thus, the mutational system as it exists in nature (rather than as conceived of by the modern synthesis) provides the ability for the mutation system itself to perform evolutionary bet hedging.

Interestingly, even while the tendency of traditional evolutionary theory to underemphasize mutational structure was noted by some (like Ferenci and Maharjan[22]), such accounts stopped short of articulating a notion of bet hedging that fully transcended the modern synthesis. While, for instance, Ferenci and Maharjan's final discussion hinted at the possibility that it "can also be argued that the broad heterogeneity of the mutational spectrum is neither random nor accidental," this idea was not developed beyond general ideas of multiple mutation types avoiding evolutionary dead ends. Researchers within the framework of the modern synthesis did not develop the stronger possibility that a mutational architecture could reliably generate particular pre-adaptive variants at frequencies related to a population's likelihood to need those variants in future environments. The modern synthesis' emphasis on mutations as random with respect to fitness made such an alignment difficult to conceive of and articulate as a population-level betting strategy.

EXPERIMENTAL RESULTS ON HEDGING

There have been a number of experimental studies on hedging, both from the classical perspective of the modern synthesis and the perspective of the extended synthesis.

From the classical perspective (focusing on traits whose geometric mean fitness is high), plant studies have pointed to strong evidence that evolutionary bet hedging is at play in several timing-oriented traits, such as seed dormancy and the timing of flowering.[23] In bacteria, researchers were able to fluctuate an environment enough to induce the evolution of a genotype which the single genotype flipped among multiple phenotypic states.[24] In Fungi, manipulating environments produced organisms selected for a dormancy fraction which matched the frequency of environmental uncertainty rather than the average expected environment.[25]

The classical perspective certainly still remains a fruitful source of ideas for evolutionary research and experimentation. However, the extended synthesis approach opens up new avenues of exploration, where organisms' internal mutation systems have a more active role in producing outcomes.

In bacteria, phase variable genes are one of the most abundant sources of bet hedging. While there are a number of mechanisms behind phase variable genes (several of which are listed in previous sections), the important points are that (a) the variations encode biologically meaningful states for certain environments, (b) the variations are produced genetically, and (c) the variations occur prior to the selection. (a) is what distinguishes this phenomena from the modern synthesis, where mutations are random without any respect to present or future fitness of the organism. (b) is what distinguishes this phenomena from classical bet hedging, where a single genotype produces a varying phenotype. (c) is what makes this bet *hedging* rather than adaptive evolution. Bayliss, Clark, et al.[26] provides a history of experimental results in the study of bacterial phase variable genetics, pointing out the long road to determining that they often function as evolutionary hedges, based on the criteria above.

Similar hedging strategies also occur in phages. Gomez et al.[27] found that T2 phages employed a bet hedging strategy similar to that of bacteria. The *agt* gene can be quickly reversed genetically to either an on or off state, and this happens prior to selection, allowing the survival in environments where that gene is beneficial or deleterious. Because it is reversible, this means that the organism maintains the ability to deploy the opposite hedge in either state. To distinguish these hedging strategies from classical evolutionary bet hedging, we will refer to them as mutational hedging strategies.

Not only are these mutational hedging strategies observed in nature, the *birth* of such mechanisms has also been seen experimentally. Barnett et al.,[28] studied the effect of rapid environmental changes on *Pseudomonas fluorescens*. By continually switching between an environment that favored and disfavored a specific gene, the organism was able to introduce a rapidly-mutating SSR from a cryptic pre-SSR locus.[i] Because the SSR was not a multiple of 3bp, copy number variations would cause frameshifts which turned the gene on and off. The ability for the organism to rapidly switch between on and off states allowed it to maintain alternate versions of itself (hedges) in the environment, and thus better preserve its offspring in the rapidly changing environments.

Interestingly, the opposite effect can also occur. In the face of unchanging environments, organisms may lose their ability to employ mutational hedging strategies. While this has not been investigated specifically in relation to bet hedging, it is well-known that organisms in unchanging environments not only lose genetic material, they specifically lose genetic material that produces mutations in constrained ways. Obligate intracellular bacteria, for instance, have extremely little environmental variance, as their environment is determined by their host. Accordingly, such organisms undergo extreme genome reduction, with much of the reduction focused on cellular mechanisms for mutation induction such as transposable elements and repeat elements.[29] While not specifically looking at SSRs, the authors did note that repeat content was largely missing from these bacteria. Thus, while we cannot say that such organisms definitively lose their bet-hedging abilities, current evidence points in this direction.

THE NEXT FRONTIER: MUTATION RATES AND KELLY BETS

Kelly betting is an optimal long-term betting strategy which has been applied to numerous fields.[30] Essentially, it assigns proper "bet sizes" to each option based on the likelihood of success or failure. Biologists have often utilized Kelly-optimal criteria for analyzing bet hedging in evolution, but only for phenotypic hedging strategies (for example, see Maslov and Sneppen[31]). However, so far, although utilizing Kelly allocations as a tool for analyzing mutations has been proposed[32], it has not yet been utilized in an empirical setting. However, the mathematical treatment given in Rivoire and Leibler[33] provide the most general application of Kelly allocation to biological bet-hedging, and should be the easiest to work into a mutational setting. The mathematics presented by Rivoire and Leibler represent mutation as a degradation of strategy, rather than as a part of the strategy. Nonetheless, the treatment is sufficiently generic that applying it to mutational strategies should not be overly difficult.

An interesting feature of mutational randomness is that it allows a population to maintain a characteristic distribution of variants without communication or coordination among its individual members. Probabilistic transitions among genetic states can produce recurring population-level proportions even though each mutation occurs independently. Thus, the structure of the genome can implicitly encode a population-level betting strategy through its pattern of mutational probabilities.

As such, one could use mutation rates to estimate an implied likelihood of an environment that the organism "thinks" (i.e., implicitly predicts) it will encounter. One could then measure the rate at which this implied likelihood follows varying rates of environmental change experimentally, and determine if and how long it takes for an organism to match its mutation rates to what the environment is presenting.

CONCLUSION

Evolutionary bet hedging has been an important part of evolutionary biology since its introduction in the 1970s. However, under the modern synthesis, only bet hedging strategies which did not involve the production of variant mutations could be considered. With the introduction of the extended evolutionary synthesis, biologists are increasingly recognizing the importance of organismal biology to their own evolution. Applied to bet hedging, both theoretical and experimental evidence shows that organisms can produce genetic variants for likely-encountered environments even when currently living in a different environment, allowing the consideration of mutational hedging strategies in the repertoire of evolutionary causation. While this direction is still in its infancy, mathematical tools like Kelly-optimal analysis can continue to be applied in new ways to the new strategies.

Recognizing mutational architectures as evolutionary hedging mechanisms expands the explanatory scope of bet-hedging theory while preserving the successful insights of classical models.

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CONFLICT OF INTEREST

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i The starting sequence of the pre-SSR was GGTGCCCGGTG. Presumably, the starting GGTG and the ending GGTG provided the basis for the initial slipped-strand mispairing, causing the sequence GGTGCCC to function as an SSR. It is unclear if this predisposition to become an SSR is an intrinsic teleonomic feature of the gene, if it was merely exploited by cellular machinery (such as epigenetic markers), or if a happenstance mutation enabled it to function in this way.

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