# Evolution's False Predictions: A Theory Evaluation Approach

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## Abstract

Charles Darwin's theory of evolution became dominant in the twentieth century and has exerted significant influence on scientific thought. This has led to an extensive apologetics literature arguing for the veracity of evolution. This literature focuses on evidences for evolution and, due to a range of factors, is less focused on contrary evidences. Here I evaluate evolutionary theory using a sampling of its false predictions. I use this sample as a framework from which to analyze evolutionary theory according to eight theory virtues: evidential accuracy, causal adequacy, internal consistency, internal coherence, universal coherence, simplicity, durability, and fruitfulness. The outcomes of the false predictions reveal shortcomings in these theory virtues.

**Keywords**: Evolution, Darwin, Predictions; Theory evaluation; Theory virtues

## Introduction

Charles Darwin presented his theory of evolution in 1859. While many scientists accepted Darwin's theory in the nineteenth century, evolution became overwhelmingly dominant in the twentieth century. The consensus position emerged that evolution was beyond reasonable doubt. A substantial apologetics literature surveyed and systematized the various evidences and arguments in support of this consensus position. (Avise; Berra; Campbell; Carroll; Coyne; Dobzhansky; Eldredge; Futuyma; Gould; Lewontin; Mayr; Monod; Ridley; Williams) Historians, philosophers and theologians followed suite, (Barbour; Haught; Kitcher; Murphy; Polkinghorne; Ruse; Sober) as exemplified by Peter Bowler's *Evolution: The History of an Idea* which informed the reader that by mid-century evolutionary theory had "come of age" and the "history of life on earth was now known." (Bowler, 347) The arts contributed to the consensus as well, such as in the widely acclaimed play and film *Inherit the Wind* which, following the historical 1925 Monkey Trial, equated evolutionary thought with enlightenment, and its opposition with bigotry. (Darrow and Bryan, 75, 76, 79, 83, 87, 177, 299; Lawrence and Lee, 62) Evolutionist Stephen J. Gould would later famously label evolution skepticism as perverse. (Gould, 254)

Against this social and value-laden backdrop it is not surprising that scientific criticism of evolution would become a third rail in the natural sciences. Individual sub-hypotheses could safely be questioned. But criticism of evolution, broadly-construed, could be career-ending. (Bergman and Wirth; Hunter, 105-6) The result is an imbalance in the scholarly literature. Peer reviewed journals have accumulated, over many decades, vast amounts of "normal science," to use Kuhn's terminology. (Kuhn, 10) That is, works which unquestioningly presuppose the reigning paradigm. On the other hand, there is little systematic, predictions-based, paradigm-critical, analysis.

Here, I contribute to the empirically based, evolution criticism literature, with a predictions-based analysis. It is not controversial that a great many predictions made by evolution have been found to be false. There is less consensus, however, on how to interpret these falsifications. In logic, when a hypothesis predicts or entails an observation that is discovered to be false, then by modus tollens the hypothesis is concluded to be false. Not so in science.

When a scientific theory makes a prediction that is discovered to be false, the theory may simply be modified to accommodate the new finding. Broad, umbrella theories, such as evolution, are particularly amenable to adjustments. Evolution states that chance events and naturalistic mechanisms are sufficient to explain the origin of species. This is a broad statement capable of generating a wide variety of specific explanations about how evolution is supposed to have occurred. In fact, evolutionists often disagree about these details. So, if one evolutionary explanation, dealing with a particular aspect of evolution, makes false predictions, there often are alternative explanations available to explain that aspect of evolution. Obviously, the theory of evolution itself is not harmed simply because one particular sub-hypothesis is shown to be wrong.

Failed expectations are not necessarily a problem for a theory. (Lakatos, 215ff) In fact evolutionists argue that false predictions made by the theory of evolution are not problems, but

rather are signs of scientific progress. With each new finding, evolutionists say, we learn more about how evolution occurred. Nonetheless, it is worthwhile to review a theory's false predictions. A theory's track record can be highly informative. The history of false predictions generated by a theory tells us about its strengths and weaknesses. For instance, a false prediction can lead to new discovery and knowledge, or it can lead to a loss of theory virtues.

Therefore, in this paper I use a sampling from the history of evolution's false predictions as a framework for theory evaluation. Specifically, I systematize evolution criticism using false predictions to assess the loss of theory virtues. Philosophers of science have long since recognized various virtues that may be important. In addition to prediction accuracy, how consistent, fertile, durable, and simple is the theory? These and other virtues have been proposed and systematized. Recently Keas has further systematized theory evaluation with his proposed four-class taxonomy of theory virtues. These virtues reveal additional dimensions of evolution's failure as a scientific theory. Consider this subset of Keas' virtues (Keas) for theory T:

| Virtue | Description |
|---|---|
| Evidential accuracy | A theory (T) fits the empirical evidence well. |
| Causal adequacy | T's causal factors plausibly produce the effects (evidence) in need of explanation. |
| Internal consistency | T's components are not contradictory. |
| Internal coherence | T's components are coordinated into an intuitively plausible whole; T lacks ad hoc hypotheses—theoretical components merely tacked on to solve isolated problems. |
| Universal coherence | T sits well with (or is not obviously contrary to) other warranted beliefs. |
| Simplicity | T explains the same facts as rivals; but with less theoretical content. |
| Durability | T has survived testing by successful prediction or plausible accommodation of new data. |
| Fruitfulness | T has generated additional discovery by means such as successful novel prediction; unification; and non ad hoc theoretical elaboration. |

I analyze evolution's false predictions on a case-by-case basis to determine what they portend in terms of these eight theory virtues. The predictions examined in this article were selected according to several criteria. They cover a wide spectrum of evolutionary theory, reflecting major tenets of evolutionary thought. They were widely held by the consensus rather than reflecting one viewpoint of several competing viewpoints. Each prediction was a natural and fundamental expectation of the theory of evolution and constituted mainstream evolutionary science. Furthermore, the selected predictions are not vague but rather are specific and can be objectively evaluated. They have been tested and evaluated and the outcome is not controversial or in question. And finally, the predictions have implications for theory virtues, as discussed in the conclusions.

This article does not maintain that the predictions presented are the only fundamental predictions of evolution, or that evolution does not have successful predictions. Those are well documented in the literature. Nor does this article maintain that the predictions presented, though false, have

not served to produce research. Also, this article does not maintain that these false predictions cannot be remedied or reversed by future scientific findings.

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## Responses to common objections

This section examines various concerns evolutionists often have regarding their theory's false predictions. Responses to each concern are given below.

### False predictions often have led to productive research

This falls under theory virtue of fruitfulness. Productive research can come from a great variety of scientific and nonscientific motivations, including false predictions. However, as we shall see, many of evolution's false predictions have not been fruitful.

### Evolutionists have fixed these false predictions

A proponent of a theory, given sufficient motivation, can explain all kinds of contradictory findings. (Quine, 40) Typically, however, there is a price to be paid in terms of theory virtues. For example, the theory may forfeit internal consistency, become more complex, and so forth.

### Ad hominem and denial

Criticism of evolution draws heated responses, and personal attacks are common. Such attacks, however, do not change the fact that evolution has generated many false predictions. Also, evolutionists sometimes ignore or deny the unexpected findings. They attempt to discredit the facts, referring to them as "tired old arguments," or "fallacies" without following up such criticisms with supporting details.

### Falsificationism is flawed

It has been argued that in order to qualify as science, ideas and theories need to be falsifiable. Also, falsified predictions are sometimes used to argue a theory is false. Such naïve falsificationism is flawed (Popper) and not used here. A single false prediction of evolutionary theory does not necessarily demonstrate that evolution is not science or that evolution is false.

False predictions are valuable in judging the quality of a theory, its explanatory power and veracity, and for improving our scientific understanding in general. Nonetheless, evolutionists sometimes reject any mention of their theory's false predictions as mere naïve falsificationism. The failures of naïve falsificationism do not give evolutionists a license to ignore substantial and fundamental failures of their theory.

### If there are so many problems evolution would have been toppled

This objection falls under the category of naïve falsificationism. Science is an active process. New evidence is processed, and theories are adjusted accordingly. But science can also be a conservative process, sustaining substantial problems before reevaluating a theory. Science also can be influenced and driven by non-empirical factors. Therefore, the reevaluation of a theory takes time. The fact that there are problems is no guarantee a theory will have been toppled. (Lakatos, 110ff; Chalmers, 66)

**Those quoted believe in evolution**

Many scientists doubt evolution, but they are not cited or quoted in this paper. Only material from evolutionists is used to illustrate that even adherents to the theory agree that the predictions are false.

**These falsifications will be remedied in the future**

As scientists, we need to evaluate scientific theories according to the currently available data. No one knows what future data may bring, and the claim that future data will rescue evolution is ultimately circular.

**There is no better alternative**

One way to evaluate a theory is to compare it to alternative explanations. This approach has the advantage of circumventing the difficulties in evaluating scientific theories. But of course, any such comparison will crucially depend on what alternative explanations are used in the comparison. If care is not taken good alternatives can be misrepresented or even omitted altogether. And of course, there may be alternatives not yet conceived. (van Fraassen, 131ff; Stanford) In any case, the success or failure of evolution's predictions depends on the science, not on alternative explanations.

**No one believes these predictions anymore**

Yes, this is the point, no one believes in the predictions because they are false. The fact that no one believes in the predictions does not imply there are no consequences of the failure. We can learn from this failed track record as it has implications for evolution's virtues, as discussed in the Conclusions.

**What about all the successful predictions?**

Theories that ultimately are overwhelmingly rejected may nonetheless produce successful predictions. For example, Ptolemy's geocentric model of the cosmos could predict accurately celestial motion and events, with no greater complexity than Copernicus' heliocentric model. (Kuhn, 169)

**These falsified predictions are not necessary predictions of evolutionary theory. They merely reflect isolated instances of a practitioner's surprise over specific sets of data.**

The predictions were considered to be necessary when they were held. And they represented consensus evolutionary science at the time they were held. They are documented in peer-reviewed research papers, popular literature authored by leading evolutionists, and interviews of leading evolutionists. They were not merely held by a few, individual evolutionists. Nor were they one of several possible competing predictions. That these predictions are not now considered to be necessary predictions of evolution is a reflection of the malleability of

evolutionary theory and is a reminder of why a history of evolution's false predictions is important.

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## False Prediction 1: The DNA code is not special

Shortly after the discovery of the DNA code, which is used in cells to construct proteins, evolutionists began theorizing how it evolved. The same code was found in very different species which means that the same code was present in their distant, common ancestor. So, the DNA code arose early in evolutionary history and remained essentially unchanged thereafter. And since it arose so early in evolutionary history, in the first primitive cell, the code must not be unique or special. For how could such a code have evolved so early in the history of life? As Nobel Laureate Francis Crick wrote in 1968, "There is no reason to believe, however, that the present code is the best possible, and it could have easily reached its present form by a sequence of happy accidents." (Crick, 377) Although Crick had no empirical evidence that the code "could have easily reached its present form by a sequence of happy accidents," his claim was influential. For example, one widely used undergraduate molecular biology text later explained, "The code seems to have been selected arbitrarily (subject to some constraints, perhaps)." (Alberts et. al., 9) And an evolution textbook further explained, "The code is then what Crick called a 'frozen accident.' The original choice of a code was an accident; but once it had evolved, it would be strongly maintained." (Ridley, 48)

In other words, somehow the DNA code arose accidentally, but it has little or no special or particular properties. But we now know that the code's arrangement uniquely reduces the effects of mutations and reading errors. As one research study concluded, the DNA code is "one in a million" in terms of efficiency in minimizing these effects. (Freeland and Hurst, 238) This problem later became even worse—by 14 orders of magnitude:

> We show that the probability of obtaining a genetic code with a cost as low as that of the SGC [standard genetic code] by chance is ca. $10^{-20}$, much lower than the previous estimate. (Omachi, Saito and Furusawa)

Similarly, other studies have discovered unique and special properties of the code. One found that the DNA code is a very rare code, even when compared to other codes which already have the error correcting capability. (Itzkovitz and Alon) Another found that the code does not optimize merely one function, but rather optimizes "a combination of several different functions simultaneously." (Bollenbach, Vetsigian and Kishony, 403) As one paper concluded, the code's properties were "unexpected and still cry out for explanation." (Vetsigian, Woese and Goldenfeld, 10696)

So how did the DNA code evolve? This question has caused yet more problems for evolution. Evolutionists have proposed a wide range of ideas. These include, in addition to Crick's frozen accident, the stereochemical, the coding coenzyme handle, the coevolution, the four-column theory, and the error minimization hypotheses. (Kun and Radványi) But none of these hypotheses succeed in explaining the origin of the code.

For example, Crick's frozen accident hypotheses lacks sufficient detail. To say that it is not causally adequate would be an understatement. Even evolutionists refer to it as a "non-explanation." (Koonin and Novozhilov, 102) But could not the code have evolved to minimize error, which surely would increase fitness? Aside from the obvious problem that code changes

would wreak havoc on the synthesis of proteins based on existing genes, today's standard genetic code in fact does not appear to minimize absolutely the translation error. Depending on the specific error minimization criteria used, (Caldararo and Di Giulio) there are better codes and the standard code is not on a local fitness peak. Furthermore, the standard code displays a structure and patterns that are not related to error minimization. The structure and patterns would not be expected under the error minimization hypothesis.

The failure of the many different code evolution hypotheses has left evolutionists considering the possibility that the code evolved via some combination of those hypotheses. But this menagerie approach makes the theory even more ad hoc and complex. These problems, in addition to the lack of causal adequacy has left evolutionists admitting that the origin of the code is a "notoriously difficult problem." (Kun and Radványi, 217) Consider these admissions:

> At the heart of this problem is a dreary vicious circle: what would be the selective force behind the evolution of the extremely complex translation system before there were functional proteins? And, of course, there could be no proteins without a sufficiently effective translation system. A variety of hypotheses have been proposed in attempts to break the circle but so far none of these seems to be sufficiently coherent or enjoys sufficient support to claim the status of a real theory. … Summarizing the state of the art in the study of the code evolution, we cannot escape considerable skepticism. It seems that the two-pronged fundamental question: "why is the genetic code the way it is and how did it come to be?", that was asked over 50 years ago, at the dawn of molecular biology, might remain pertinent even in another 50 years. (Koonin and Novozhilov, 110)

> To be honest, we are stuck. … Most papers on the origin of the genetic code are reviews (like this) and not original research. It seems that the field has been stalled. (Kun and Radványi, 225)

The prediction that the DNA code is not special not only has been falsified, but it has also revealed a host of concerns with evolutionary theory, such as complexity, ad hoc explanations, and a lack of causal adequacy.

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## False Prediction 2: The cell's fundamental molecules are universal

In addition to the DNA code, there are other fundamental molecular processes that appear to be common to all life. One intriguing example is DNA replication which copies both strands of the DNA molecule, but in different directions. Evolution predicts these fundamental processes to be common to all life. Indeed, this was commonly claimed to be an important successful prediction for the theory. As Niles Eldredge explained, the "underlying chemical uniformity of life" was a severe test that evolution passed with flying colors. (Eldredge, 41) Likewise Christian de Duve declared that evolution is in part confirmed by the fact that all extant living organisms function according to the same principles. (de Duve, 1) And Michael Ruse concluded that the essential macromolecules of life help to make evolution beyond reasonable doubt. (Ruse, 4)

But this conclusion that the fundamental molecular processes within the cell are common to all species was superficial. In later years, as the details were investigated, important differences between species emerged. For example, key DNA replication proteins surprisingly "show very little or no sequence similarity between bacteria and archaea/eukaryotes." (Leipe, Aravind and Koonin, 3389) Also different DNA replication processes have been discovered. These results were not what were expected:

> In particular, and counter-intuitively, given the central role of DNA in all cells and the mechanistic uniformity of replication, the core enzymes of the replication systems of bacteria and archaea (as well as eukaryotes) are unrelated or extremely distantly related. Viruses and plasmids, in addition, possess at least two unique DNA replication systems, namely, the protein-primed and rolling circle modalities of replication. This unexpected diversity makes the origin and evolution of DNA replication systems a particularly challenging and intriguing problem in evolutionary biology. (Koonin, 1)

In addition to replication proteins and processes, important differences across species have also been found in the cell's fundamental plasma membrane and lipid biosynthesis pathways. (de Farias, Jose, and Prosdocimi) Given this list of unexpected fundamental differences across species, some evolutionists are reconsidering the assumption that all life on Earth shares the same basic molecular architecture and biochemistry, and instead examining the possibility of independent evolution, and multiple origins of life's different lineages. (Cleland; Leipe, Aravind and Koonin; de Farias, Jose, and Prosdocimi) This, of course, would constitute a fundamental departure from evolutionary thought and common descent, introducing an internal inconsistency. Evolution does not pass this test with flying colors, as Eldredge put it, but in fact fails this test. And rather than evolution's common descent explaining, in a stroke, a wide range of observations, evolution takes on arbitrary and contradictory components.

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### False Prediction 3: Mutations are not adaptive

In the twentieth century, the theory of evolution predicted that mutations are not adaptive or directed. In other words, mutations were believed to be random with respect to the needs of the individual. As Julian Huxley put it, "Mutation merely provides the raw material of evolution; it is a random affair, and takes place in all directions. … in all cases they are random in relation to evolution. Their effects are not related to the needs of the organisms." (Huxley, 36) Or as Jacques Monod explained:

> chance alone is at the source of every innovation, of all creation in the biosphere. Pure chance, absolutely free but blind, at the very root of the stupendous edifice of evolution: this central concept of modern biology is no longer one among other possible or even conceivable hypotheses. It is today the sole conceivable hypothesis, the only one that squares with observed and tested fact. And nothing warrants the supposition—or the hope—that on this score our position is likely ever to be revised. (Monod, 112)

Furthermore, Ronald Fisher's seminal theoretical work, modeling the evolutionary process, were based on the premise that mutations are "random with respect to the needs of organisms." (Orr, 121) This fundamental prediction persisted for decades as a recent paper explained: "mutation is assumed to create heritable variation that is random and undirected." (Chen, Lowenfeld and Cullis, 38)

But that assumption is now known to be false. The first problem is that the mutation rate is adaptive. For instance, when a population of bacteria is subjected to harsh conditions it tends to increase its mutation rate. It is as though a signal has been sent saying, "It is time to adapt." Also, a small fraction of the population increases its mutation rates even higher yet. These hypermutators ensure that an even greater variety of adaptive change is explored. (Foster) Experiments have also discovered that duplicated DNA segments may be subject to higher mutation rates. Since the segment is a duplicate, it is less important to preserve and, like a test bed, appears to be used to experiment with new designs. (Wright)

The second problem is that organisms use strategies to direct the mutations according to the threat. Adaptive mutations have been extensively studied in bacteria. Experiments typically alter the bacteria food supply or apply some other environmental stress causing mutations that target the specific environmental stress. (Burkala, et. al.; Moxon, et. al; Wright) Adaptive mutations have also been observed in yeast (Fidalgo, et. al.; David, et. al.) and flax plants. (Johnson, Moss and Cullis) One experiment found repeatable mutations in flax in response to fertilizer levels. (Chen, Schneeberger and Cullis) Another exposed the flax to four different growth conditions and found that environmental stress can induce mutations that result in "sizeable, rapid, adaptive evolutionary responses." (Chen, Lowenfeld and Cullis, 47)

"Why did it become so important to associate mutation with randomness?" asks one evolutionist, because that view "has been breaking down from the moment it was proposed." (Stoltzfus, 7, 12) Nonetheless, in response to this failed prediction some evolutionists now are saying that evolution somehow created the mechanisms that cause mutations to be adaptive. But this is an ad hoc elaboration on evolutionary theory. First, this explanation lacks parsimony and causal

adequacy, for these mechanisms are far too complex to have arisen by chance factors. Further, this explanation implies foresight and teleology. For even if such a mechanism could somehow arise by chance, it likely would not be positively selected for in meaningful timescales.

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### False Prediction 4: Competition is greatest between neighbors

Darwin's basic theory of evolution, by itself, did not account for the tree-like, hierarchical pattern the species were thought to form. Darwin was keenly aware of this shortcoming and wrestled with it for years. He finally conceived of a solution for why modified offspring would continue to evolve away and diverge from their parents. The principle of divergence, the last major theoretical addition before Darwin published his book, held that competition tends to be strongest between the more closely related organisms. This would cause a splitting and divergence, resulting in the traditional evolutionary tree pattern. (Desmond and Moore 1991, 419-420; Ridley, 378-379)

But no such trend has been observed. In a major study of competition between freshwater green algae species, the level of competition between pairs of species was found to be uncorrelated with the evolutionary distance between the pair of species. As the researchers explained, Darwin "argued that closely related species should compete more strongly and be less likely to coexist. For much of the last century, Darwin's hypothesis has been taken at face value […] Our results add to a growing body of literature that fails to support Darwin's original competition-relatedness hypothesis." (Venail, et.al., 1289, 1296) The team spent months trying to resolve the problem, but to no avail. The National Science Foundation, which supported the research financially, explained that the "research findings contradict one of Darwin's lesser known hypotheses" and that "The researchers … were so uncomfortable with their results that they spent the next several months trying to disprove their own work. But the research held up." (Cimons) Here is how the lead researcher described the results:

> It was completely unexpected. We sat there banging our heads against the wall. Darwin's hypothesis has been with us for so long, how can it not be right? … We went into it assuming Darwin to be right, and expecting to come up with some real numbers for conservationists. When we started coming up with numbers that showed he wasn't right, we were completely baffled. … If Darwin had been right, the older, more genetically unique species should have unique niches, and should compete less strongly, while the ones closely related should be ecologically similar and compete much more strongly—but that's not what happened. We didn't see any evidence of that at all. We found this to be so in field experiments, lab experiments and surveys in 1,200 lakes in North America. … We should be able to look at the Tree of Life, and evolution should make it clear who will win in competition and who will lose. But the traits that regulate competition can't be predicted from the Tree of Life. (Cimons)

Why this long-standing prediction was not confirmed remains unknown. Perhaps the algae species are not diverging in the genes that control competition—maybe they are diverging in other genes. Perhaps nature has much more mutualisms than were expected. Perhaps the algae species are co-evolving so they are more productive as a team than they are individually. (Cimons) But these explanations raise several theory virtue problems, including evidential accuracy, causal adequacy, internal consistency and coherence, simplicity and durability. They also forfeit Darwin's original theoretical work on the principle of divergence, which he needed to explain the relationships between the species.

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## False Prediction 5: Protein evolution

Protein coding genes make up only a small fraction of the genome in higher organisms but their protein products are crucial to the operation of the cell. They are the workers behind a great many tasks in the cell, including digesting food, synthesizing chemicals, structural support, energy conversion, cell reproduction and making new proteins. And like a finely tuned machine, proteins do their work very well. Proteins are ubiquitous in all of life and must date back to the very early stages of evolution. So, evolution predicts that proteins evolved when life first appeared, or not long after. But despite enormous research efforts the science clearly shows that such protein evolution is astronomically unlikely.

One reason the evolution of proteins is so difficult is that most proteins are extremely specific designs in an otherwise rugged fitness landscape. This means it is difficult for natural selection to guide mutations toward the needed proteins. One protein engineering review concluded that "not all protein designs are evolvable." (Brustad and Arnold, 207) In fact, four different studies, done by different groups and using different methods, all report that roughly $10^{70}$ evolutionary experiments would be needed to get close enough to a relatively small, workable, protein before natural selection could take over to refine the protein design. For instance, one study concluded that $10^{63}$ attempts would be required for a relatively short protein. (Reidhaar-Olson) And a similar result ($10^{65}$ attempts required) was obtained by comparing protein sequences. (Yockey) Another study found that from $10^{64}$ to $10^{77}$attempts are required (Axe) and another study concluded that $10^{70}$ attempts would be required. (Hayashi) In that case the protein was only a part of a larger protein which otherwise was intact, thus making for an easier search. Furthermore, these estimates are optimistic because the experiments searched only for single-function proteins whereas real proteins perform many functions. (Donohue)

This conservative estimate of $10^{70}$ attempts required to evolve a simple protein is astronomically larger than the number of attempts that are feasible. And explanations of how evolution could achieve a large number of searches, or somehow obviate this requirement, require the preexistence of proteins and so are circular. For example, one paper estimated that evolution could have made $10^{43}$ such attempts. But the study assumed the entire history of the Earth is available, rather than the limited time window that evolution actually would have had. Even more importantly, it assumed the preexistence of a large population of bacteria (it assumed the earth was completely covered with bacteria). And of course, bacteria contain proteins. Clearly such bacteria would not exist before the first proteins evolved. (Dryden) Even with these unrealistic assumptions the result was twenty-seven orders of magnitude short of the requirement.

Given these several significant problems, the chances of evolution finding proteins from a random start are, as one evolutionist explained, "highly unlikely." (Tautz) Or as another evolutionist put it, "Although the origin of the first, primordial genes may ultimately be traced back to some precursors in the so-called 'RNA world' billions of years ago, their origins remain enigmatic." (Kaessmann, 1323) Therefore, this false prediction reveals a lack of causal adequacy in evolutionary theory.

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## False Prediction 6: Histone proteins cannot tolerate much change

Histones are proteins which comprise the hubs about which DNA is wrapped. They are highly similar across vastly different species which means they must have evolved early in evolutionary history. As one textbook explains, "The amino acid sequences of four histones are remarkably similar among distantly related species. … The similarity in sequence among histones from all eukaryotes indicates that they fold into very similar three-dimensional conformations, which were optimized for histone function early in evolution in a common ancestor of all modern eukaryotes." (Lodish et. al., Section 9.5) And this high similarity among the histones also means they must not tolerate change very well, as another textbook explains: "Changes in amino acid sequence are evidently much more harmful for some proteins than for others. … virtually all amino acid changes are harmful in histone H4. We assume that individuals who carried such harmful mutations have been eliminated from the population by natural selection." (Alberts et. al. 1994, 243)

So, the evolutionary prediction is that in these histone proteins practically all changes are deleterious: "As might be expected from their fundamental role in DNA packaging, the histones are among the most highly conserved eucaryotic proteins. For example, the amino acid sequence of histone H4 from a pea and a cow differ at only at 2 of the 102 positions. This strong evolutionary conservation suggests that the functions of histones involve nearly all of their amino acids, so that a change in any position is deleterious to the cell." (Alberts et. al. 2002, Chapter 4)

This prediction has also been given in popular presentations of the theory: "Virtually all mutations impair histone's function, so almost none get through the filter of natural selection. The 103 amino acids in this protein are identical for nearly all plants and animals." (Molecular Clocks: Proteins That Evolve at Different Rates, 1) Note that this claim, that "Virtually all mutations impair histone's function" was not based on empirical evidence but rather evolutionary theory. If evolution is true, then histone proteins must not tolerate much change.

But this prediction has turned out to be false. An early study suggested that one of the histone proteins could well tolerate many changes. (Agarwal and Behe) And later studies confirmed and expanded this finding: "despite the extremely well conserved nature of histone residues throughout different organisms, only a few mutations on the individual residues (including nonmodifiable sites) bring about prominent phenotypic defects." (Kim, et. al., 5779)

Similarly, another paper documented these contradictory results: "It is remarkable how many residues in these highly conserved proteins can be mutated and retain basic nucleosomal function. … The high level of sequence conservation of histone proteins across phyla suggests a fitness advantage of these particular amino acid sequences during evolution. Yet comprehensive analysis indicates that many histone mutations have no recognized phenotype." (Dai et. al., 1067, 1072) In fact, even more surprising, many mutations actually raised the fitness level. (Dai et. al., 1072) The result is an internal incoherency in evolutionary theory.

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## False Prediction 7: The molecular clock keeps evolutionary time

In the 1960s molecular biologists learned how to analyze protein molecules and determine the sequence of amino acids that comprise a protein. It was then discovered that a given protein molecule varies somewhat from species to species. For example, hemoglobin, a blood protein, has similar function, overall size and structure in different species. But its amino acid sequence varies from species to species. Emile Zuckerkandl and Linus Pauling reasoned that if such sequence differences were the result of evolutionary change occurring over the history of life, then those differences could be used to estimate past speciation events—a notion that became known as the *molecular clock*. (Zuckerkandl and Pauling)

In later decades this concept of a molecular clock, relying on the assumption of a roughly constant rate of molecular evolution, became fundamental in evolutionary biology. (Thomas, et. al.) As the National Academy of Sciences explained, the molecular clock "determines evolutionary relationships among organisms, and it indicates the time in the past when species started to diverge from one another." (Science and Creationism, 3) Indeed the molecular clock has been extolled as strong evidence for evolution and, in fact, a common sentiment has been that evolution was *required* to explain these evidences. As a leading molecular evolutionist wrote, the molecular clock is "*only* comprehensible within an evolutionary framework." (Jukes, 119, emphasis in original) Note the supreme confidence expressed here in not merely a scientific statement of what evolution predicts, but a universal claim of all possible explanations.

The claim that the molecular clock can only be explained by evolution is, however, now a moot point as the mounting evidence shows that molecular differences often do not fit the expected pattern. The molecular clock which evolutionists had envisioned does not exist. The literature is full of instances where the molecular clock concept fails. For example, it was found early on that different types of proteins must evolve at very different rates if there is a molecular clock. For example, the fibrinopeptide proteins in various species must have evolved more than five hundred times faster than the histone IV protein. Furthermore, it was found that the evolutionary rate of certain proteins must vary significantly over time, between different species, and between different lineages. (Thomas, et. al.; Andrews, 28)

The proteins relaxin, superoxide dismutase (SOD) and glycerol-3-phosphate dehydrogenase (GPDH), for example, all contradict the molecular clock prediction. On the one hand, SOD unexpectedly shows much greater variation between similar types of fruit flies than it does between very different organisms such as animals and plants. On the other hand, GPDH shows roughly the reverse trend for the same species. As one scientist concluded, GPDH and SOD taken together leave us "with no predictive power and no clock proper." (Ayala, 73)

Evolutionists found growing evidence that the purported rates of molecular evolution must vary considerably between species for a wide range of taxa, including mammals, arthropods, vascular plants, and even between closely related lineages. For example, consider how one study described the problem:

> The false assumption of a molecular clock when reconstructing molecular phylogenies can result in incorrect topology and biased date estimation. … we can conclude that significant

> rate variation is a common feature of metazoan phylogenies and that the molecular clock cannot be assumed for the invertebrates as a whole. … This study shows that there is significant variation in the rate of molecular evolution between metazoan lineages. This rate variation is observable not only between deep divisions (e.g., among families) but also between closely related genera and species. … we have not found any evidence of systematic variation in rate of molecular evolution that would allow the molecular clock to be "corrected." (Thomas, et.al., 7366, 7367, 7369)

Evolutionists continue to use the molecular clock concept, but the many calibration and correction factors highlight the fact that the sequence data are being fit to the theory rather than the other way around. As one evolutionist warned long ago, "It seems disconcerting that many exceptions exist to the orderly progression of species as determined by molecular homologies; so many in fact that I think the exception, the quirks, may carry the more important message." (Schwabe, 280) The failure of this molecular clock prediction forces evolutionists to appeal to a wide range of explanatory mechanisms, increasing the ad hoc and complex nature of evolutionary theory.

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## False Prediction 8: The pentadactyl pattern and common descent

The pentadactyl structure—five digits (four fingers and a thumb for humans) at the end of the limb structure—is one of the most celebrated proof texts for evolution. The pentadactyl structure is found throughout the tetrapods and its uses include flying, grasping, climbing and crawling. Such diverse activities, evolutionists reason, should require diverse limbs. There seems to be no reason why all should need a five-digit limb. Why not three digits for some, eight for others, 13 for some others, and so forth? And yet they all are endowed with five digits. As Darwin explained, "What can be more curious than that the hand of a man, formed for grasping, that of a mole for digging, the leg of the horse, the paddle of the porpoise, and the wing of the bat, should all be constructed on the same pattern, and should include similar bones, in the same relative positions?" (Darwin, 382)

Such a suboptimal design must be an artefact of common descent—a suboptimal design that was handed down from a common ancestor rather than specifically designed for each species. And the common descent pattern formed by this structure is often claimed as powerful and compelling evidence for evolution. (Berra, 21; Campbell et. al., 509; Futuyma, 47; Johnson and Losos, 298; Johnson and Raven, 286; Mayr, 26) One text calls it a "classic example" of evolutionary evidence. (Ridley, 45)

But this prediction is now known to be false as the digit structure in the tetrapods does not conform to the common descent pattern. In fact, appendages have various digit structures and they are distributed across the species in various ways. This is found both in extant species and in the fossil record. As evolutionist Stephen Jay Gould admitted, "The conclusion seems inescapable, and an old 'certainty' must be starkly reversed." (Gould, 67)

This means that evolutionists cannot model the observed structures and pattern of distribution merely as a consequence of common descent. Instead, a complicated evolutionary history is required (Brown) where the pentadactyl structure re-evolves in different lineages, and appendages evolve, are lost, and then evolve again. And as one recent study concluded, "Our phylogenetic results support independent instances of complete limb loss as well as multiple instances of digit and external ear opening loss and re-acquisition. Even more striking, we find strong statistical support for the re-acquisition of a pentadactyl body form from a digit-reduced ancestor. …The results of our study join a nascent body of literature showing strong statistical support for character loss, followed by evolutionary re-acquisition of complex structures associated with a generalized pentadactyl body form." (Siler and Brown, 2641) Note that this claim of "strong statistical support" for evolutionary changes is within what Kuhn referred to as normal science. That is, the conclusion of strong statistical support entails the premise that evolution is true. There is no such support from a theory-neutral perspective.

This false prediction about the pentadactyl pattern forces evolutionists to construct far more complex and ad hoc evolutionary histories, introducing internal inconsistency. The venerable common descent model may be employed where possible, but otherwise dropped and replaced with circuitous explanations involving any number of de-evolution or re-evolution events of biological structures.

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## False Prediction 9: Serological tests reveal evolutionary relationships

Early in the twentieth century scientists studied blood immunity and how immune reaction could be used to compare species. The blood studies tended to produce results that parallel the more obvious indicators such as body plan. For example, humans were found to be more closely related to apes than to fish or rabbits. These findings were said to be strong confirmations of evolution. In 1923 H. H. Lane cited this evidence as supporting "the fact of evolution." (Lane, 47) Later in the century these findings continued to be cited in support of evolution. (Berra, 19; Dodson and Dodson, 65)

But even by mid-century contradictions to evolutionary expectations were becoming obvious in serological tests. As J.B.S. Haldane admitted in 1949, "Now every species of mammal and bird so far investigated has shown quite a surprising biochemical diversity by serological tests. The antigens concerned seem to be proteins to which polysaccharides are attached." (quoted in Gagneux and Varki, 747)

Indeed, these polysaccharides, or glycans, did not fulfill evolutionary expectations. As one paper explained, glycans show "remarkably discontinuous distribution across evolutionary lineages," for they "occur in a discontinuous and puzzling distribution across evolutionary lineages." (Bishop and Gagneux, 23R) These glycans can be (i) specific to a particular lineage, (i) similar in very distant lineages, (iii) and conspicuously absent from very restricted taxa only.

Here is how another paper described glycan findings: "There is also no clear explanation for the extreme complexity and diversity of glycans that can be found on a given glycoconjugate or cell type. Based on the limited information available about the scope and distribution of this diversity among taxonomic groups, it is difficult to see clear trends or patterns consistent with different evolutionary lineages." (Gagneux and Varki, 747)

To accommodate this false prediction, evolutionists must increasingly turn to complex and ad hoc explanations that are not internally coherent or consistent with the larger evolutionary theory. The glycans present yet another "one-off" problem insofar as evolution is concerned.

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### False Prediction 10: Biology is not lineage specific

Evolution expects the species to fall into a common descent pattern. Therefore, a particular lineage should not have highly differentiated, unique and complex designs, when compared to neighboring species. But highly differentiated, unique designs within a lineage has increasingly been found to be the case, so much so that this pattern now has its own name: lineage-specific biology.

For example, transcription factors are proteins that bind to DNA and help to regulate which genes are expressed. Yet despite the importance of these proteins, their DNA binding sites vary dramatically across different species. As one report explained, "It was widely assumed that, like the sequences of the genes themselves, these transcription factor binding sites would be highly conserved throughout evolution. However, this turns out not to be the case in mammals." (Rewiring of gene regulation across 300 million years of evolution) Evolutionists were surprised when transcription factor binding sites were found to be not conserved between mice and men, (Kunarso et. al.) between various other vertebrates, and even between different species of yeast. So now evolution is believed to have heroically performed a massive, lineage-specific "rewiring" of cellular regulatory networks. (Pennacchio and Visel, 558)

There are many more such examples of lineage-specific biology. Although flowers have four basic parts: sepals, petals, stamens and carpels, the daffodil's trumpet is fundamentally different and must be an evolutionary "novelty." (Oxford scientists say trumpets in daffodils are 'new organ') Out of the thousands of cockroach species, *Saltoblattella montistabularis* from South Africa is the only one that leaps. With its spring-loaded hind legs it accelerates at 23 g's and outjumps even grass hoppers. (Picker, Colville and Burrows) An important immune system component, which is highly conserved across the vertebrates, is mysteriously absent in the Atlantic cod, *Gadusmorhua*. (Star, et. al.) The brown algae, *Ectocarpus siliculosus*, has unique enzymes for biosynthesis and other tasks. (Cock) And the algae *Bigelowiellanatans* has ten thousand unique genes and highly complex gene splicing machinery never before seen in a unicellular organism. It is, as one evolutionist explained, "unprecedented and truly remarkable for a unicellular organism." (Tiny algae shed light on photosynthesis as a dynamic property)

Another fascinating example of lineage-specific biology are the many peculiar morphological and molecular novelties found in disparate, unrelated unicellular protists. As one study concluded, "Both euglenozoans and alveolates have a reputation for 'doing things their own way,' which is to say that they have developed seemingly unique ways to build important cellular structures or carry out molecular tasks critical for their survival. Why such hotspots for the evolution of novel solutions to problems should exist in the tree of life is not entirely clear." (Lukes, Leander and Keeling, 9963)

As with so many of evolution's false predictions, the failure of this prediction does not generate new discovery or otherwise lead to new knowledge. In other words, the failure is not, ultimately, fertile or fruitful, insofar as evolutionary theory is concerned. Instead, it simply is a bare failure, requiring ad hoc and complex components to be tacked onto evolution. Those components do not fit within the larger theory. They merely solve an isolated problem and explain no other important observations beyond the problem at hand. Therefore, this false prediction indicates a

loss of evidential accuracy, causal adequacy, internal coherence, simplicity, duration, and fruitfulness.

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### False Prediction 11: Similar species share similar genes

The only figure in Darwin's book, *The Origin of Species*, showed how he envisioned species branching off of one another. Similar species have a relatively recent common ancestor and have had limited time to diverge from each other. This means that their genes should be similar. Entirely new genes, for instance, would not have enough time to evolve. As François Jacob explained in an influential paper from 1977, "The probability that a functional protein would appear de novo by random association of amino acids is practically zero." (Jacob, 1165) Any newly created gene would have to arise from a duplication and modification of a pre-existing gene. (Zhou et. al.; Ohno) But such a new gene would retain significant similarity to its progenitor gene. Indeed, for decades evolutionists have cited minor genetic differences between similar species as a confirmation of this important prediction. (Berra, 20; Futuyma, 50; Johnson and Raven, 287; Jukes, 120; Mayr, 35)

But this prediction has been falsified as many unexpected genetic differences have been discovered amongst a wide range of allied species. (Pilcher) As much as a third of the genes in a given species may be unique, and even different variants within the same species have large numbers of genes unique to each variant. Different variants of the *Escherichia coli* bacteria, for instance, each have hundreds of unique genes. (Daubin and Ochman)

Significant genetic differences were also found between different fruit fly species. Thousands of genes showed up missing in many of the fruit fly species, and some genes showed up in only a single species. (Levine et. al.) As one science writer put it, "an astonishing 12 per cent of recently evolved genes in fruit flies appear to have evolved from scratch." (Le Page) These novel genes must have evolved over a few million years, a time period previously considered to allow only for minor genetic changes. (Begun et. al.; Chen et. al., 2007)

Initially some evolutionists thought these surprising results would be resolved when more genomes were analyzed. They predicted that similar copies of these genes would be found in other species. But instead, each new genome has revealed yet more novel genes. (Curtis et. al.; Marsden et. al.; Pilcher)

Next evolutionists thought that these rapidly evolving unique genes must not code for functional or important proteins. But again, many of the unique proteins were in fact found to play essential roles. (Chen, Zhang and Long; Daubin and Ochman; Pilcher) As one researcher explained, "This goes against the textbooks, which say the genes encoding essential functions were created in ancient times." (Pilcher) The result of this false prediction is a loss of evidential accuracy, causal adequacy, durability, and fruitfulness.

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## False Prediction 12: MicroRNA

Genes hold information that is used to construct protein and RNA molecules which do various tasks in the cell. A gene is copied in a process known as transcription. In the case of a protein-coding gene, the gene copy may be edited before it is then converted into a protein in a process known as translation. All of this is guided by elaborate regulatory processes that occur before, during and after this sequence of transcription, editing and translation.

For instance, some of our DNA, which evolutionists thought to be of little use, actually has a key regulatory role. This DNA is transcribed into strands of about 20 nucleotides, known as microRNA. These short snippets bind and interfere with the gene copy when the production of the gene needs to be slowed.

MicroRNAs can also help to modify the translation process by stimulating programmed ribosomal frameshifting. Two microRNAs attach to the gene copy resulting in a pseudoknot, or triplex, RNA structure form which causes the reading frameshift to occur. (Belew)

MicroRNAs do not only come from a cell's DNA. MicroRNAs can also be imported from nearby cells, thus allowing cells to communicate and influence each other. This helps to explain how cells can differentiate in a growing embryo according to their position within the embryo. (Carlsbecker)

MicroRNAs can also come from the food we eat. In other words, food not only contains carbohydrates, proteins, fat, minerals, vitamins and so forth, it can also contain information—in the form of these regulatory snippets of microRNA—which regulate our gene production. (Zhang)

While microRNAs regulate the production of proteins, the microRNAs themselves also need to be regulated. So, there is a network of proteins that tightly control microRNA production as well as their removal. "Just the sheer existence of these exotic regulators," explained one scientist, "suggests that our understanding about the most basic things—such as how a cell turns on and off—is incredibly naïve." (Hayden)

Two basic predictions that evolutionary theory makes regarding microRNAs are that (i) like all of biology, they arose gradually via randomly occurring biological variation (such as mutations) and (ii) as a consequence of this evolutionary origin, microRNAs should approximately form evolution's common descent pattern. Today's science has falsified both of these predictions.

MicroRNAs are far too unlikely to have gradually evolved via random mutations. This is because too many simultaneous or nearly simultaneous mutations are required. Without the prior existence of genes and the protein synthesis process microRNAs would be useless. And without the prior existence of their regulatory processes, microRNAs would wreak havoc.

Given the failure of the first prediction, it is not surprising that the second prediction has also failed. The microRNA genetic sequences do not fall into the expected common descent pattern. That is, when compared across different species, microRNAs do not align with the hypothetical

evolutionary tree. As one scientist explained, “I've looked at thousands of microRNA genes and I can’t find a single example that would support the traditional [evolutionary] tree.” (Dolgin, 460)

While there remain questions about these new phylogenetic data, “What we know at this stage,” explained another evolutionist, “is that we do have a very serious incongruence.” (Dolgin, 462) In other words, different types of data report very different evolutionary trees. The conflict is much greater than normal statistical variations.

“There have to be,” added another evolutionist, “other explanations.” (Dolgin, 462) For example, perhaps microRNAs evolve in some unexpected way. In any case, microRNAs are yet another example of evidence that does not fit evolutionary expectations. Once again, the theory will need to be modified in complex ways to fit the new findings.

In the meantime, scientists are finding that imposing the common descent pattern, where microRNAs must be conserved across species, is hampering scientific research:

> These results highlight the limitations that can result from imposing the requirement that miRNAs be conserved across organisms. Such requirements will in turn result in our missing bona fide organism-specific miRNAs and could perhaps explain why many of these novel miRNAs have not been previously identified. (Londin, E1111)

Evolutionary theory has been limiting the science. While the common descent pattern has been the guide since the initial microRNA studies, these researchers “liberated” themselves from that constraint, and this is leading to good scientific progress:

> In the early days of the miRNA field, there was an emphasis on identifying miRNAs that are conserved across organisms … Nonetheless, species-specific miRNAs have also been described and characterized as have been miRNAs that are present only in one or a few species of the same genus. Therefore, enforcing an organism-conservation requirement during miRNA searches is bound to limit the number of potential miRNAs that can be discovered, leaving organism- and lineage-specific miRNAs undiscovered. In our effort to further characterize the human miRNA repertoire, we liberated ourselves from the conservation requirement … These findings strongly suggest the possibility of a wide-ranging species-specific miRNA-ome that has yet to be characterized. (Londin, E1113)

The two microRNA predictions have been falsified and the failure has not been fruitful as the expectation has hampered the scientific research of how microRNAs work. The failed predictions also indicate weaknesses in three more theory virtues: evidential accuracy, causal adequacy, and durability.

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### False Prediction 13: Genomic features are not sporadically distributed

A fundamental concept in evolutionary theory is the inheritance of genetic variations via blood lines. (Forbes) This so-called vertical transmission of heritable material means that genes, and genomes in general, should fall into a common descent pattern, consistent with the evolutionary tree. Indeed, such genes are often cited as a confirmation of evolution. But as more genomic data have become available, an ever-increasing number of genes have been discovered that do not fit the common descent pattern because they are missing from so many intermediate species. (Andersson and Roger 2002; Andersson and Roger 2003; Andersson 2005; Andersson, Sarchfield and Roger 2005; Andersson 2006; Andersson et. al. 2006; Andersson 2009; Andersson 2011; Haegeman, Jones and Danchin; Katz; Keeling and Palmer; Richards et. al 2006a; Richards et. al 2006b; Takishita et. al.; Wolf et. al.)

This type of pattern is also found for genome architecture features that are sporadically distributed and then strikingly similar in distant species. In fact, these similarities do not merely occur twice, in two distant species. They often occur repeatedly in a variety of otherwise distant species. This is so widespread that evolutionists have named the phenomenon "recurrent evolution." As one paper explains, the recent explosion of genome data reveal "strikingly similar genomic features in different lineages." Furthermore, there are "traits whose distribution is 'scattered' across the evolutionary tree, indicating repeated independent evolution of similar genomic features in different lineages." (Maeso, Roy and Irimia, 486)

One example is the uncanny similarity between the kangaroo and human genomes. As one evolutionist explained: "There are a few differences, we have a few more of this, a few less of that, but they are the same genes and a lot of them are in the same order. We thought they'd be completely scrambled, but they're not." (Taylor)

It is now well recognized that this prediction has failed: "Vertical transmission of heritable material, a cornerstone of the Darwinian theory of evolution, is inadequate to describe the evolution of eukaryotes, particularly microbial eukaryotes." (Katz, 1893) And these sporadic, patchy patterns require complicated and ad hoc scenarios to explain their origin. As one paper explained, the evolution of a particular set of genes "reveals a complex history of horizontal gene transfer events." (Wolf et. al., 689) The result is that any pattern can be explained by arranging the right mechanisms. Features that are shared between similar species can be interpreted as "the result of a common evolutionary history," and features that are not can be interpreted as "the result of common evolutionary forces." (Maeso, Roy and Irimia, 496)

These common evolutionary forces are complex and must have been created by evolution. They can include horizontal (or lateral) gene transfer, gene loss, gene fusion, and even unknown forces. For instance, one study concluded that the best explanation for the pattern of a particular gene was that it "has been laterally transferred among phylogenetically diverged eukaryotes through an unknown mechanism." (Takishita et. al., 2) Even with the great variety of mechanisms available, there still remains the unknown mechanism. Hence, this failed prediction reveals a weakness in causal adequacy, as well as evidential accuracy, internal consistency and coherence, simplicity, and durability.

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## False Prediction 14: Gene and host phylogenies are congruent

Evolution predicts that genetic change drives evolutionary change. Genetic changes that confer improved fitness are more likely to be selected and passed on. This means that evolutionary trees based on comparisons of genes should be similar, or congruent, with evolutionary trees based on comparisons of the entire species. Simply put, gene trees and species trees should be congruent. But while this has often been claimed to be a successful prediction, it is now known to be false. As one study explained, "Perhaps most unexpected of all is the substantial decoupling, now known in most, although not all, branches of organismal life, between the phylogenetic histories of individual gene families and what has generally been accepted to be the history of genomes and/or their cellular or organismal host lineages." (Ragan, McInerney and Lake, 2169)

The molecular and the visible (morphological) features often indicate "strikingly different" evolutionary trees that cannot be explained as due to different methods being used. (Lockhart and Cameron, 84) Making sense of these differences between the molecular and the morphological features has become a major task, (Gura) so common that it now has its own name: reconciliation. (Stolzer, et. al.)

"The growing gap between molecular analyses and the fossil record," concluded one study, "is astounding." (Feduccia, 175) Instead of a single evolutionary tree emerging from the data, there is a wealth of competing evolutionary trees. (de Jong) And while the inconsistencies between molecular and fossil data were, if anything, expected to be worse with the more ancient, lower, parts of the evolutionary tree, the opposite pattern is observed. As one study explained, "discord between molecular divergence estimates and the fossil record is pervasive across clades and of consistently higher magnitude for younger clades." (Ksepka, Ware and Lamm, 3)

Furthermore, the substantial increase in available genetic data has not helped, as incongruence in the phylogenomics era "remains pervasive." It is thought that a whole host of factors, "such as incomplete lineage sorting, horizontal gene transfer, hybridization, introgression, recombination and convergent molecular evolution, can lead to gene phylogenies that differ from the species tree." (Steenwyk, et. al., 1) In other words, evolutionists have been surprised and astounded by the scientific evidence, but nonetheless they have a host of explanatory mechanisms to draw upon. The price to be paid for incorporating such explanatory mechanisms, however, is loss of internal coherence and simplicity.

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### False Prediction 15: Gene phylogenies are congruent

Just as evolution predicts that gene trees and species trees should be congruent, it also predicts that different gene trees should be congruent. In 1982 David Penny and co-workers tested this prediction. They wrote that "The theory of evolution predicts that similar phylogenetic trees should be obtained from different sets of character data." (Penny, Foulds and Hendy, 197) Their character data came from five different proteins and they concluded "there is strong support from these five sequences for the theory of evolution." (Penny, Foulds and Hendy, 199)

But in later years, as more genetic data became available, it was clear that different genes led to very different evolutionary trees. As one study explained, the sequences of genes, "often disagree and can seldom be proven to agree." (Doolittle and Bapteste, 2043) It is now well understood that "Gene and genome trees conflict at many levels" (Haggerty, et. al., 2209) and that "Incongruence between gene trees is the main challenge faced by phylogeneticists in the genomic era." (Galtier and Daubin, 4023) For evolutionists this failed prediction will require more complicated models of evolutionary history. As Penny now writes, he is "not rejecting the tree per se but enriching the tree concept into a network." (Penny, 3) Therefore, this false prediction has led to a loss of evidential accuracy, causal adequacy, internal coherence, simplicity, and durability.

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### False Prediction 16: The species should form an evolutionary tree

Ever since Darwin the universal evolutionary tree has been a unifying principle in evolutionary biology. Evolution predicted that this universal tree can be derived by arranging the species according to their similarities and differences. And as more data became available, particularly from the dramatic breakthroughs in molecular biology in the latter half of the twentieth century, expectations were high for the determination of this tree. As one paper explains, "Once universal characters were available for all organisms, the Darwinian vision of a universal representation of all life and its evolutionary history suddenly became a realistic possibility. Increasing reference was made to this universal, molecule-based phylogeny as the "comprehensive" tree of the "entire spectrum of life." (O'Malley and Koonin, 2) But those expectations were dashed: "By the mid-1980s there was great optimism that molecular techniques would finally reveal the universal tree of life in all its glory. Ironically, the opposite happened." (Lawton)

As one study explained, the problem is so confusing that results "can lead to high confidence in incorrect hypotheses." (Dávalos, 991) And although evolutionists thought that more data would solve their problems, the opposite has occurred. With the ever-increasing volumes of data, incongruence between trees "has become pervasive." (Dávalos, 991) As another researcher explained, "Phylogenetic incongruities can be seen everywhere in the universal tree, from its root to the major branchings within and among the various taxa to the makeup of the primary groupings themselves." (Woese, 6854) These incongruities are not minor statistical variations and the general failure to converge on a single topology has some researchers concluding that the data do not support "tree-thinking." (Bapteste, et. al., 1) Nor are these incongruities limited to protein-coding genes. As one researcher commented, "I've looked at thousands of microRNA genes, and I can't find a single example that would support the traditional [evolutionary] tree." (Dolgin, 460)

These incongruities have forced evolutionists to filter the data carefully in order to obtain evolutionary trees. As one paper explains, "selecting genes with strong phylogenetic signals and demonstrating the absence of significant incongruence are essential for accurately reconstructing ancient divergences." (Salichos and Rokas, 327) But this raises the question of whether the resulting tree is real: "Hierarchical structure can always be imposed on or extracted from such data sets by algorithms designed to do so, but at its base the universal TOL [tree of life] rests on an unproven assumption about pattern that, given what we know about process, is unlikely to be broadly true." (Doolittle and Bapteste, 2043).

A large-scale study of plants showed that they also do not reveal an evolutionary tree pattern, but rather comprise discrete clusters, leaving large areas of morphospace unoccupied. (Clark) Furthermore, the fossil data, rather than filling in an evolutionary tree structure, confirm the discrete pattern:

> The phylomorphospace constructed from both fossil and extant taxa shows that fossil taxa do not alter the fundamental pattern of morphospace occupation seen in extant taxa and none lie beyond the regions of morphospace occupation circumscribed by the extant phylomorphospace. (Clark)

The researchers hypothesize that unknown evolutionary innovations occurred in the gaps between clusters, and left no fossil evidence:

> Here we show that the living divisions comprise discrete clusters within morphospace, separated largely by reproductive innovations, the extinction of evolutionary intermediates and lineage-specifc evolution. (Clark)

Some evolutionists have contended that the failure of the evolutionary tree prediction, more generally, is due to ubiquitous horizontal (or lateral) gene transfer between species, resulting in a network model rather than a tree model. (Blais and Archibald) But as with unknown evolutionary innovations occurring in gaps, ubiquitous horizontal gene transfer is a theory-driven inference rather than an empirical observation. Furthermore, the resulting network model has substantially greater complexity than the tree model and can fit a much greater range of outcomes. Therefore, this is another example of a false prediction that resulted in a loss of simplicity, internal coherence, and fruitfulness.

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## False Prediction 17: Complex structures evolved from simpler structures

"To suppose that the eye," wrote Darwin, "could have been formed by natural selection, seems, I freely confess, absurd in the highest possible degree." But Darwin argued that we must not be misled by our intuitions. Given natural selection operating on inheritable variations, some of which are useful, then, if a sequence of numerous small changes from a simple and imperfect eye to one complex and perfect can be shown to exist, and if the eye is somehow useful at each step, then the difficulty is resolved. (Darwin, 143) The key was to identify "a long series of gradations incomplexity, each good for its possessor" which could lead to "any conceivable degree of perfection." (Darwin, 165)

But ever since Darwin the list of complex structures in biology, for which no "series of gradations in complexity" can be found, has continued to grow longer. Both the fossil record and genomic data reveal high complexity in lineages where evolution expected simplicity. As one evolutionist explained:

> It is commonly believed that complex organisms arose from simple ones. Yet analyses of genomes and of their transcribed genes in various organisms reveal that, as far as protein-coding genes are concerned, the repertoire of a sea anemone—a rather simple, evolutionarily basal animal—is almost as complex as that of a human. (Technau, 1184)

Early complexity is also evident in the cell's biochemistry. For instance, kinases are a type of enzyme that regulate various cellular functions by transferring a phosphate group to a target molecule. Kinases are widespread across eukaryote species and so they must persist far down the evolutionary tree. And the similarity across species of the kinase functions, and their substrate molecules, means that these kinase substrates must have remained largely unchanged for billions of years. In other words, the complex regulatory actions of the kinase enzymes must have been present early in the history of life. (Diks)

This is by no means an isolated example. Histones are a class of eukaryote proteins that help organize and pack DNA, and the gene that codes for histone IV is highly conserved across species. So again, the first histone IV must have been very similar to the versions we see today. An example of early complexity in eyes is found in the long-extinct trilobite. It had eyes that were perhaps the most complex ever produced by nature. One expert called them "an all-time feat of function optimization." (Levi-Setti, 29) Reviewing the fossil and molecular data, one evolutionist explained that there is no sequential appearance of the major animal groups "from simpler to more complex phyla, as would be predicted by the classical evolutionary model." (Sherman, 1873)

One team of evolutionists concluded, "comparative genomics has confirmed a lesson from paleontology: Evolution does not proceed monotonically from the simpler to the more complex." (Kurland, 1011) So whereas evolutionists once viewed evolution as gradually producing increasing complexity, now evolution can produce complexity *tabula rasa*, in the absence of any such gradual increase. For example, the Universal Genome hypothesis suggests that a universal genome, encoding all the genes required to create all the animals, somehow arose early in evolutionary history:

> According to this model, (a) the Universal Genome that encodes all major developmental programs essential for various phyla of Metazoa emerged in a unicellular or a primitive multicellular organism shortly before the Cambrian period; (Sherman, 1873)

But why would evolution create such an extensive set of genes when there is no need for them and, otherwise, there would be no fitness improvement? Furthermore, why do we not find many of these genes in the lower taxons? Perhaps evolution removed them:

> This apparent problem could be explained not by a progressive evolution of more complex gene systems from primitive forms, but rather by the loss of certain unused elements of the Universal Genome in primitive forms during the last 530 million years. (Sherman, 1875)

To summarize, this failed prediction was met with the hypothesis that evolution created enormous complexity early on, when there was no apparent need for it, and then later eliminated that complexity where it was not used.

This response appears to be more of an ad hoc explanation to resolve a particular failure, than a consistent, coherent, parsimonious and fruitful elaboration on the theory. Thus, this failed prediction has led to the loss of several theory virtues, including evidential accuracy, causal adequacy, internal consistency, internal coherence, universal coherence, simplicity, durability, and fruitfulness.

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## False Prediction 18: Structures do not evolve before there is a need

A fundamental premise of evolutionary theory is that evolution has no foresight. It is a blind process responding to current, not future, needs. This means that biological structures are not expected to evolve before they are needed. But many examples of exactly this have been discovered in recent years. For instance, in the embryonic stages of a wide variety of organisms, the development of the vision system is orchestrated by similar control genes, known as transcription factors. As one paper explained, "All eyes, invertebrate and vertebrate, develop through a cascade of similar transcription factors despite vast phylogenetic distances." (Wake, Wake and Specht, 1034) Because these transcription factors are so prevalent across the evolutionary tree, they must have evolved in the very early stages of evolution, in an early common ancestor. But that was before any vision systems had evolved. The vision system is just one of several such examples showing that, under evolutionary theory, the genetic components of many of today's embryonic development pathways must have been present long before such pathways existed. Under evolutionary theory this unexpected phenomenon must be widespread, including in plants:

> Recent comparative genomic studies have shown that many key phenotypic novelties evolved long after the genes implicated in their development. Thus, the episodic increases in plant disparity may have resulted from the realization of genomic and developmental potential through ecological opportunity. (Clark)

Evolutionists now refer to the appearance of these genetic components, before they were used as such, as *preadaptation*:

> Genome comparisons show that the early clades increasingly contain genes that mediate development of complex features only seen in later metazoan branches. … The existence of major elements of the bilaterian developmental toolkit in these simpler organisms implies that these components evolved for functions other than the production of complex morphology, preadapting the genome for the morphological differentiation that occurred higher in metazoan phylogeny. (Marshall and Valentine, 1189)

Such preadaptation extends beyond embryonic development. For example, several key components of the human brain are found in single-celled organisms called choanoflagellates. Therefore, under evolution, these key components must have evolved in single-celled organisms, long before animals, brains and nerve cells existed. As one evolutionist explained, "The choanoflagellates have a lot of precursors for things we thought were only present in animals." (Marshall)

Another example is the molecular machines for protein transport across the mitochondria inner membrane which must have evolved long before mitochondria existed. (Clements et. al.) As one evolutionist explained, "You look at cellular machines and say, why on earth would biology do anything like this? It's too bizarre. But when you think about it in a neutral evolutionary fashion, in which these machineries emerge before there's a need for them, then it makes sense." (Keim)

So, to summarize, evolution predicts that structures do not evolve before there is a need for them, but the empirical evidence, when interpreted according to evolutionary theory, indicates otherwise—structures must have evolved before there was a need for them. In response to this failed prediction, evolutionists have suggested neutral evolution. At the genetic level neutral evolution refers to mutations that are neutral with respect to fitness. But here, the suggestion goes far beyond mere mutations. The suggestion is that elaborate, complex structures such as entire genes, genetic networks, cellular machines, and so forth evolved without assisting the fitness of the organism.

This is an entirely different, and much more demanding, version of neutral evolution. It is the accumulation of a large number of neutral mutations. While a single neutral mutation is not at all unlikely, a large number of them that fortuitously construct larger structures, that would later be useful, is unlikely. The probability decreases exponentially with the number of mutations required, and a large number is required to evolve these larger structures. Furthermore, under evolutionary theory, since selection does not act under such neutral evolution, the long sequence of new mutations would not persist, but would be subject to yet further, destabilizing, mutations. Therefore, this failed prediction indicates a loss of evidential accuracy, causal adequacy, universal coherence, and durability.

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### False Prediction 19: Ultra-conserved elements

According to evolution, while DNA mutations are often harmful, they may also be neutral or even beneficial and drive evolutionary change. Furthermore, this on-going process of DNA mutation can be seen in the species genomes. Similar species share similar genomes because not much time has passed since they emerged from their common ancestor. On the other hand, highly different species have very different genomes because they have been evolving independently for a much longer time. What is not expected is highly similar DNA segments in otherwise distant species.

But that is exactly what has been discovered in the so-called ultra-conserved elements (UCEs). Stretches of DNA, dozens or even hundreds of base pairs in length, have been found across a wide range of species. In fact, across the different species some of these sequences are 100% identical. Species that are supposed to have been evolving independently for long evolutionary epochs were certainly not expected to have identical DNA segments. “It absolutely knocked me off my chair,” remarked one evolutionist. (Pearson) “It can’t be true” another commented. (Pennisi, 1591)

The problems that UCEs pose for evolution are several. Consider, for example, this 796 base pair UCE:

ATATGAATTTTTGAAACTCTTAAGAAAACCTCTAGTCCCAGGAGATACTTGAGTTTGCAGCCTTGATCGTTGAGTTA
ACTCCCATCATCTTCCTAACTGGAGATACCCAGTCCCATCAGTCCATCTGTTTGGTGGACGTCCCTCAAAGATCTCA
TACCACCTACTCAAGAGAAGCTGTTATTAGGAAATGTTCTTCCAGCCCCCTCAGCATTTTTATTTGAGAGGAAGATA
CTAGCATTTCCCAATCAAAATAGACTATGGCCAGGATAGTTTGGAGCTCAACAGTGAATCCAGCCCCTGATATTATA
CTTTAATTAAACCTAAGGCATAGAAATTGAACTCTCTTATTAAAGATTTCTGGATTTTTTAAGTCCTTTTTTAGATA
CATTGTGTTACCTGTTTCTTTTCTACTAAGCATCTGAGCAGCAGACTTCTTTTTCTTTTGAGGGGCTTTTTCGGCTT
GAAGGTATCAGTTTTAGAGAGCAGGACTTCATTAATTTTTGCCAAGAGGCCTTCGATCCACCTCAAAGCAAATGTAC
ACAAACCAAGTTCTGTAGCTGAAAGTGAACCTGAATAGGGAAACAGGGGTACATGCATCTGACAGTCTCAGCCGTGT
GCAGTCAGAGCAAAGTGCAAATCACTCCTGATGAGAAATACGTTTTTTACCGCTTCATGACAAGCAACCATTTTTTC
TGTTGTTCGTCTCTCCAGTTTTGGTCATAAACACAGAACCATTCCAGCAGTCTTCCACAATTCTGTTTGTAATTTAA
GTTTTTTTTTTTTTAAACCTTGAGTC (Ryu, Seridi and Ravasi)

This sequence, or portions of it, are found in very few species. Notably, it is found in its entirety, with a 100% match, in the human and sea anemone genomes. In other words, two species with very different body plans, internal organs, behaviors, and environments, and that according to evolution have been evolving independently for almost a billion years, nonetheless share a long, 796 base pair stretch of DNA that is an exact match. And on top of that, the genomes of neighboring species, such as the other primates, do not contain the sequence.

So, this, and other UCEs, must have arisen in a primitive species, relatively early in evolutionary history. Presumably it had an important function, as tests have found for many UCEs. (Dickel, et. al.) So, it was conserved throughout evolutionary history via purifying selection. Any changes were filtered out.

But in this hypothesis, how did the UCE initially evolve? If its function is crucially dependent on the entire sequence, then its evolution by random mutation, which would be gradual, would not be possible. Furthermore, most of the functions envisioned for human UCEs involve higher level

capacities, such as embryonic development, the immune system, sensory reception, and so forth. These functions would not yet exist at the early, initial origin of the UCE.

Beyond this, why would such an important and lengthy sequence evolve in so few, sporadic, species? Under evolution, the sequence must be crucial in the sea anemone and human, and yet is eliminated, for example by deletion or by drift, in most other species.

UCEs contradict evolutionary expectations in multiple ways. And while it is not that evolutionists cannot come up with explanations for UCEs, but in doing so they resort to arbitrary and ad hoc mechanisms that make evolutionary theory capable of explaining just about any outcome. Perhaps UCEs exist in mutational "cold spots." Perhaps purifying, or negative, selection acted in particular lineages. Perhaps random sampling, natural catastrophes, genetic drift or population bottlenecks occurred. The result is a vague narrative that lacks specificity and detail. As one study explained:

> We concluded that each UCE group arose independently on a specific lineage and was "frozen" on the genome as a regulatory innovation after the divergence of specific taxa. (Ryu, Seridi and Ravasi, 2)

But this explanation does not address the problems presented by UCEs. Why would genetic sequences, often hundreds of residues in length, evolve in ancient, primitive organisms? If they were simply random sequences generated from random mutations, then how could they later attain function? And furthermore, attaining a function that required 100% sequence identity? It would be highly unlikely for a sequence of random mutations to serendipitously land on the exact sequence required. And why would the sequence be essential to be maintained in distant, isolated, species, and not in their neighboring species? And this unlikely series of events must have occurred over and over, to account for the many different UCE groups.

In addition to the lack of evidential accuracy revealed by UCEs, the evolutionist's non explanation (Koonin and Novozhilov, 102) that UCEs were "frozen" indicates a lack of causal adequacy, internal and universal coherence, durability, and fruitfulness.

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## False Prediction 20: Nature does not make leaps

Evolution is a process. It occurs gradually via variations within populations. The tempo may vary, but "the canon of 'Natura non facit saltum,'" as Darwin explained, was "on this theory intelligible." (Darwin, 414) This was an understatement. As Stephen Jay Gould explained, "Darwin was a strict adherent" to the doctrine that nature does not make leaps. (Gould, 179) But today, this expectation and prediction of evolution has failed.

The first problem, that species appeared abruptly in the strata, could be explained as a spotty fossil record, though incredible stretches of evolutionary progress would have to have gone missing. But the fossil record is not the only evidence for leaps. Since Darwin, rapid change has been directly observed in species ranging from unicellular organisms to plants and animals. As one evolutionist concluded, evolution "can occur much more rapidly than we previously thought. Rapid evolution is pervasive, and the list of examples is growing." (Cornell University)

This means that evolutionary theory needs a new mechanism of change. In fact, it appears doubtful that minor biological variation leads to large-scale change. As one evolutionist put it, macroevolution is more than repeated rounds of microevolution. (Irwin, 61) Increasingly evolutionists have recognized the need for a new mechanism to explain evolutionary change. (Gould, 579, 582) In recent years evolutionists have considered precisely what Darwin ruled out: saltational evolution. Here are some examples:

> As nature does jump, exclusive gradualism is dismissed. Saltatory evolution is a natural phenomenon, provided by a sudden collapse of the thresholds which resist against evolution. The fossil record and the taxonomic system call for a macromutational interpretation. (van Waesberghe, 3)

> We offer evidence for three independent instances of saltational evolution in a charismatic moth genus with only eight species. … Each saltational species exhibits a markedly different and discrete example of discontinuous trait evolution (Rubinoff and Le Roux, 1)

> Major transitions in biological evolution show the same pattern of sudden emergence of diverse forms at a new level of complexity. The relationships between major groups within an emergent new class of biological entities are hard to decipher and do not seem to fit the tree pattern that, following Darwin's original proposal, remains the dominant description of biological evolution. The cases in point include the origin of complex RNA molecules and protein folds; major groups of viruses; archaea and bacteria, and the principal lineages within each of these prokaryotic domains; eukaryotic supergroups; and animal phyla. In each of these pivotal nexuses in life's history, the principal "types" seem to appear rapidly and fully equipped with the signature features of the respective new level of biological organization. No intermediate "grades" or intermediate forms between different types are detectable. (Koonin, 1)

> Here we provide for the first time evidence of major phenotypic saltation in the evolution of segment number in a lineage of centipedes (Minelli, Chagas-Júnior and Edgecombe, 318)

Titles of research papers, which include phrases such as “farewell to Darwinism, neo- and otherwise,” “when natura non facit saltum becomes a myth,” “Saltational evolution: hopeful monsters are here to stay,” and “a Neo-Goldschmidtian view of unicellular hopeful monsters,” highlight this falsification of evolution’s prediction that there are no leaps. The problem is, aside from ad hoc explanations such as that evolutionary change occurs extremely rapidly and leaves no fossil evidence, there are no compelling evolutionary mechanisms to explain these saltational patterns, indicating a loss of causal adequacy. And the addition of Ad hoc explanations indicates a loss of internal coherence, simplicity, and fruitfulness.

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### False Prediction 21: The major plant groups share a common ancestor

Charles Darwin completed the last of his revisions to the *Origin* in the 1870s, but he nonetheless continued to have concerns about his theory of evolution. One of those concerns was the abrupt appearance of certain plants in the fossil record. Plant classification is complicated and has undergone several reformulations in past centuries. The plants in question—which in the 1870s were known as dicotyledons—grow at their tips rather than internally. Darwin knew since the 1830s that these plants showed no signs of a gradual origin, and by the 1870s the problem remained unresolved. William Carruthers, the Keeper of Botany at the British Museum, argued these plants contradicted evolution for several reasons. For example, they had "numerous differences" and were "sharply separated" from the other major plant groups. (Buggs 2021) By the end of the decade Darwin lamented in a letter to botanist Joseph Hooker that "The rapid development as far as we can judge of all the higher plants within recent geological times is an abominable mystery." (Buggs 2017, 169)

In the twentieth century Darwin's "abominable mystery" became a popular catch phrase, and it highlights that evolutionary theory expects all the major plant groups to be related by intermediate lineages. The plant groups should not be isolated and apart, sequestered in their own clusters.

But this prediction has failed as ever since 1879 the major plant groups, despite vastly more data, remain "sharply separated," as Carruthers put it. This failure was highlighted in a major 2023 study of all aspects of plant morphology, from sperm cell structure to gross plant architecture. The study assembled a "super matrix" composed of 548 traits for 248 living taxa representing every phylum, amounting to 131,280 data points. (Clark)

The results clearly confirmed that the major plant groups fall into discrete clusters, leaving large areas of morphospace unoccupied. Furthermore, the fossil data, rather than filling in an evolutionary tree structure, confirm the discrete pattern:

> The phylomorphospace constructed from both fossil and extant taxa shows that fossil taxa do not alter the fundamental pattern of morphospace occupation seen in extant taxa and none lie beyond the regions of morphospace occupation circumscribed by the extant phylomorphospace. (Clark)

The researchers hypothesize that unknown evolutionary innovations occurred in the gaps between clusters, and left no trace of fossil evidence:

> Here we show that the living divisions comprise discrete clusters within morphospace, separated largely by reproductive innovations, the extinction of evolutionary intermediates and lineage-specific evolution. (Clark)

In other words, to save the theory evolutionists must appeal to unobserved mechanisms, creating unobserved intermediate species, at unknown times, in unobserved lineages. Therefore, this failed prediction reveals failure in evidential accuracy, causal adequacy, internal consistency, internal coherence, universal coherence, simplicity, durability, and fruitfulness.

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## False Prediction 22: Altruism

In *Origins*, Darwin did not examine the question of altruistic behavior in great detail. But he did explain that natural selection could not result in destructive behavior. Afterall, evolution is driven by reproductive differentials and "every single organic being may be said to be striving to the utmost to increase in numbers." (Darwin, 52)

But today we know of many examples of unambiguous altruism which are destructive to reproductive chances. It is not controversial that the evolutionary prediction Darwin issued has been falsified many times over. Indeed, a plethora of designs are "more injurious than beneficial" (Darwin, 162) to reproduction. They are found everywhere, from the mindless, single-cell bacteria to the many subtle behavior patterns of humans.

Consider those who choose to have few or no children. Such behavior is not uncommon, and it certainly harms one's reproductive success. There are also many examples of altruism including giving blood and donating organs, giving to charities, helping the needy, and heroic wartime acts such as smothering a grenade or rescuing prisoners. Such acts of love and kindness falsify the evolutionary expectation that organisms should be oriented toward high levels of reproductive success.

### Kin selection

In the last fifty years evolutionists have proposed several explanations for altruistic behavior. As a consequence, the theory has become enormously more complex and incredible. First, the hypothesis of kin selection was proposed by William Hamilton in the early 1960s. (Hamilton) It has since become fundamental in evolutionary explanations of altruism. The idea is that altruistic behavior is a consequence of shared genes. For example, consider a genetic modification that encourages siblings to help each other. Such altruism increases the reproductive success of the sibling. If the sibling shares the genetic modification (as they well might), then the altruistic gene ends up helping to propagate a copy of itself. Thus, the behavior is not quite so altruistic after all. From the evolutionary perspective of reproductive success, altruistic behavior makes sense where there are shared genes.

Therefore, the hypothesis of kin selection implies that altruism will be greatest where gene sharing is greatest, such as between siblings and between parent and child, in human relationships. On the other hand, altruism will be weaker when there is less gene sharing (e.g., between cousins).

In addition to the degree of gene sharing, the hypothesis of kin selection also implies that altruism will depend on the number of individuals being helped. A person will be more inclined to aid multiple siblings, for there would be more shared genes at stake. As Hamilton put it, the hypothesis implies that while no one is prepared to sacrifice his life for any single person, everyone will sacrifice it for more than two brothers, or four half-brothers, or eight first cousins. (Hamilton)

### A more complicated selection process

Within a few years kin selection was used to explain a wide range of behaviors in addition to altruism. (e.g., Trivers, 1971; Williams) But these explanations brought with them an enormously complex evolutionary process. Consider altruism between siblings. Evolution's unguided genetic modifications must have somehow created this complex behavior. This new modification created a medium level of altruism toward people that could be recognized as sisters or brothers. It was not too much altruism and not too little. It was not toward females rather than males, short people rather than tall people, or blondes rather than brunettes. Presumably all these, and many more, types of behavior would be just as likely to have randomly arisen as was the needed sibling altruism. So, evolution must have constructed, tested and selected from an enormous set of potential behaviors before finding the few, rare behaviors that fit the kin selection criteria.

And the testing of these behaviors would not be simple. Initially, a new behavior, such as sibling altruism, would not fit the kin selection criteria. This is because, initially, the genes for the new behavior are in only a single individual. Not until the next generation could the genes possibly be distributed amongst siblings. And when that time does come, there is the question of whether the altruistic behavior would actually enhance the reproductive chances of the sibling. Being kind to a sibling does not necessarily do the job the first time. Many generations might be needed, as kin selection can only occur when an altruistic act genuinely improves the reproductive success of the sibling.

**Evolution's creative powers**

Even more of a problem for evolution is the creation of these complex behaviors. Somehow unguided genetic modifications must have resulted in genes for a wide range of attitudes and behaviors. The list is staggering. There are of course the obvious behaviors such as love, hate, guilt, retribution, social tendencies and habits, friendship, empathy, gratitude, trustworthiness, a sense of fulfillment at giving aid and guilt at not giving aid, high and low self-esteem, competition, and so forth.

These behaviors are supposed to have evolved according to the kin selection criteria, along with many more nuanced behaviors. For instance, love not only evolved, but in varying degrees depending on the degree of shared genes. It is weaker within the extended family than within the family. Low self-esteem behavior not only evolved, but the art of not hiding it can be advantageous and so also evolved. Sibling rivalries evolved, but only to a limited degree. In wealthy families, it is more advantageous for siblings to favor sisters while in poor families, siblings ought to favor brothers. So those behaviors evolved. Mothers in poor physical condition ought to treat daughters as more valuable than sons. Likewise, socially or materially disadvantaged parent sought to treat daughters as more valuable than sons.

Evolutionists explain all these nuanced behaviors according to the calculus of kin selection. For instance, consider sympathy and compassion. According to evolution, compassion and sympathy are nothing more than cleverly disguised manipulations. For while we may like to think our sympathy is pure, in fact it comes at a price. The unspoken yet universal expectation is: "you owe me one." As one science writer put it, "Exquisitely sensitive sympathy is just highly

nuanced investment advice. Our deepest compassion is our best bargain hunting." (Wright, 205) What such explanations fail to explain is the enormous complexity now added to the theory. Yes, the altruism is explained as advantageous, but such nuanced behaviors must somehow have arisen in the first place, in order to be later selected.

And, evolutionists warn, we should not be fooled by our intuition that certain behaviors are "obvious," or "right." For instance, love for one's children and grief at the death of a child may seem to be natural reactions, but evolutionists explain that what seems to us to be common sense is, itself, merely a manifestation of our evolved behaviors. Yes, we love our children, but only because such a behavior was selected. We have evolution to thank for our heartfelt emotions.

But do not many of our moral sentiments and behaviors reflect right and wrong? Are not loyalty, sacrifice, honor, our sense of justice, obligation and shame, remorse and moral indignation more than merely the result of mutations and selection? No, warn evolutionists, such appeals only reveal the power of evolution. As one writer put it, "It is amazing that a process as amoral and crassly pragmatic as natural selection could design a mental organ that makes us feel as if we're in touch with higher truths. Truly a shameless ploy." (Wright, 212)

In fact, evolutionists explain, evolution has constructed elaborate deception mechanisms. Children use temper tantrums to manipulate parents. Parents countered this with the ability to discern and children, in turn, refined their manipulation with heartfelt whining. All a result of the complexities of natural selection. Cheating, suspicion, exaggeration, embellishment, hypocrisy, displays of morality, false compliments, self-serving dishonesty, boasting and self-deprecation are all evolved behaviors in accordance with natural selection.

Deception is rampant and evolutionists believe it evolved in biology to enhance reproduction. In turn, the ability to recognize deception has evolved, which in turn spurred the evolution of some degree of self-deception, to better fool the opponent. This self-deception should not be underestimated. It really means that we are, to a certain degree, truly deceived about the world around us. Our brains did not evolve to know truth, but some skewed version of reality. As one evolutionist concluded, "the conventional view that natural selection favors nervous systems which produce ever more accurate images of the world must be a very naïve view of mental evolution." (Trivers, 1976, XXVI)

Here evolution aligns itself with radical skepticism. Nothing can be known to be true. If evolution is true, then not only are our minds nothing more than the product of unguided natural processes, but those very processes inbred a certain degree of falsehood. The evolutionist's claim that evolution is a fact is self-refuting, for it leads to the conclusion that they cannot know that evolution is a fact.

Regardless of how deceived we are, we do know that evolution now calls for unguided genetic variation to create an incredible menagerie of complex and nuanced behavior. The enormous inventory of human behavior, which was selected, is only a tiny fraction of what must have been created. It would be swamped by the myriad behaviors which were not advantageous. In order to explain altruism, evolutionists now make a staggering claim about what must have arisen in nature. But the claim is a trade secret, as it is rarely discussed. Evolution has become a theory of

seemingly endless speculation about behavior with little explanation of how the specific behaviors actually are supposed to have arisen. Evolutionists speculate at length about how behaviors could have been advantageous, with little consideration of the origin of such behaviors. Here is a representative example of this speculation, regarding an imagined behavioral strategy called "Selfish Punisher," which exploits altruists and punishes other selfish individuals.

> Individuals who behave altruistically are vulnerable to exploitation by more selfish individuals within their own group, but groups of altruists can robustly out-compete more selfish groups. Altruism can therefore evolve by natural selection as long as its collective advantage outweighs its more local disadvantage. All evolutionary theories of altruism reflect this basic conflict between levels of selection. It might seem that the local advantage of selfishness can be eliminated by punishment, but punishment is itself a form of altruism. For instance, if you pay to put a criminal in jail, all law-abiding citizens benefit but you paid the cost. If someone else pays you to put the criminal in jail, this action costs those individuals something that other law-abiding citizens didn't have to pay. Economists call this the higher-order public goods problem. Rewards and punishments that enforce good behavior are themselves forms of good behavior that are vulnerable to subversion from within. (Binghamton University)

Hypotheses such as this are now rampant within evolutionary theory. They are required to explain the wide range of behaviors in biology, and they force evolution to unprecedented levels of complexity. Unguided genetic change must be capable of somehow generating a wide array of behaviors with incredible precision.

And not only must all these varied and nuanced behaviors have arisen via unguided genetic modifications, but orders of magnitude more behaviors, which were not advantageous, must also have arisen. If unguided genetic variations were able to generate such pinpoint behaviors from which selection could choose, then there must also have been a vast menagerie of bizarre behaviors that were not selected. For the genetic variations were unguided. There was no foreknowledge of which behaviors were advantageous and which were not. The latter vastly outnumber the former, and so any given variation was most likely selected against. Only the rare exceptions were advantageous and evolutionary history must be chocked full of never observed pathologies that would not pass evolution's test.

**Problem of non-reciprocal altruism**

In addition to the tremendous complexity that kin selection adds to the theory of evolution, there is the problem that it does not explain altruistic behaviors for which no advantage to the individual can be imagined. Why do soldiers smother grenades? Why do rescuers risk their lives? Why does Mother Theresa help the needy in faraway countries? Kin selection does not explain altruistic acts where there is no advantage to one's own genes.

To explain such altruism, evolutionists must turn to unlikely speculation. For instance, a popular explanation is that in earlier ages our ancestors lived in small clans and villages where blood relations where more common. If most everyone in the village was a relative of yours, then altruistic behaviors would be advantageous more often. By the time civilization expanded into

cities and nations, the altruistic behavior had evolved. So now we give aid to unrelated people because our evolved genes consider all people to have at least some relation to us.

In this model today's examples of altruism that do not seem explainable using kin selection are viewed as vestigial behaviors. They were selected in the past, but now are operating outside the scope of kin selection. So, although, as we saw above, evolution must have tremendous precision in creating finely tuned, nuanced behaviors, here evolution becomes a crude instrument. When needed, evolution can act with surgical precision. But when problems arise, evolution is suddenly clumsy. It is remarkable that, on the one hand Mother Theresa is left clueless that orphans on the other side of the world do not share her genes, yet on the other hand evolution can precisely construct detailed behaviors such as the Selfish Punisher strategy, the detailed altruism profiles between wealthy and poor families, and so forth. Mother Theresa falsifies the evolutionary expectations. As a consequence, the theory is forced to adopt low probability, high complexity modifications.

Several other explanations have also been contemplated. For instance, perhaps aiding another individual enhances one's status and attractiveness. Perhaps selection occurs at higher levels than the gene. (Wilson, Wilson; Bowles) Or perhaps what seems to be selfless altruism actually plays to self-centered motives. Yes, "Mother Theresa is an extraordinary person," explained one evolutionist, "but it should not be forgotten that she is secure in service of Christ and the knowledge of her Church's immortality." (Wilson, 173) Ultimately, even helping the poor on the other side of the world can be rationalized with natural selection. With these and other explanations, evolutionists are able to provide some sort of selection rationale for practically any behavior.

### Altruism conclusions

Darwin's theory of evolution led him to several expectations and predictions, regarding behavior in general, and altruism in particular. We now know those predictions to be false. Furthermore, in order to explain many of the behaviors we find in biology, evolutionists have had to add substantial serendipity to their theory. The list of events that must have occurred to explain how evolution produced what we observe is incredible and the theory has become highly complex.

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## False Prediction 23: Cell death

According to evolutionary theory, biological variation that supports or enhances reproduction will increase in future generations—a process known as natural selection. The corollary to this is that biological variation that degrades reproduction will not be selected for. Clearly, natural selection could not result in destructive behavior. Here are two representative statements from *Origins*:

> we may feel sure that any [biological] variation in the least degree injurious would be rigidly destroyed. (Darwin, 63)
>
> Natural selection will never produce in a being any structure more injurious than beneficial to that being, for natural selection acts solely by and for the good of each. (Darwin, 162-3)

But do we not find examples of such "injurious" behavior in nature? When the rattlesnake rattles its tail, is this not injurious to its hunt for food, and ultimately to its reproductive chances? Darwin argued that this and other such examples are signals to frighten away enemies, not warn the intended prey.

But today we have many examples of injurious behavior that falsify Darwin's prediction that natural selection "will never produce in a being any structure more injurious than beneficial to that being." In bacteria, for example, phenomenally complicated mechanisms carefully and precisely destroy the individual. Clearly, this suicide mechanism is more injurious than beneficial to the bacteria's future prospects.

One such mechanism consists of a toxic gene coupled with an antitoxic gene. The toxic gene codes for a protein that sets the act of suicide into motion and so ultimately kills the bacteria. The antitoxic gene inhibits the toxic gene from executing its mission. Except, that is, when certain problems arise. Lack of proper nutrients, radiation damage and problems due to antibiotics can all cause the antitoxin to be diluted, thus allowing the toxin to perform its mission. (Chaloupka, Vinter; Engelberg-Kulka, Hazan, Amitai; Engelberg-Kulka, Amitai, Kolodkin-Gal, Hazan; University of Nebraska)

This bacterial suicide is probably good for the bacteria population on the whole. If nutrients are running low, then better for some bacteria to die off so the neighbors can live on. Not only will the reduced population require less nutrients, but the dismantled bacteria help to replenish the food supply. Therefore, evolutionists can explain the suicide mechanism as having evolved not for the individual bacteria, but for the population. But the explanation introduces major problems for the theory.

Suicide is probably good for the bacteria population, on the whole, in challenging conditions. Since gene sharing occurs within a bacteria population, evolutionists have no problem explaining such altruism as a result of kin selection (see False Prediction 22: Altruism). Such a facile response, however, misses the profound problem of how such a design could arise in the first place, for the mechanism is immensely complex.

In the example of bacteria suicide, the antitoxic gene normally inhibits the toxic gene from executing its mission. When the antitoxic gene is diluted then the toxic gene can perform its mission. The toxin does not, however, single-handedly destroy the cell. The toxin is an enzyme that cuts up the copies of DNA (i.e., messenger RNA, or mRNA) that are used to make other proteins. By slicing up the mRNAs, the cell no longer produces the proteins essential for normal operation. But the toxin does not cut up all mRNAs. Some mRNAs escape unscathed, and consequently a small number of proteins are produced by the cell. These include death proteins that efficiently carryout the task of disassembling the cell.

Death proteins are not the only proteins that the toxin allows to be produced. As researchers reported, the toxin "activates a complex network of proteins." (Amitai, 2) While some of the proteins bring death to the bacteria, others can help the cell to survive. The result is that most cells in the population are destroyed, but a fraction is spared. This of course makes sense. The suicide mechanism would not help the bacteria population if every individual was destroyed. Instead, some survive, and they can be the founders of a new population when conditions improve.

This suicide mechanism and "behavior" is altruistic. Some bacteria die off to save others. And the explanation that this bacteria suicide is due to kin selection adds tremendous complexity to the theory of evolution. Kin selection can select from only that which is available. This elaborate suicide mechanism must have just happened to arise from some combination of random mutations, and then remained in place in spite of not being selected for, until such time when it would succeed in surviving a stressful environment. The toxin and antitoxin genes with their clever functionality, the death and survival proteins, the inter cellular communications—all these were required to be in place and to be coordinated before the kin selection could even begin to act. This is highly unlikely and adds considerable complexity to the theory.

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## Conclusions

In the more than a century and a half since Darwin published his theory of evolution, science has been able to test many of its predictions. The result has been overwhelming. As documented in the example predictions described above, evolution's fundamental predictions have consistently been found to be false. According to one popular criterion for a scientific theory—its ability to predict future observations—evolution has consistently failed.

But prediction accuracy is not the only virtue on which to judge scientific theories. Philosophers of science have long since recognized various virtues that may be important. In addition to prediction accuracy, how consistent, fertile, durable, and simple is the theory? Other virtues have also been proposed. Recently Keas has further systematized theory evaluation with a four-class taxonomy of theory virtues. (Keas) In this study I have used 23 failed predictions as a framework, with which to evaluation evolutionary theory according to a subset of theory virtues: evidential accuracy, causal adequacy, internal consistency, internal coherence, universal coherence, simplicity, durability, and fruitfulness.

Evolution repeatedly fails on these eight theory virtues, and this can be seen in the failed predictions documented above. It is straightforward that the many failed predictions show that evolution fails significantly on *evidential accuracy* and *durability*. Evolution's false predictions show that the theory does not fit the empirical evidence well, and that the theory has not passed the testing of its fundamental predictions.

But the failed predictions also reveal repeated failure on the other six virtues listed. The many false predictions repeatedly show a lack of *causal adequacy*—causal factors that could plausibly produce the evidence in need of explanation. Furthermore, the failed predictions reveal failures of both *internal consistency* and *internal coherence*. That is, evolution takes on components that are both contradictory and ad hoc, tacked on to solve isolated problems.

Evolution lacks *universal coherence* as it contradicts other warranted beliefs and knowledge from science and mathematics. Evolution also lacks *simplicity* and parsimony as the theory takes on enormous levels of complexity and theoretical content to explain the evidence. Finally, evolution lacks *fruitfulness* as the failures merely generate ad hoc theory elaboration rather than additional discovery.

This paper describes a sampling of evolution's predictions which have proven false. These reveal a consistent pattern of evolutionary theories failure to fit the empirical evidence. Furthermore, these false predictions reveal how evolution repeatedly fails on several theory virtues that historians and philosophers of science view as important. Clearly, evolution does not pass the test of science.

### References

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