# Classic Text 30 – Philosophy of Biology: Further Problems of Darwinism

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In Classic Text 26 we considered the possibility of biological laws and how they might inform biological theories. In this study unit we follow a reading of the seminal paper by Stephen J. Gould and Richard Lewontin (1979): *The Spandrels of San Marco and the Panglossian Paradigm: A Critique of the Adaptationist Programme*, which may be may be downloaded for free here. (Under South Africa copyright law individual academic papers may be reproduced for educational purposes.) Their paper is primarily concerned with adaptation and the errors and temptations in to which many biologists have fallen in their enthusiasm to see and infer adaptation in virtually every biological phenomenon. The academic debate that the paper elicited continues to inform our current understanding of research into evolutionary biology. Once again, we shall be relying on Alex Rosenberg and Daniel McShea's (2008) textbook *Philosophy of Biology - A Contemporary Introduction* as a guide. Their Chapter 3 "Further problems of Darwinism: Constraint, drift, function" informs the following discussion.[1]

The evolutionary biologist David C. Queller (1995) rather uncharitably described Gold and Lewontin's a paper as "an opinion piece, a polemic, a manifesto, and a rhetorical masterpiece". That it is a rhetorical masterpiece is undeniable; however it is neither an opinion piece, nor a polemic, nor a manifesto. On the contrary, it is a scholarly article that relies on sustained argumentation, employing analogy and factual observations which are carefully referenced. In short, it is an exemplar of philosophy of biology.

*One of the Spandrels in St. Mark's Basilica that Inspired One of the Paper's Analogies*

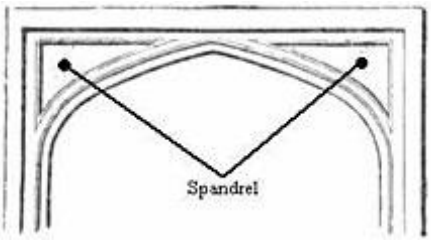
The title of the paper requires some decoding for the uninitiated. In 1978, Gould, who was the main author, had visited St. Mark's Basilica in Venice shortly before penning the paper. He delivered it as a talk to the Royal Society in the same year and it was published the following year with both Gould and Lewontin listed as authors. The decorated spandrels that Gould had seen in St. Mark's are roughly triangular spaces at the intersection of the four supporting arches at right angles to the dome above. According to Gold and Lewontin, the mosaic design on the spandrels in St. Mark's Basilica is

> … so elaborate, harmonious, and purposeful that we are

[1] While South Africa copyright law allows for the reproduction of individual chapters; we cannot, unfortunately, make any further chapters of Rosenberg & Daniel McShea (2008) available on-line as this would constitute the reproduction of a substantial proportion of the book. We therefore strongly recommend buying or borrowing the book from your local or university library.

> tempted to view it as the starting point of any analysis, as the cause in some sense of the surrounding architecture. But this would invert the proper path of analysis. The system begins with an architectural constraint: the necessary four spandrels and their tapering triangular form. They provide a space in which the mosaicists worked; they set the quadripartite symmetry of the dome above. (1979)

In other words, wherever an arch meets rectangular frame or a dome at right angles, a spandrel is an architectural necessity. Spandrels are not constructed *in order* to provide decorative spaces; though they may subsequently be decorated. Gould and Lewontin use this simple observation as the source for the first of their titular analogies.




*Two Spandrels above a Tudor Arch*

The source for the second analogy is drawn from the character of Dr Pangloss from Voltaire's (1759) satire *Candide*. Dr Pangloss is a caricature of the German philosopher Gottfried Leibniz noted *inter alia* for his unique brand of philosophical optimism. Leibniz concluded that, "our universe is, in a restricted sense, the best possible one that God could have created". (Wikipedia: Gottfried Wilhelm Leibniz) Voltaire derides such optimism via his character, Dr Pangloss who claims: "Things cannot be other than they are... Everything is made for the best purpose. Our noses were made to carry spectacles, so we have spectacles. Legs were clearly intended for breeches, and we wear them". Even in a pre-Darwinian era, such a claim would have been ridiculous; however Gould and Lewontin draw a powerful analogy from the absurdity with the tendency of some biologists of the day to focus exclusively on adaptation as an explanation for almost every biological trait. These analogies will be developed below; however Rosenberg and McShea first consider the phenomenon of genetic drift. (2008 p. 65)

As we saw in Classic Text 22, genetic drift is a chance outcome in evolution in which there is a change in the frequency of an existing allele within a population over time. As Rosenberg and McShea point out, there a difference between acknowledging genetic drift as a factor in evolution and agreeing on what it is, how it works, and how it is related to adaptation. These questions turn on differences in the interpretation of probability by philosophers of science for at least the last two centuries. (2008 p. 65)

**Adaptationism: for and against**

In Classic Text 26 we discussed the unfalsifiability of the principle of natural selection when fitness is defined in terms of reproductive success. There it became apparent that there are practical problems in defining fitness differences in terms of alternative sets of design problem solutions. Gould and Lewontin go further in criticising "adaptationism" as a research program in biology which "regards natural selection as so powerful and the constraints upon it as so few that direct production of adaptation through its operation becomes the primary cause of nearly all organic form, function and behaviour". Adaptationism, they argue, begins by "atomizing" an organism into separate traits and then seeks the design problem that each of the separate traits optimally solves. When the optimality of a trait cannot be demonstrated with respect to a single design problem, the notion of "trade-offs" is introduced by which organisms are interpreted as best compromises among competing demands. This is where the Panglossianism paradigm is introduced. Recall that Dr

Pangloss explained the function of human traits, such as the bridge of the nose or legs as supporting the wearing of glasses or breaches, respectively. More generally, Gould and Lewontin argue that it is not possible to tell whether a trait is adaptive just by examining a few generations of organisms. This entails a process of "reverse engineering" of function supported, not by scientific evidence but by "**just-so stories**" as in Rudyard Kipling's (1902) *Just So Stories for Little Children* – all Lamarckian origin fables such as, how the camel got its hump, or how the leopard got its spots, *etc.* (Rosenberg & McShea 2008 p. 66) Michal Hubálek (2020), on the other hand, regards just-so stories as "an equivalent term for a (provisional) speculative answer, evolutionary hypothesis, or heuristic that is, a starting point of evolutionary inquiry that might, but of course might not, promote fertile (scientific) investigation of evolutionary history".

The mistake of treating traits not selected for as adaptations is analogous to treating spandrels as if they were design features intended by the architect to provide spaces for mosaics and other decorations, instead of being necessary architectural constraints. Gould and Lewontin trace the adaptationist program through four "common styles of argument" summarized by Rosenberg and McShea (2008):

1. "If one adaptive argument fails, try another." If antlers do not really protect against predators, then perhaps they are selected for interspecies competition. If neither protection nor interspecies competition works, try sexual selection.
2. "If one adaptive argument fails, assume that another as yet undiscovered exists." Disconfirmation is only a reason to look further for another design problem the trait solves.
3. In the absence of a good adaptive argument in the first place, attribute failure to ignorance of the organism's structure and behavior.
4. "Emphasize immediate utility and exclude other attributes of form." (p. 67)

Under the following heading of "Telling Stories", Gould and Lewontin conclude:

> We would not object so strenuously to the adaptationist programme if its invocation, in any particular case, could lead *in principle* to its rejection for want of evidence. We might still view it as restrictive and object to its status as an argument of first choice. But if it could be dismissed after failing some explicit test, then alternatives would get their chance. (Gould & Lewontin, 1979 p. 587)

The problem with the adaptationist program then is not that natural selection explains the form and behaviour of most organisms but that that it is unfalsifiable because it refuses to admit any disconfirming evidence or take cognoscence of a lack of evidence. This is not the same claim that the theory of natural selection is unfalsifiable because the definition of fitness is circular. (See Classic Text 26.) However, according Rosenberg and McShea, the upshot is similar; although there are several components to Gould and Lewontin's charge that are questionable. Firstly, do all, or even some, evolutionary biologists embrace adaptationism as defined and the research strategy as characterised by Gould and Lewontin? Secondly, is there some form of adaptationism that is a defensible strategy? Thirdly, are there some nonselective factors that play a central role under certain conditions? Fourthly, how are we to understand the relationship between selection and constraint, and what is the appropriate role of each in evolutionary explanation? (Rosenberg & McShea, 2008 p. 67)

According Rosenberg and McShea, the first question has two answers. One is that times have moved on since 1979. If there ever were unreserved adaptationists they have since taken cognoscence of Gould and Lewontin's influential paper, with the result that adaptive hypotheses are now more carefully framed, falsifiable and open to non-adaptive explanations. The second answer is that evolutionists never were unrestrained adaptationists. Even before Gould and Lewontin's paper, it was acknowledged that adaptation is constrained in that form, physiology, and behaviour cannot result from adaptation alone. Rosenberg and McShea, for example, mention that Francis Crick believed that the genetic code was a "frozen accident" - that could have been otherwise. Similarly, there is no consensus among evolutionary biologists as to why African rhinoceroses have two horns whereas two of the three Asian species have just one. The difference could have been due to genetic drift but we can't say for sure. Also, there are many examples of poor adaptation or imperfections of design. Think of the blind spot of the vertebrate eye where the optic nerve enters in front of the retina rather than behind it as in cephalopods. Think also of the blind "moment" when we blink both eyes simultaneously rather than alternatively. Such "frozen accidents" or imperfections are constraints on selection that have been accepted as such without a problem by adaptationists. Indeed, they are as much the outcome of selection as they are due to constraint and contingency. (Rosenberg and McShea, 2008 p. 68)

As to the second question above, it does seem that in some cases at least, the search for adaptive explanations to questions such as sexual reproduction are taken seriously, but not to the extent derided by Gould and Lewontin. Consider that sexual reproduction is a central issue in biology. Most animals and plants do reproduce sexually, with asexual reproduction being the exception rather than the norm. Recall from Classic Text 26 that the most widely accepted definition of a species – the biological species concept – relies explicitly on sexual reproduction: *i.e. a* species is an interbreeding group of populations reproductively isolated from other such groups. And for this very reason, despite its popularity, it remains problematic. According to Rosenberg and McShea, sexual reproduction "is also probably the best example of the adaptationist's refusal to consider alternative explanations. Paradoxically, the reason is that there are features of sexual reproduction that are on their faces extremely difficult to reconcile with the theory of natural selection". (*l.c.*)

Those recalcitrant features are as follows: Sexual reproduction involves meiosis which produces gametes with only half the genetic material of the adult organism which gets passed onto the next generation. Therefore, if the number of copies of genes in the next generation is a function of fitness, then the cost of sexual reproduction in terms of fitness must be very high! *Ceteris paribus*, one would expect alternative methods of reproduction such as asexual budding or **parthenogenesis** ("virgin birth" from an unfertilized embryo, as seen in some insects like aphids but also in some vertebrates, such as the all female whiptail lizards). Yet parthenogenesis is very rare in the animal kingdom. Therefore, either sexual selection is itself an adaptation that arose by chance and/or constraint or it confers such an enormous benefit that it swamps the cost in fitness by meiosis. It is vanishingly unlikely that either chance or constraint could maintain sex against such a great disadvantage as meiosis; therefore according to adaptationists, sex must indeed be an adaptation. (p. 69)

According to Rosenberg and McShea, following the adaptationist program, evolutionary biologists have hypothesised alternative benefits that sex might confer and sought evidence that might support such hypotheses. None however has been sufficiently well confirmed to reconcile sexual

reproduction with natural selection. Almost all such hypotheses propose that the recombining or "shuffling" achieved by chromosomal crossing over (see Classic Text 10) where two homologous chromosome non-sister chromatids pair up with each other and exchange different segments of genetic material) is what makes sex adaptive. One intriguing hypothesis, owing to William D. Hamilton, suggests that recombination helps animals resist parasites. Because parasites reproduce and mutate quickly, they are able to exploit many opportunities to invade a host. One strategy to counter this is to regularly change the host traits to which parasites would otherwise become adapted. Recombination of genes from two genetically different host parents allows the rate of trait variation to outpace that of mutation among parasites. (p. 69)

However the hypothesis, even if true, has at least two problems. Firstly, it is unlikely that such a wide-spread and costly phenomenon as sexual reproduction could confer only one adaptive advantage, instead of many cumulative advantages. If this were so one would expect asexual reproduction to be to be present in only a limited number of environments, in which these "multiple advantages" are not necessarily an advantage, which is what we see. Secondly, given the difficulty of tracking the complexities of host-parasite competitive interactions across different lineages of animals, plants, fungi and micro-organisms, empirical evidence in support of the hypothesis will be hard to find. (p. 69)

Despite the intricacy of this task in general, adaptationists have not been deterred. Instead, they have adopted the very four styles of argument that Gould and Lewontin decried. *I.e.*

1. If one adaptive argument fails, try another.
2. If one adaptive argument fails, assume that another must exist…
3. In the absence of a good adaptive argument in the first place, attribute failure to imperfect understanding of where an organism lives and what it does.
4. Emphasize immediate utility and exclude other attributes of form.

According to Rosenberg and McShea, "[t]he adaptationist has to admit the possibility in principle that this approach could lead nowhere, that sex could – again in principle – be the result of some unknown constraint". However, given the very high cost of sexual reproduction along with its near ubiquity in nature, the search for an adaptive explanation is surely justified. Despite Gould and Lewontin's disapproval, they would perhaps have been prepared to accept an adaptive explanation that acknowledged alternatives. It is thus possible to read Gould and Lewontin's paper not as a blanket condemnation of the adaptationist program but as a condemnation of the reckless, superficial and complacent application of the same. (p. 70)

**Constraint and Adaptation**

The third question above was whether there are some *nonselective factors* that play a central role under certain conditions. Several areas within biology, which are not primarily about finding adaptive explanations, do focus on constraint, either as an operating assumption or a target of investigation. Darwin recognised a central role for **constraint** within evolution which he called the "unity of type". By this he meant that there is a fundamental conformity or limitation in the structure of organisms of the same class or order, which is quite independent of the ability of natural selection to effect change. Gould and Lewontin use the German word *bauplän* for "building

plan" (of a body) as an example. Essentially, a ***bauplän*** refers to the plan or fundamental structure on which a natural group of organisms or their systems of organs are assumed to be constructed. Thus, all vertebrates have a segmented spinal column; crustaceans have the same number and sequence of appendages on their heads; while insect bodies are always composed of a head, thorax and abdomen. Such body plans are almost universal in their respective groups and even though such arrangements may be less than optimal for some species the pattern (almost) always persists in the face of natural selection. According to Gould and Lewontin,

> the basic body plans of organisms are so integrated and so replete with constraints upon adaptation… that conventional styles of selective arguments can explain little of interest about them. It does not deny that change, when it occurs, may be mediated by natural selection, but it holds that constraints restrict possible paths and modes of change so strongly that the constraints themselves become much the most interesting aspect of evolution. (Gould & Lewontin 1979)

The constraint argument above does not deny that certain features of a body plan might have been adaptive at the time of their origin, even though many have subsequently become fixed by chance as "frozen accidents". Either way, earlier constraints on a body plan limits adaptation of subsequent organisms to their local environment. (Rosenberg & McShea 2008 p. 71)

According to Rosenberg and McShea, constraint has a "flip side" as an aid to adaptive radiation. Recall from Classic Text 22 that later traits are built on earlier ones, allowing ever more complex adaptations to be built up in a step-wise succession. Think of the most successful class of animals ever: the Insects, with some 5.5 million species, and endless variations built on a highly conserved body plan. "Constraint on early arising traits, imposed by the need to preserve later ones, is one of the mechanisms that makes such cumulative evolution possible". Constraint is involved in adaptation in yet another way, dubbed by Gould and palaeobiologist Elizabeth Vrba (1982) as **exaptations**. These are traits originally adapted to solve one design problem that have subsequently been co-opted by natural selection to solve a different design problem. (Exaptations in the past would have been called "pre-adaptations", except that the latter term is no longer used because it implies that natural selection had some sort of "foresight".) The wrist bones and digits of bats are one example of exaptation. Originally they evolved to solve one design problem but were later co-opted to solve another that involves supporting a wing membrane. Also recall from Classic Text 22 that evolution is opportunistic, often exploiting solutions in a way that is "quick and dirty", so long as they confer an immediate advantage on offspring who possess the "problem solving trait". Constraint is one reason why "better but slower to emerge" solutions may appear or may not be available at all. Indeed constraint on variation puts the break on natural selection's optimality, selecting instead from readily available variation. (p. 71)

Gould and Lewontin also mention brain size as an example of a type of constraint that Darwin called **correlation of parts**. This occurs when some body part that is not under selection as solution to a design problem but is instead the result of selection acting on another body part or the whole body with which it is correlated. Clearly brain size in humans has increased over time in the human lineage; however it is not clear whether larger brains are the result of selection for superior intelligence or whether increased brain size is simply the result of its correlation with an overall increase in body size. According to Rosenberg and McShea, constraint, in this example, has both

positive and negative features. Body size constrains brain size but also affords opportunities for producing change when such change may have no immediate selective advantage. (p. 71 - 72)

The constraints discussed above are classed as **phylogenetic constraints**, or limitations on change arising in the evolutionary history of a group (phylogeny). Ordinarily, these are described as limitations internal to the organism arising out of its development *i.e.* **developmental constraints**. The latter were central to the study of 19th century biology, especially in the German speaking world. Recall the debate over Haeckel *vs*. von Baer's embryological theories in Classical Text 26. Advances in population genetics in the 20th century however saw a shift to the study of adaptation, leaving what some perceived as a "gap" in the Darwinian research program. Gould and Lewontin (1979) drew attention to this gap and since then there has been a resurgence of interest in developmental constraints under the rubric of evolutionary-developmental biology, or "evo-devo". (p. 72)

Evolutionary-developmental biology compares the molecular mechanisms underlying developmental processes (ontogeny) of different organisms to infer the ancestral relationships between them and how developmental processes evolved in different groups (phylogeny). An important finding was the discovery of certain homologous genetic control units or *Hox* genes in animals as diverse as insects and vertebrates, that control embryonic development along the major axis of the body. These genes appear to have been highly conserved through hundreds of millions of years of evolution and therefore represent a major constraint on animal organization. (p. 72; Wikipedia: Evolutionary developmental biology)

Constraints are also important in the reconstruction of phylogenetic relations among species within a group in such a way as to distinguish between ancestral and descendent species. In order to illustrate the point, Rosenberg and McShea ask us to imagine how the world would look in the absence of such constraints If natural selection were able to optimally modify every species, there would be no homology, *i.e.* all similarity due to descent from a common ancestor would be erased. If, for the sake of argument, all bony fishes were optimised for their aquatic environment, then marine mammals which independently evolved in their aquatic environment, would ultimately be indistinguishable from bony fishes.

> Whales would have evolved scales. The tail fin would have rotated 90 degrees to lie vertically (instead of horizontally as it does in actual whales). Whales would also have lost the ability to nurse their young. (We would never know they were mammals.) And they would resemble fish not just at the gross structural level but in every detail, down to the level of tissue morphology and molecular physiology. (p. 72 - 73)

**Convergent evolution** is the independent evolution of similar features in different species in different geographical regions or different periods or epochs in time. If there were no constraint, all evolution would all be dominated by convergence on a number of similar design solutions. Constraint however imposes a barrier on adaptation that prevents perfect conversion. Indeed, one of the assumptions made in modern phylogenetic reconstruction is that of parsimony. *i.e.* that change is highly constrained, and that convergence from different traits to similar ones in different species via natural selection is rare. This assumption allows for the identification phylogenetic trees with the minimum number of changes and hence the minimum number of convergences. According to Rosenberg and McShea, one phylogenetic reconstruction method proceeds with, constraint,

rather than adaptation, as the default assumption in explaining shared traits over long periods of evolution. (p. 73; Wikipedia: Convergent evolution)

Constraint however is not the only cause of constancy of conserved traits. Selection too can produce constancy. The similarity of structure of the protein Cytochrome c involved in the electron transport chain is highly conserved across many plants, animals and protists, and is likely the result natural selection to perform the same metabolic function. Constraint is also likely to be involved where there has been a change in function. *E.g.* the persistence of pelvic (hip) bones in whales and dolphins could be the result of constraint, on the assumption that pelvic bones are not functional in these marine mammals. On the other hand, they may continue to provide an evolutionary advantage because the muscles that control a cetacean's penis - which has a high degree of mobility - attach directly to its pelvic bones. (p. 73; Wikipedia: Cytochrome c; Dines *et al.*, 2014)

Rosenberg and McShea mention three other classes of constraint: formal, physical, and architectural. **Formal constraints** are those that are necessitated by mathematics or geometry. The cross-sectional hexagonal shape of wax cells in honeybee hives may not be the result of natural selection but a geometric inevitability of packing the most cells into a given volume with the minimum of space between them. Cells that start out cylindrical in shape soon become hexagonal in cross-section due to close packing. **Physical constraints** are those imposed by the laws of physics or chemistry. All organisms are constrained by the effects of gravity, diffusion, thermodynamics, fluid dynamics *etc.* **Architectural constraints** are those imposed by the properties of the materials out of which organism are built, including their organization or structure. (p. 73)

The field of **biomechanics** is concerned with all three of the above, using the principles of mechanics to study the structure, function and motion of the mechanical aspects of biological systems, at all levels of organisation. Generally speaking, it is assumed that most organisms are well adapted, if not optimal, for the physical constraints imposed upon them; however there are notable exceptions, especially among modern humans. Due to the huge evolutionary advantage for human ancestors to have adopted a bipedal form of locomotion, tremendous pressure is now exerted on our lower vertebrae causing lower back pain in an estimated 80% of human adults, according to one estimate. (p. 73; Wikipedia: Biomechanics; Rowe, 2015)

Constraint driven research is not *anti*-adaptationist. Phylogenetic reconstructions, for example, are one means to discover adaptation. Phylogenetic trees in which convergences have been minimised will have remaining terminal branches that are likely to represent adaptations. Rosenberg and McShea suggest that biomechanics could be construed as "the study of the materials and the configuration of those materials that are immediately available to evolving lineages to solve the design problems the organisms in these lineages face". Constraint research programs and adaptationist programs are only alternatives in so far as they differ in interest or focus. Gould and Lewontin did *not* argue that selection and constraint offer alternative routes to adaptation, although this is a common misreading. Had they done so they would have been rejecting Darwin. Instead, they acknowledge that natural selection is the only explanation of adaptation (when it occurs). However, they do emphasize that there is more to an organism than its adaptations and that these other aspects are also worthy of study. (p. 74)

Rosenberg and McShea's fourth and final question above was: how the relationship between selection and constraint is to be properly understood? According to a heuristic proposed by Elliott

Sober and elaborated by Roger Samson (2003): constraint "proposes" and selection "disposes". *E.g.* Zebras are under major selective pressure because they are predated by lions. Zebras could exploit the full range of anatomical, physiological and behavioural options that are *conceivable* for them.

> Imagine not only zebras as they are, but zebras that are faster, or bigger, or have tougher hides, and so on. But don't stop there. The range of the conceivable includes zebras with vertical take-off ability, zebras with rear-mounted AK-47s, and zebras with predator-mind-control capability. The conceivable includes the full range of what is thinkable, however unrealistic or absurd. (p. 74)

The role of constraint, then, is to reduce the range of what is conceivable to that which can actually be produced by natural variation. Zebras with vertical take-off ability or mind-control powers are developmentally inaccessible or impossible due to biological, physical, architectural and biomechanical reasons. In short, they are ruled out by constraint leaving only true-to-life natural alternatives such as those that are better camouflaged, faster, larger, with thicker hides and so on. But these too have constraints. *E.g.* It is not possible for a zebra to be simultaneously much larger *and* swifter. (p. 74)

This is the stage at which natural selection operates to reduce the range of what is available to natural variation to what can actually survive and reproduce in a given environment. In other words, after constraint has eliminated what cannot be realised by natural variation, natural selection operates on what remains available to produce a few variants that are more fit that others – perhaps a larger, if less agile zebra. Rosenberg and McShea sum up the heuristic as follows:

> constraints culls the (truly enormous) range of the conceivable and offers up to selection the (much smaller) range actually available. Selection then culls the range of natural variation leaving the few that are more fit. Constraint proposes, and selection disposes. (p. 74 - 75)

This heuristic demonstrates how certain explanations in evolutionary biology depend as much on selection as on constraint. This is true in particular of questions concerning the cause or origins of a trait. If we were to ask, what is the cause of the large body size of zebras, constraint alone could not provide the answer because, in the absence natural selection, mediums sized and even miniature zebras would also exist. More generally, without selection every developmentally natural variant would exist, which is clearly not the case. (p. 75)

Conversely, selection alone is insufficient to address questions concerning the causes or origins of a trait. Without constraint, it is not clear that evolution even makes much sense. *E.g.* Without constraints, zebras with rear-mounted AK-47s or vertical take-off ability would be "fitter" than those merely of larger size or with thicker hides. Moreover without constraints, even the word "fitter" is not meaningful unless applied to a specified or potentially specifiable range of possibilities. "Thus the Sober–Samson heuristic reveals an asymmetry in the roles of selection and constraint in explaining the evolutionary origins of traits. Selection without constraint seems meaningless. Constraint without selection is merely unrealistic". (p. 75)

That selection logically depends on constraint is well understood; however according to Rosenberg and McShea, this may account for the backlash with which some adaptationists responded to Gould and Lewontin article. For some, the authors seemed to be saying that adaptationists overlook

constraint. Yet arguments about the cause or origin of a trait, constraint must, if only implicitly, be a part of such arguments because selection without constraint is meaningless. To such adaptationists it must have seemed that Gould and Lewontin were accusing them of arguing, absurdly, against something logically required by their explanatory strategy. (p. 75)

However, constraint and adaptation *can* sometimes pull in opposite directions. In such cases Gould and Lewontin's objection that adaptationists sometimes ignore constraint is credible. Consider, for example, evolutionary questions about what might account for a difference in certain traits. We can meaningfully ask what accounts for the difference in body size between the much smaller ancestral species of modern horses, *Hyracotherium* that lived some 50 million years ago, and horses and zebras alive today. In other words, we can ask what made the difference in equine evolution between some trait, such as small body size, then and some alternative trait, such as large body size, today. Either selection, constraint or some combination of the two could be the answer. Perhaps selection made the difference, favouring an increase in overall body size. Alternatively, it could be that selection favoured longer, swifter legs and that since leg length and overall body size are developmentally correlated, the increase in overall body size was the result of constraint. However it could be some combination of the two because there need not be single factor causes. (p. 75 - 76)

**What is Genetic Drift?**

Gould and Lewontin mention genetic drift as the first of several alternatives to adaptationism. According to Rosenberg and McShea, this has been the source of controversy in both evolutionary biology and its philosophy for much of the last century. Of genetic drift, Gould and Lewontin write:

> At present, population geneticists are sharply divided on the question of how much genetic polymorphism within populations and how much of the genetic differences between species is, in fact, the result of natural selection as opposed to purely random factors. Populations are finite in size and the isolated populations that form the first step in the speciation process are often founded by a very small number of individuals. As a result of this restriction in population size, frequencies of alleles change by *genetic drift*, a kind of random genetic sampling error. The stochastic process of change in gene frequency by random genetic drift… has several important consequences. First, populations and species will become genetically differentiated... in the complete absence of any selective force at all.
>
> Secondly, alleles can become fixed in a population *in spite of natural selection....*
>
> Thirdly, new mutations have a small chance of being incorporated into a population, even when selectively favoured… (Gould & Lewontin 1979; extract edited by Rosenberg & McShea 2008 p. 76)

Genetic drift refers to the stochastic or probabilistic change in the frequency of an existing gene variant (allele) in a population due to random sampling of organisms. Recall that the definition of the principle of natural selection (PNS) in Classic Text 26 is probabilistic. Sometimes, especially when populations are small, changes in gene frequency that do not necessarily lead to an increase in fitness may occur purely by chance. For the same reason, a fair coin may turn up tails six times in a row, purely by chance. According to Rosenberg and McShea, Gould and Lewontin's contemporary

evolutionary biologists did not deny this. Where they differed was the extent to which random drift was seen in the actual course of descent with modification that comprises evolution. Some biologists such as Gould and Lewontin argued for a significant role, while others have denied this or downplayed the effect. Even among the latter however, genetic drift is seen as important in certain evolutionary processes, like speciation. In the case of speciation due to geographical isolation, the appearance of a geographical barrier such as a mountain range, desert or seaway separates a subpopulation from the parent population, such that the smaller founder population continues to reproduce in isolation from the parent population. If the founder population is relatively small it is unlikely to carry the full range of genetic variation of the parent population. Moreover, novel combinations of gens are more likely to arise in smaller populations giving rise to novel phenotypes which may become fixed, purely by chance. Eventually, cumulative changes in the isolated founder population gene pool and range of phenotypes may become sufficiently different from the parent population that a new species emerges. More generally, changes in the distribution of traits, purely due to chance, can occur in populations of any size. Those that do not lead to adaptation are often characterised as the result of drift. However, the relative importance of such changes in shaping the evolutionary trajectories of most species, most of the time, is an empirical issue that is hotly debated. (p. 76 - 77)

Aside from the empirical issue, there are philosophical problems that arise from conflicting ideas about how drift works and how it arises. One idea is to treat drift and selection as independent evolutionary forces or causes that combine to determine the evolutionary trajectories of most species. On this interpretation, drift is seen as running counter to selection, slowing it down or preventing it from working altogether in determining evolutionary change. Another idea is to treat drift as reflecting the character of natural selection as a population-level process. On this interpretation, drift is always operating, because natural selection is a stochastic or statistical process that operates solely on populations and is not reducible to the heritable differences in individual fitness between competing organisms. On this view drift is like other statistical level phenomena, like entropy in thermodynamics that disappears if we fixate on individual molecules in a closed system. Yet another idea is to treat drift as an indication of our ignorance of all the selective forces that together determine a unique evolutionary outcome. These very different conceptions of drift lead to very different ways of understanding the theory of natural selection. (p. 77)

In dealing with these questions of drift Rosenberg and McShea return to the definition of the principle of natural selection (PNS) in Classic Text 26. Accordingly,

> *PNS*: For any two individuals, $x$ and $y$, if $x$ is fitter than $y$ in environment $E$, then *probably*, $x$ will have more offspring than $y$ in $E$.

The qualifier 'probably' in the above definition is the doorway to drift and is essential to the truth of PNS and its explanatory / predictive power. Thus, for example, we can predict that the larger the difference in fitness between $x$ and $y$, the more probably $x$'s offspring will outnumber $y$'s and the more probably this will happen sooner rather later. Similarly, we can predict that the larger the populations of $x$ and $y$, the higher the probability that $x$'s offspring will outnumber $y$'s. And when the population is large, the greater the variance in fitness, the sooner such demographic differences will appear. This is expressed by R.A. Fisher's (1930) **fundamental theorem of natural selection** as follows: "The rate of increase in fitness of any organism at any time is equal to its genetic variance in

fitness at that time". Later, Fisher (1941) restated his theorem more clearly: "The rate of increase in the average fitness of a population is equal to the genetic variance of fitness of that population". According to Rosenberg and McShea, the fundamental theorem of natural selection follows from the PNS along with certain assumptions about population size – that it should be very large or effectively infinite, and about the phenotypic effects of individual genes – that they should be small and gradual. (p. 78)

If, in general, population sizes were infinite and survived indefinitely we could dispense with the word "probably" in the definition of PNS; however they are neither and so the qualifier "probably" is required to render the definition true. But as we saw in Classic Text 26, this is the very qualifier that insulates the PNS from falsification because any counter-example to an evolutionary theory can be dismissed as a chance event and still be consistent with the theory. A similar problem of unfalsifiability arises if we ask whether a change in the traits of a lineage are the result of adaptation and not merely chance events. According to Rosenberg and McShea, "It seems that invoking drift in evolutionary explanation may expose evolutionary theory to the same charge of untestability that Gould and Lewontin raise against excessive adaptationism. (p. 78)

Hitherto we have been speaking of probability in a generic sense; however let us try to pin down exactly what sort of probability the PNS invokes. Firstly, there are **objective probabilities** in the world that are independent of mental states such as preferences, expectations, beliefs and desires. Then there are **subjective probabilities** which are the odds that a rational agent would assign to the outcome of a particular event, given the available evidence. In a sense both sorts of probabilities are "objective" in that they both obey the mathematical axioms and theorems of probability theory. Subjective probabilities in particular are described by Bayes' theorem because it reveals how ideally rational agents would recalculate or update their probabilities as new information becomes available. (See Critical Reasoning 10) (p. 78 - 79)

Of course, subjective probabilities only exist so long as there are rational agents to estimate their odds, or that would estimate their odds if they were to exist. According to Rosenberg and McShea, "Bayesian probabilities were first introduced to deal with statistical data in which it seemed difficult to define objective probabilities". We can, for example, calculate the objective probability of drawing a queen of hearts from a pack of playing cards because there are exactly 52 cards and only one queen of hearts. Therefore the probability is exactly $\frac{1}{52}$. However the objective probability of it reaching exactly 28.000… $^0$C on a clear summer's day in Paris is either zero or undefined because there are infinitely many objective thermometer readings between, say, 27.5 and 28.5 $^o$C. But the subjective probability of it reaching 28.000… $^0$C cannot be zero because it measures the strength of belief that some people have, or potentially could have, the relevant belief. The strength of such a belief could be very low, but it could not be zero because that would imply that some people believe that it is absolutely impossible for it to reach 28.000… $^0$C. (p. 79)

Yet Bayesian subjective probabilities are useful in biology because we could, for example, measure fitness in terms of the odds that a rational agent would assign to the number of viable offspring that a particular organism, or group of organisms, would leave. Biologists could also make subjective probability assessments about evolutionary outcomes. Because the process of evolution is so complicated and multifactorial, a biologist's rational appraisal of the odds of such outcomes may, at best, be in the form of subjective probability statements. However, few philosophers of biology

would be willing to regard the probabilities involved in the PNS as subjective. After all, the operation of evolution by natural selection has anteceded the appearance of rational beings on our planet by billions of years. Therefore the probability involved in the PNS must be objective, although even in a deterministic world in which biologists had perfect knowledge it would have to be statistically stated. And if the probability in the consequent of the PNS is objective, then so is drift as a force or factor or cause of evolutionary trajectories. (p. 79 - 80)

So, if dirt is a real and the probability in the PNS is objective, then what is it about the world that makes claims about such probabilities (and hence the magnitude of drift) true? According to Rosenberg and McShea, there are three kinds or sources of objective probability. Firstly, there are long-run relative frequencies such as repeatedly flipping a coin or drawing a card from a pack. Secondly, there is quantum indeterminism which is the claim that sometimes the most basic constituents of matter behave in an indeterministic manner. Although we may know the half-life of a given radioactive isotope, we cannot objectively predict which atom will be the next to undergo radioactive decay. Thirdly, there is the sort of probability reflected in the second law of thermodynamics, which states that the entropy of a closed system will probably increase over time towards a thermodynamic equilibrium.

The kind of randomness that characterises events at the level described by quantum mechanics has no cause or antecedent. No prior state of any atom or sample of atoms undergoing radioactive decay can be used to predict when it or another atom might do so. Few macroscopic, including biological, states are thought to arise directly from the probabilistic propensities of subatomic entities “percolating up” from the subatomic level. There are of course exceptions, such as the click of a Geiger counter or point mutations of genetic material caused by high energy gamma rays. The latter may have evolutionary significant outcomes; however biologists do not regard quantum indeterminism *sui generis* as a major additional factor contributing to the stochastic character of natural selection. (p. 80)

Instead, many biologists regard the statistically describable character of natural selection as more like infinitely long-run relative frequencies, except that the outcomes of interest in evolutionary biology are almost never repeatable. However, even if we stipulated that long-run relative frequency is what we mean by the concept of probability involved in the PNS, we would still want to know the source of that objective probability. *I.e.* “… what particular set of facts about the fitness to the environment of those particular organisms at that particular time produced the particular value of the objective chance that one would leave more offspring than the other[?]” (p. 81)

Compare the simpler question about the source of the objective chance that a particular coin would come up heads when tossed. Of course the odds would still be 0.5 even in a deterministic world in which there were no chance events. In the case of a coin, we might say that the source of the objective chance is due to a combination of an element of the set of all possible positions of a coin on a thumb and an element of the set of all possible values of momentum imparted by a thumb, and that the number of such combinations are equal. However this claim is problematic in more than one way. Firstly, the assumption that every physical possibility is equally probable is unfounded and more likely false. Not every toss (and not every state or position of matter) is equally likely. Yet we could still ask where the probability of each comes from and why the sum of their combinations should be equal. Secondly, the set of combinations of position and momentum is infinite, so the

probability of obtaining any particular combination is zero; therefore sum of such probabilities cannot be 0.5. Thirdly, since the types of infinities involved are the same, there is no telling which is the larger. If this is a real problem for coin tosses, it is an even greater problem for objective chances in biology. If we say that for $x$, the chance of producing more offspring due to the combinations of its set of traits is larger than that for $y$, due to the combinations of its set of traits, then the definition of what we mean by "larger" is still wanting. (p. 81 - 82)

The origin of the objective probability involved in drift is equally problematic. In the case of a sequence of coin tosses, drift is analogous to the sequence of tosses that depart from the proportion of 0.5 heads. Thus, a larger departure represents a larger drift and conversely a sequence of a tosses that approaches more closely to a proportion of 0.5 heads represents less drift. In the case of natural selection, drift refers to the changes of proportion of actual descendants that depart from the probable proportions determined by the PNS. Such departures are likely to be greater when the population is small and smaller when the population is large. Therefore, the source of the objective chance of an outcome in natural selection and its converse, drift, must be the same. (p. 81 - 82)

We can calculate the probability of drawing a particular card from a pack because the number of cards in a standard pack is always the same and the particular card we are interested in is unique, *i.e.* 1 of 52 ≈ 0,019. This probability is known *a priori*. No experiments are required to verify our calculation. However ecosystems are different. They are not composed of finite sets of equipossible outcomes; therefore particular outcomes cannot be calculated *a priori*. Likewise, we are not justified in assigning equal probabilities to "basic" outcomes since we do not know what or how many of them they are. Therefore the nature and source of the objective probability required by the PNS is unknown. (p. 82)

**Central Tendencies, Subjective Probabilities, and Theism**

If drift and differences in fitness together produce evolution, then we to need discover their source in the world. If we cannot, we may not be able to understand them or their relationship correctly, especially given the difficulty in empirically telling either apart. According to Rosenberg and McShea, the role of drift in the actual course of biological evolution and in any potential evolutionary change, is of interest, partly for historical reasons. According to the American geneticist Sewall Wright, evolution almost always requires a good deal of drift that occurs due to inbreeding within small populations in which genes, genotypes, and phenotypes are unrepresentative of the larger parent population. Wright reasoned that fitness differences within large populations were seldom great enough to produce a significant amount of evolution in the time available. In small populations however, repeated chance events affecting births and deaths will cause frequencies of particular traits to depart from the objective chances dictated by natural selection, just as fewer coin tosses will be more likely to depart form the expected value of 0.5 heads. R.A. Fisher, whom we have already met, disputed this. According to Fischer, differences in fitness are sufficiently large so that we do not need to evoke drift. At any rate, the dispute appears to be an empirical one that matters of fact should be able to resolve. (p. 82 - 83)

Consider the following scenario by John Beatty (1984) modelled on Bernard Kettlewell's well-known study of the microevolution of industrial melanism in the peppered moth. This species has light and dark variants. Since the Industrial Revolution, burning of coal in the English Midlands produced a lot

of soot which darkened trees on which the peppered moths settle. Kettlewell observed that during the coal burning era, the fitness differences between light and dark moths shifted in favour of the darker variants and back again when the UK reduced its burning of coal following World War II. Rosenberg and McShea propose the following scenario: Suppose that 40% of trees in a forest have light-coloured bark and 60% dark. Suppose further that trees of both colour are evenly distributed throughout the forest. Therefore dark coloured moths will be fitter in this environment. However, it is conceivable that in some seasons, dark coloured moths are more heavily predated by birds; perhaps because they happen by *chance* to land disproportionately more often of light coloured trees. Were this to happen in a given season, the proportion dark coloured moths will decrease, even though they are fitter in this environment of 60% dark coloured trees. A probabilistic PNS will indicate that this outcome is unlikely but not altogether impossible. And since it is unlikely we should consider the decline of dark moths during the season as an outcome of drift, not selection (p. 83)

But now suppose that during the same season, exactly the same number of white moths land on dark trees and are predated by birds. This time we are inclined say that natural selection is at work picking off the less fit white moths in a forest of predominantly dark trees. Given that the demographics are the same, what then makes dark moths landing on light trees a matter of drift and light-coloured moths landing on dark trees a matter of selection? There was an equal decline in numbers of both dark and light varieties during the period. According to Beatty, "the problem… is that of distinguishing between random drift and the *improbable results of natural selection*…" In Beatty's scenario, the decline in the population of dark-coloured moths in one season is a matter of drift because their darker colour solves a design problem, *i.e.* camouflage. For the same reason, the equal decline in the number of light-coloured moths is a matter of them being selected against. In real world instances of evolution by natural selection however, we have no prior access to such orderly design problems and solutions by which to differentiate drift from selection. On the contrary, according to Rosenberg and McShea, the line of inference goes in the opposite direction from demographic changes of populations to fitness differences between them. And, as shown in the above scenario, it is not empirically possible to distinguish drift from selection on the basis of the demographics alone. (p. 83 - 84)

It may be argued that if we only had prior knowledge of the design problems faced by individual members of a population and how they might be solved, we would be able to make (at least subjective) probability judgements about demographic changes over time. Many biologists and philosophers of biology however would point out that natural selection is not only about such "finely grained differences" between individual competitors in specific environments but rather about "central tendencies" in large populations. Besides which, drift and selection are inextricable features of populations, not discrete causes. Regarding the theory of natural selection as a claim about central tendencies in the evolution of populations was first proposed by the American philosopher C.S. Peirce in the 19$^{th}$ Century and taken up by R.A. Fisher in the 20$^{th}$. Both regarded the PNS and the equally probabilistic second law of thermodynamics as analogous. For gasses, the second law of thermodynamics states that, probably, the entropy of a given quantity of gas in a closed container will increase over time. However it makes no sense to speak of the entropy of an individual gas molecule because entropy is an attribute of the entire assemblage of molecules, although it does make sense to speak of a molecule's position and momentum (even though both cannot be simultaneously know with certainty). Similarly, the high probability that entropy will increase is not a function of the indeterminism of movement of individual gas molecules in the container but rather a

property of the entire assemblage molecules as they become more disorganized over time (or, improbably if they don't). Analogously, it makes no sense to speak about drift at the level of births, deaths and reproduction of individual organisms, even though they are the cause of population-level changes. Instead, drift is an attribute of the entire population of evolving organisms. When the population is large, drift is small and the central tendency in its evolution is a matter of selection. 'Drift' and 'selection' therefore designate population-level evolutionary tendencies. However, populations are never of infinite size; therefore there will always be some combination of drift as a population size effect as well as selection. (p. 84)

Notwithstanding, this still leaves us with the ontological question of where drift and its population level objective probability come from. The analogy with the second law of thermodynamics cannot help us further here, if only because philosophers of physics and physicists cannot agree where the probability involved in thermodynamics comes from either; though both agree that it is objective. According to Rosenberg and McShea,

> The behavior of gas particles is supposed to be perfectly deterministic, yet it produces objective probabilities about entropies. If the central tendencies in large populations are the aggregation of a huge number of individual cases of comparative fitness differences in the actual environments where animals compete and, in these cases, everything – including differential reproduction – is determined, then the same question must arise: where does the drift that inevitably obtains at population levels come from? (p. 85)

As far as we know, quantum indeterminism is largely irrelevant to macroscopic organisms; therefore we can assume that between any two given organisms, the difference in their fitness is fully determined by their specific environment, even though we do not know all the internal and external factors that determine the difference. Because our knowledge is incomplete, any proposed prediction or explanation will necessarily be subjectively probabilistic, reflecting the incompleteness of our knowledge. The apparent randomness of evolutionary processes is not stochastic; rather it is "pseudorandom" reflecting our inevitable ignorance. If we aggregate pair-wise fitness differences into lineage, population, and species fitness differences, and use this information to predict or explain evolutionary trajectories, our ignorance about the details is compounded; although the values of our subjective probability estimates may improve. If however these subjective probabilities are reflected in drift, then drift cannot be a separate "objective" evolutionary force that with selection determines the evolutionary trajectory of populations. In that case drift is simply a reflection of our ignorance of all the factors to which lineages are adapting over time. (p. 85)

Treating drift as reflecting subjective probabilities solves the problem of its origins, namely our ignorance; however it invites some serious objections, especially anthropocentrism. Could a matter of subjective probabilities be a force on a par with adaptation and/or constraint or constitute a serious alternative to biological process explaining evolution? Secondly, many events with significant evolutionary outcomes really do look completely random in their effects on reproduction. Consider flash floods, lightning strikes, earthquakes, continental drift, meteorite impacts, including the one that ended the age of the dinosaurs. Such events do not discriminate between organisms on the basis of their fitness under normal circumstances. The fact that the occurrence of such events is not vanishingly infrequent means that we must either add a *ceteris paribus* clause to the PNS, or recognise the inevitable probabilistic character of natural selection. Of course, we would never be

able to enumerate such a *ceteris paribus* clause in the form of a finite list of excluding conditions. However, the impact of such environmental processes need not be as dramatic as a meteorite collision to have an impact on reproductive rates without being a factor of a design problem that an ordinary organism might face. Moreover, they make the process of evolution objectively uncertain, not just probabilistic matter of our ignorance. (p. 85 - 86)

**Function, Homology, and Homoplasy**

Since the publication of *On the Origin of Species*, the term 'adaptation' has been entirely co-opted by Darwinists, so that it has become bound up with imposing a pattern of variation and selection on its former usage. Pre-Darwinian scholars however recognised two sorts of adaptation in nature and sought to explain them by an omnipotent designer. Firstly, there is the adaptation of different parts of the body to one another – the way they fit so harmoniously together. Secondly, there is the adaptation of living forms to their environment – the way that succulents are well-matched to deserts or polar bears are to the arctic. That adaptation is so significant to biology is reflected in its many terms, labels and predicates being functional, rather than structural, say. *I.e.* much of biology is defined in terms of cause and effect, rather than structure, specifically those effects that were selected for and that represent adaptations. Thus wings are not defined in terms of their anatomy or composition because they are so varied, but rater in terms of their effects. (p. 87 - 88)

But a wing produces many effects,

> … it adds weight, takes up space, usually makes an animal more visible, diffuses heat, and casts shadows (and so sometimes alerts prey and other times reduces the prey's view of the winged predator). But among all the effects of a wing, there is one or a small number that defines it: its ability to produce flight (or its homology with a structure that produced flight in an ancestor – think of penguins). So, wings are defined in terms of one of their effects on some organism that has wings. Which effect? The one that performs a function that some animal employs to deal with a design problem presented by the environment – [locomotion] in most cases. (p. 88)

Aside from wings, many structural terms in biology are named after the adaptive process they perform. This is true across all levels of organisation from the

- molecular, *e.g.* codons, introns, transcription factors, genes, and enzymes; to the
- anatomical, *e.g.* organelles and organs such as flagellum, vacuole, valve, vessel, heart; to the
- ecological, *e.g*. predator, parasite, reproduction, altruism, *etc*.

As Rosenberg and McShea point out, these structures look like they subserve purposes of the larger systems that contain them. Furthermore, the language used to describe them sounds distinctly goal directed or teleological. As we saw in Classic Text 22, the major problem with goal directed or teleological causation is that the effect seems to precede the cause, one that modern scientific thought rejects. Consider Harvey's discovery of the circulation, including the function of the heart to pump blood. Harvey's discovery certainly seems to explain why vertebrates have hearts – in order to pump blood. But how can the cause of having a heart be a property of hearts? Surely the cause of a thing must precede the thing itself. Unfortunately, talk of function becomes deeply problematic if

we expunge purposes, goals, ends, and other final causes from scientific discourse. Even the most mechanistic biological opponent of teleological discourse still believes that vertebrate hearts exist *in order to* pump blood, that the *purpose* of enzymes is to catalyse reactions and that the *function* of leopards' spots is for camouflage. (p. 88)

According to Rosenberg and McShea, one of the preoccupations of the philosophy of science between the end of the 1940's and the early 1970's was to rid biology of its functional terminology, with its time reversed teleological causality, while acknowledging the very real difference in terminology and phenomenology between biology and the physical sciences. Even though biological systems are composed of physical matter, there appear to be no "functions" in the physical sciences. The apparent contradiction was resolved by Larry Wright (1972, 1973) who recognised that Darwinian natural selection could play a role in providing teleology-free functional explanation. That functions explain the presence of traits (or behaviours) is a matter of their **aetiology**, *i.e.* the historical circumstances of their emergence. And that vertebrates have hearts in order to pump blood is the result of their aetiology or prior causal history of ancestral hearts or heart-like organs that were randomly varied and successively selected for their fitness enhancing capacity (in ancestral environments) in causing the circulation of blood. Vertebrate hearts are not a teleological cause; instead the pumping of blood was a *consequence* or effect of ancestral heart-like organs that were *selected for* in the course of evolution. Rosenberg and McShea point out that the italicised terms above are often employed as labels in the analysis of functional explanations that exploit *consequence etiologies* and identify *selected effects*. (p. 88 - 89)

Once we understand Wright's point about functional explanation, it is easy to see how the analysis of how such explanations work can be extended to account for the meaning of functional terms and concepts in biology. We are already familiar with the distinction between types and tokens from Classic Text 16. *E.g.* The category or type, 'heart' is exemplified by a large number of particular organs in the bodies of many animals, including vertebrates. Presently, there are over 8 billion tokens of the type 'human heart' and a much larger number of tokens of the type 'mammalian heart' beating on Earth. Now consider a particular heart, Charles Darwin's, say. The function of Darwin's heart was to pump blood because (as a matter of aetiology) organs of this type *were selected for* owing to their capacity to pump blood. In general then, a token organ or biological structure has one or more functions in virtue of its type having a natural selection aetiology. Note that Darwinian natural selection is not the only sort of aetiology that can confer functions. Artefacts such as spears points, hand axes and hide scrapers have functions as tools in virtue of an aetiology that reflects a history of human intentions, desires, and designs. The difference between natural functions and artefactual functions is in their aetiologies. Furthermore, a trait may have a label that no longer reflects its current function but some other function that its ancestors fulfilled. Recall Gould and Vrba's exaptations. *E.g.* a penguin's wings do not support flight but are very good for swimming, which explains why penguins have retained these appendages. Then there are some biological traits or vestigial structures that have no present function, such as human male nipples or "wisdom" teeth, even though their names reflect some adaptive aetiology. (p. 89 - 90)

Furthermore, as Ruth Millikan (1984) pointed out, there are some biological traits or structures that are functionally characterised, even though they fail to have the effects which would have been the outcome of their aetiology. Most white-oak acorns, for example, fail to germinate or function as they should. Yet they are still characterised as seeds in what Millikan and Neander call the "proper"

or “normal” functional sense, because their existence is due to the successful functioning of tokens like them in their aetiology. (p. 90)

Rosenberg and McShea regard “proper” or “normal” in this sense as evaluative or normative notions:

> What is “proper” for a given trait is not its actual structure in any particular organism or even the mean or typical structure of it in the species as a whole, but rather the structure of the trait that was selected for in the trait’s etiology. This of course makes “normality” relative to a selective environment, a target that moves as environments change. (p. 90)

The question of “normality” is significant in bioethics where it is often important in distinguishing treatments from enhancements. A treatment may involve the removal or repair of clearly dysfunctional traits, whereas enhancement is the modification of traits that are functioning normally. Thus treatments are considered as morally obligatory, while enhancements are considered optional or discretionary. However, the standard of normality varies according to environments with different trait distributions, including different social values. *E.g.* Supplying a course of human growth hormone injections to a teenager of significantly less than average stature, but with a normally functioning pituitary gland, may be considered enhancement under certain circumstances but treatment under others. This is not a question of subjectivity but depends very much on whether the shorter stature is considered “normal” or not, as a function of the social environment in which the teenager finds themself. (p. 90)

Wright’s aetiological or **selected effects** (SE) **analysis of function** is useful because, *inter alia*, it allows biologists to interpret their functional terminology as a literal account of the world, without appealing to notions of teleology and hence backward causation. Running concurrently to Wright’s analysis was Robert Cummins (1975) so-called **causal role** (CR) **account of functions**. First advanced as a as a way of distinguishing functional from anatomical or structural terms in psychology and cognitive science, CR has subsequently been embraced by several philosophers of biology. While some have contrasted CR and SE analyses, others regard both as compatible theories picking out different aspects of function at work in biology. According to Cummings’ analysis of functional description and explanation, terms such as ‘heart’ or ‘gene’ refer to ‘nested capacities’ which are components of larger systems to whose behaviour they make a causal contribution. Such behaviour may or may not be goal directed; however the term itself makes no reference to teleology. According to Cummins, attributing a function $F$ to $x$ is relative to an “analytic account” of how $x$’s functioning $F$ contributes to the “programmed manifestation” of some more complex capacity by a system that contains $x$. Thus, the concept of a ‘gene’ is a functional one, not because its sequence of nucleotides has some selected function, though surely it does in a reductionist sense, but because the function of the sequence *as a gene* is relative to an analytical account of how the sequence’s capacity, to encode and transcribe the primary sequence of a protein or polypeptide, contributes to development and hereditary capacities of the organism that contains it. (p. 90 - 91)

Cummins’ account of function is unintentionally wider than that of Wright’s. According to the former, the nested causal capacity in which being a function consists can be realised by any number of non-biological systems in which contained capacities contribute to the manifestation of containing capacities. *E.g.* an analytical account can be given of how the position and composition of boulders in a stream contribute to the capacity of the stream’s rapids to upend canoes, or for how

hydroelectric turbines make it difficult for salmon to swim upstream, even though no one would suppose that such are the functions of boulders or turbines. Rather than modify or abandon the causal role account, defenders of the theory have argued that such an objection is "not a bug but a feature[2]". *I.e.* functional attributions lie on a continuum from less to more interesting, largely reflecting the complexity of contained and containing capacities; and that biological systems require such a teleology-free analysis of functional description. (p. 91)

Furthermore, advocates of CR analysis point out that there are sub-disciplines within biology such as anatomy and palaeontology in which it is important to be able to ascribe functions to structures with nested capacities without having to commit to their selected effects etiologies either way. On this view, stating and testing alternative hypotheses about the consequences of aetiologies requires that describing traits that feature in such hypotheses must be done in a way that is neutral with respect to such aetiologies. Consider, by way of example, whether traits in different organisms are homologies or homoplasies[3], *i.e.* whether they are the result of common descent or independent convergent evolution. According to Rosenberg and McShea,

> wings have evolved 40 or more separate times. Accordingly, each of these instances has its own unique consequence etiology. In one instance, the wing may have been favored originally as an organ for heat dissipation. In another it may have evolved as a sexual signal, later co-opted as an exaptation for flight. (p. 91)

Thus, it is not simply a matter of asking whether the wings in any two of these instances are homologous or convergent. The term 'wing' is a functional one, which according to SE theorists, refers to its selective history, which may be unknown. Therefore, according to CR theorists, we cannot simply pose the question of homology *vs*. convergence because we cannot be sure that both species have wings in the same functional sense. Instead, we must first be able to identify functions in common in structures prior to knowing anything about their aetiologies. However, both SE and CR functions must be feasible in biology according to the context in which they are invoked. Indeed, this is consistent with Gould and Lewontin in maintaining a more self-conscious CR approach to functional attribution as a remedy to extreme adaptationism allowed by the SE conception of function. (p. 91 - 92)

Not all SE theorists would agree to this compromise. Some may go on to argue that CR analysis presupposes the SE analysis of functions, or at the very least, that every CR function in biology is, in fact, a selected effect, which is the result of a consequence aetiology. Either way, the evolutionary biologist's distinction between homologies and homoplasies must be accommodated (p. 92)

Consider the claim above by CR analysts that the distinction between homology and homoplasy requires neutrality with respect to 'selected effects' aetiologies by the kind of terms in which we describe a trait of evolutionary interest. SE theorists will concede that some aetiologies are more generic and others more specific but that disputes over homology *vs*. homoplasy must always assume some common aetiology. "That", according to Rosenberg and McShea, "is the point of

[2] "It's not a bug, it's a feature" is an excuse sometimes used by software developers when they try to convince a user that a flaw in their program is actually what it is intended to do.

[3] **Homoplasy** is the development of organs or other bodily structures within different species, which resemble each other and have the same functions, but did not have a common ancestral origin. These organs arise via convergent evolution and are thus analogous, not homologous to each other. (SinceDirect.com)

Darwin's claim that every creature on the Earth is a twig on the tree of life". Consider the eye which has evolved independently as many as 40 times during metazoan development. The gross anatomical differences between them and the diverse ways in which they realise the CR function of sight suggest quite distinct consequence aetiologies. However on a molecular level, the PAX6 gene family which encodes the PAX6 protein acts as a "master control" gene for the development of CR-functionally divers eyes (and other sensory organs). The PAX6 protein function is highly conserved across bilaterian species from insects to vertebrates suggesting that underlying these definitive convergences, such eyes do share an important consequence aetiology, if we go back far enough. According to Rosenberg and McShea,

> The SE analysis of functions never commits the biologist to any particular etiology, only to the generic claim that each item in biological taxonomy has some etiology or other, that if we go back far enough all traits share parts of a shorter or longer consequence etiology. Thus, when we ask whether the wings of two phylogenetically distant species are homologous or homoplastic, the SE theorist claims that we are asking how much of their consequence etiologies overlap and how recently they overlapped. And this way of understanding the distinction between homologies and homoplasy requires no further notion of function beyond the SE notion. (p. 92)

Furthermore, SE theorists may question whether CR analysis implicitly adopts the SE account despite the claim that it is an incompatible alternative to the analysis of function motivated by Wright. Recall Cummins' original CR definition of function by the 'analytic account' of how $x$'s functioning $F$ contributes to the 'programmed manifestation' of some more complex capacity by a system that contains $x$. In the case of artefacts, we understand that the programming is accomplished human design, intentions, plans *etc.* However, prior to the appearance of cognitive agents capable of programming manifestations of the required kind, it is clear that only Darwinian blind variation and environmental filtration could have provided for the CR function of features such as the elephant's trunk or the panda's thumb. If so, then every biologically interesting CR function will have a consequence etiology that is an SE function. And being so also makes it a CR function but not *vice versa*. The reason for the asymmetry is that the history of successive improvements embodied in a consequence etiology that are not nested in the way required by a CR account can be programmed in such a way by variation and selection. (p. 92 - 93)

On the other hand, the CR theorist could reply that parts of an organism could become connected in some way, purely by chance, so as to serve some novel CR function, even before selection has had an opportunity to act *i.e.* before any SE aetiology. Indeed, it has been argued that all SE functions are CR functions at their outset, before there has been any chance for a history of selected effects. On this interpretation, a thermoregulatory appendage that first helped loft an organism, even for a moment, was acting as a wing in the CR sense, but not yet in the SE sense. (p. 93)

Although such a distinction is something that would probably only interest philosophers, there are implications for professional biologists from both sides of the debate. From the CR theorist, the CR point of view is important for the biologist because it draws attention to the key role that constraint plays in evolution. Recall Gould and Lewontin's (1979) proposal that brain size was driven by selection for body size. If correct, then up to a point, our brains may have had little or no pre-hominid history of selection for large size, and hence no SE functionality. In spite, or because of this,

constrained increase in brain size could have yielded enormous CR functionality, including much of our behavioural and social complexity. More generally, constraint can yield transformations that are by chance functional in a CR sense. And such functionality can be maintained even in the absence of active selection. A combination of constraint and CR functionality could be a hitherto overlooked major source of novelty in evolution. (p. 93)

From the SE theorist perspective, adopting the SE approach will be advantageous for the behavioural and social sciences in which functional language and explanations are rife, though with no more justification to invoke final causes to explain functional explanations and descriptions than biology once did. Like biology, they will have to come up with an efficient cause–consequence aetiology for the functions they invoke. This does not commit the SE theorist to selective explanations at any particular level because functional behaviour could be the outcome of selective mechanisms such as learning or synaptic pruning. According to Rosenberg and McShea, The point is that, for an SE theorist, to explain function in nature, Darwinism at some level is "the only game in town". (p. 93)

Finally, Rosenberg and McShea have used the terms 'random', 'stochastic' and 'predictable / unpredictable' somewhat loosely. We have not attempted to edit them in their text; however we provide the following table of their correct meaning and usage:

| | Random | Pseudorandom | Non-random |
|---|---|---|---|
| **Processes** | Stochastic | Chaotic | Classical |
| **Example** | Radioactive decay of a specific atom | Random number generators *e.g.* a die | Swing of a pendulum |
| **Predictability** | Not predictable | Predictable in theory but not in practice | Predictable |
| **Cumulative predictability** | Probabilistic | Probabilistic | Predictable |

A few points to note: While radioactive decay of a specific atom is not predictable, even in theory, the roll of a dice would be predictable if, like Laplace's Demon, we knew the starting positions and momentum precisely and had an unlimited powers of calculation; however we never do. Therefore, the roll of a die is not predictable in practice. The same is true of the swing of a double-pendulum (a pendulum with another pendulum attached to its end); however the motion of a single pendulum is as predictable as clockwork (literally).

**Task**

Both the substance and rhetoric of Gould and Lewontin (1979) come under heated attack in Daniel Dennett's *Darwin's Dangerous Idea* Chapter 10 (1995). How is it that these intellectual giants, who are staunch advocates of Darwinism, could have failed to agree or at least come to a compromise on the topic? What does that say for the public understanding of biology?

**Feedback**

Unfortunately, there is no easy response to the above challenge. Indeed Rosenberg and McShea simply leave Dennett's attack as one of several "suggestion for further reading". Please consult.

We believe that as with Dennett's (1991) *Consciousness Explained* and (1995) *Darwin's Dangerous Idea* Chapter 10, that both were valiant attempts to popularize difficult biological/philosophical problems that left the layperson with more questions than answers. Obviously, this doesn't auger well for the public understanding of biology, which as it is, receives very little attention.

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