

*Evolutionary theory, causal completeness,  
and theism*

*The case of “guided” mutation*

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Michael Ruse (2004) and I (Sober 2010, 2011) are both “accommodationists.” We think that evolutionary theory and some types of theism can be reconciled. We also have in common the negative fact that neither of us is a theist.

It is obvious that some kinds of theism are logically inconsistent with evolutionary theory; it is equally obvious that other kinds of theism are logically compatible with that theory. Young Earth Creationism says that life on Earth began some 10,000 to 50,000 years ago by God’s separately creating each species (or each “basic kind” of organism). Evolutionary theory says that life began about 3.8 billion years ago and that all current species are genealogically related. There is no reconciling evolutionary theory with this form of theism; one must be wrong if the other is right. At the other end of the spectrum is deism, which is the view that God created (1) the universe, (2) the laws of nature, and (3) the initial conditions of the universe, and then sat back, allowing everything that happens in nature to be a consequence of those three items. Evolutionary theory fails to conflict with this form of theism for the simple reason that the theory says nothing at all about the origin of the universe, or its initial state, or where the laws of nature come from.

The kind of theism that interests me lies in between these two extremes. I am interested in interventionist<sup>1</sup> theisms that say that God not only

I gave lectures on this material at Florida State University, the University of Chicago, the University of Connecticut at Storrs, and the University of Cambridge. I am grateful to members of these audiences for useful discussion. I also thank Steve Nadler and Mike Steel for their comments.

<sup>1</sup> I am using the term “intervention” in a way that is broader than what many theologians prefer. They often use the word to name only the piecemeal local effects that God has concerning the details of some small aspect of the universe’s total history. They prefer to use the term “interaction” for divine causation that influences large-scale, global features of the universe. If God parted the Red Sea, this would be for them an intervention in the narrow sense; if God sustains the whole universe from each moment to the next, this is an example of an interaction, not an intervention. I am using the term “intervention” in a broader sense, so that it applies both to narrow-sense interventions and to interactions.

produced the (1)–(2)–(3) that Deism describes, but also intervened in nature after the universe's beginning. Traditional Judaism, Christianity, and Islam (if I may use the loose term "traditional") are theisms of this sort. My question is whether a God that intervenes in human history, and in nature more generally, can be reconciled with evolutionary theory. I won't discuss all of the interventions that an interventionist might want to endorse. Rather, I'll limit myself to the idea that God intervenes in the evolutionary process by causing this or that mutation to occur at a given time and place.<sup>2</sup> I say that I am interested in interventionist theisms, but this is not because I believe any of them (recall my first paragraph). I am interested in them because many religious people accept interventionist theisms and think that this obliges them to reject evolutionary theory. Biologists say that they have abundant evidence that mutations are unguided. This seems to mean that God does not intervene in the evolutionary process, at least not by causing this or that mutation to occur. I'll argue that what biologists mean, or ought to mean, when they say that mutations are unguided says nothing about whether God ever causes a mutation to occur.

The argument I'll give for thinking that evolutionary theory is logically compatible with this type of divine intervention is simple; it relies just on the fact that evolutionary theory, properly understood, is a probabilistic theory. The argument therefore generalizes; it applies to pretty much *any* probabilistic theory, not just to evolutionary theory. What I'll say in what follows expands on what I have said in Sober (2010, 2011) by filling in some details and by replying to some objections. Although I'll argue that evolutionary theory, properly understood, does not rule out God's causing some mutations, the theory does rule this out when you add something to it. But the something else is a philosophical thesis, not a scientific theory at all.

### Evolutionary theory and determinism

Evolutionary theory, in its application to finite populations of organisms, is a probabilistic theory. The theory does not tell you what must happen in the future, given a description of the population's present state. Rather,

<sup>2</sup> This idea has a history, beginning with Darwin's relationship with the Harvard botanist Asa Gray. Gray was Darwin's foremost advocate in North America, but he urged Darwin (both in print and in their extensive and congenial private correspondence) to add "in the philosophy of his hypothesis, that variation has been led along certain beneficial lines" (Gray 1888). Gray had God in mind as the agent that was doing the guiding. Darwin always demurred. It is worth mentioning that both deists and interventionists can maintain that God has arranged for this or that mutation to occur; the difference is that deists think that God does this indirectly while interventionists think that God acts more directly.

it tells you that different futures are possible and assigns a probability to each. This fact about the theory isn't immediately obvious when you look at various simple mathematical models of the evolutionary process. For example, consider a haploid population in which there are just two alleles  $A$  and  $B$  at a locus that have frequencies  $p$  and  $q$  and constant fitnesses  $w(A)$  and  $w(B)$ . The change in trait frequency in the next generation is (Crow and Kimura 1970, 179):

$$\Delta p = \frac{spq}{\bar{w}}$$

The selection coefficient  $s$  represents the fitness difference [ $w(A) - w(B)$ ] and  $\bar{w}$  is the average fitness of individuals in the parental generation. This equation says what will happen, not just what will probably happen. This is because the model describes an infinite population. As soon as you take account of the fact that real populations are finite, you need to reinterpret this equation; it tells you about the *mathematical expectation* of the change in trait frequency. The fitnesses (and so the selection coefficient) are probabilistic quantities; in a finite population there are different possible changes in trait frequency that might ensue, each with its own probability; the expected value of  $\Delta p$  is the probabilistically weighted *average* of the changes that might ensue.

What does the probabilistic character of evolutionary theory (for the case in which populations are finite) tell you about whether determinism is true? Determinism is the following thesis:

Determinism: A complete description of the history of the universe up to time  $t$  uniquely determines what the future of the universe will be after time  $t$ .

If you assume that the history of the universe has the Markov property, this formulation can be replaced with something that is logically stronger:

Determinism<sub>M</sub>: A complete description of the universe at time  $t$  uniquely determines what the future of the universe will be after time  $t$ .

In both formulations, a complete description of the past leaves open just one possible future; this is the one that must happen. Indeterminism, on the other hand, says that a complete description of the past leaves open multiple possible futures, each of which has its own probability. I'll use the Markovian formulation of determinism in what follows.

To see what evolutionary theory says about determinism, let's switch to an easier question. When we toss a coin we usually assume that the coin is fair, meaning that

(1)  $\Pr(\text{the coin lands heads at } t_2 \mid \text{the coin was tossed at } t_1) = 0.5.$

Notice that this conditional probability describes a relationship between two propositions, the ones that flank the conditional probability sign “ $\mid$ ”. Given just that you toss the coin at  $t_1$ , the probability of getting heads at  $t_2$  is  $1/2$ . Although proposition (1) is true (let us suppose), it says nothing about whether determinism is true because (1) does not say, one way or the other, whether “the coin was tossed at  $t_1$ ” is a complete description of what is true at that time. It is perfectly compatible with proposition (1) that the following is true:

(2)  $\Pr(\text{the coin lands heads at } t_2 \mid \text{a complete description of the state of the coin-tossing set-up at } t_1) = 1.0.$

Proposition (1) also is logically compatible with the probability described in (2) having an intermediate value (in which case determinism would be false). Do not make the mistake of thinking that there is one true probability that the coin has of landing heads at  $t_2$  and that therefore (1) and (2) are in conflict. Probability is like distance. There is no such thing as the one true distance to Madison; there's the distance from Omaha to Madison and also the distance from Atlanta to Madison. Probability, like distance, is an inherently relational concept (Sober 2011).<sup>3</sup>

If (1) and (2) are both true, (1) provides a *causally incomplete* representation of the coin-tossing system. By this I don't mean that (1) fails to mention all of the many causes that affect the outcome of this coin toss, a set of events that traces back into the past all the way to the origin of the universe. Rather, what I mean by causal incompleteness is this:

$X$  at  $t_1$  is a causally incomplete description of whether  $Y$  is true at  $t_2$  if and only if there exists a proposition  $H$  that is true at  $t_1$  such that  $\Pr(Y \text{ at } t_2 \mid X \text{ at } t_1) \neq \Pr(Y \text{ at } t_2 \mid X \text{ at } t_1 \ \&\ \& \ H \text{ at } t_1).$ <sup>4</sup>

If (1) is true but causally incomplete, there is a “hidden variable” ( $H$ ). This means that there is a causally relevant factor at  $t_1$  that is “hidden” in the

<sup>3</sup> Renyi (1970, 34) says that “every probability is in reality a conditional probability” and puts this sentence in italics, for emphasis. Renyi's idea was that “conditional probability” should be taken as primitive, and that a concept of unconditional probability could then be derived from it. Current practice is mainly to do the opposite. My idea that probabilities are relational pertains to both conditional and to so-called unconditional probabilities. Unconditional probabilities make sense when they are based on a model;  $\Pr_M(A)$  involves a relation between propositions just as much as  $\Pr_M(A \mid B)$  does. My thesis of relationality has an exception: tautologies and contradictions can be said to have their probabilities (0 and 1, respectively) nonrelationally.

<sup>4</sup> I assume that both of the probabilities in this inequality are well defined. Note that if determinism is true, then a causally complete model must be deterministic, whereas if determinism is false, then

sense that it goes unmentioned in (1). Probability statements like (1) can be true without being causally complete.<sup>5</sup>

There is nothing special about this coin-tossing example. *Any* probability statement you please (perhaps with the exception of the laws of quantum mechanics, which raise special questions that I won't address here) can be true without being causally complete. Evolutionary theory, I've suggested, is a probabilistic theory. This means that it can be true without being causally complete. The theory doesn't rule out the possibility that there are hidden variables. This means that it, like many other probability statements, doesn't rule out the possibility that there are *supernatural* hidden variables.<sup>6</sup>

### Guided mutations

Biologists now know vastly more about the mutation process than Darwin did. They often summarize this knowledge by saying that mutations are "unguided." This seems to entail that no one guides mutations, not even God. Here I'll explain why our scientific knowledge of mutation, properly understood, does not entail that God never guides mutations.

In his book *The Variation of Animals and Plants under Domestication*, Darwin (1868, 249), without any knowledge of Mendelism, managed to characterize what remains the modern understanding of the biological idea that mutations are unguided:

Let an architect be compelled to build an edifice with uncut stones, fallen from a precipice. The shape of each fragment may be called accidental; yet the shape of each has been determined by the force of gravity, the nature of the rock, and the slope of the precipice, — events and circumstances all of which depend on natural laws; but there is no relation between these

a causally complete model cannot be deterministic and still be true. I also should mention that the concept of causal completeness (like the concept of determinism itself) requires one to be careful about how one understands a description of what is true "at a given time." If you pack the whole future of the world into what you call "a description of the system at time  $t_n$ ," then determinism is trivially true. This logical trick should not be taken to show that the question of determinism is silly. This point about choice of descriptors is made by Earman (1986), among others.

<sup>5</sup> It is interesting that a deterministic theory, if true, must be causally complete. Suppose that if  $C_1, C_2, \dots, C_n$  occur at  $t_n$  then  $E$  must occur at  $t_n$ . It follows that  $\Pr(E \text{ at } t_n \mid C_1, C_2, \dots, C_n \text{ at } t_n) = 1$ . Since probabilities of 0 and 1 are "sticky," there can't be a hidden variable at  $t_n$  that, when included in the conditioning proposition, confers on  $E$  at  $t_n$  a probability that differs from 1. In contrast, an indeterministic theory can be true without being causally complete.

<sup>6</sup> My argument has points of contact with ideas that Russell (2008) developed for a view he calls NODA (noninterventionist objective divine action); one difference is that he defends the existence of supernatural hidden variables, while I am merely noting their compatibility with evolutionary theory.

laws and the purpose for which each fragment is used by the builder. In the same manner the variations of each creature are determined by fixed and immutable laws; but these bear no relation to the living structure which is slowly built up through the power of selection, whether this be natural or artificial selection.

“Unguided” does not mean *uncaused*. What it does mean is that mutations do not arise because they would benefit the organisms in which they occur. Once again, an analogy with gambling devices is apt. Coins land heads or tails when tossed and these outcomes have their causes, but one thing that does not influence the outcomes is that gamblers have bet on them.

To understand more precisely what is involved in the biological claim that mutations are unguided, I want to consider a simple experiment (Sober 2011). Imagine a species of blue organisms; we’ll take a large number of them and put them in a red environment and an equally large number of those blue organisms and put them in a green environment. We will monitor how often those blue organisms mutate to red and how often they mutate to green in each of the two environments, thus obtaining the data shown in Table 2.1. The experiment involves a large number of blue organisms so that we get mutation frequencies different from zero (mutation probabilities are small). And there is one more detail: these organisms gain a selective advantage from matching their environments. Red organisms outsurvive green organisms in a red environment, but the reverse is true in a green environment. This might be because predators use color vision to hunt for the organisms we’re talking about, or because human experimenters follow the protocol of allowing organisms that match their environment to survive and reproduce while preventing non-matching organisms from doing so.

What does a model of guided mutations say about this experiment? A model of this type will describe how the probabilities of various mutations are influenced by what would be good for the organisms in question. The model proposes the following two probabilistic inequalities:

$$(G) \quad \begin{array}{l} \Pr(\text{red mutation} \mid \text{red environment}) > \\ \Pr(\text{red mutation} \mid \text{green environment}) \\ \Pr(\text{red mutation} \mid \text{red environment}) > \\ \Pr(\text{green mutation} \mid \text{red environment}). \end{array}$$

These two inequalities are logically independent of each other. The first says that blue organisms have a higher probability of mutating to red in a red environment than they have of doing so in a green environment. The second says that, in a red environment, red mutations have a higher

Table 2.1 *The frequencies with which blue organisms mutate to red and to green in each of two environments*

|                | Red environment | Green environment |
|----------------|-----------------|-------------------|
| Red mutation   | $f_1$           | $f_2$             |
| Green mutation | $f_3$           | $f_4$             |

Table 2.2 *The model of guided mutations entails four probabilistic inequalities. Below are probabilities of the form  $\Pr(\text{mutation} \mid \text{environment})$* 

|                | Red environment |   | Green environment |
|----------------|-----------------|---|-------------------|
| Red mutation   | $p_1$           | > | $p_2$             |
|                | $\vee$          |   | $\wedge$          |
| Green mutation | $p_3$           | < | $p_4$             |

probability of arising than do green mutations. Both inequalities are needed to express what the idea of guided mutation means. The first, by itself, could be true just because red environments are more mutagenic than green environments. The second, by itself, could be true just because red mutations are more probable than green ones, regardless of the environment. To complete our statement of what the guided model says about our experiment, we need to add two further inequalities:

$$\begin{aligned} & \Pr(\text{green mutation} \mid \text{green environment}) > \\ & \quad \Pr(\text{green mutation} \mid \text{red environment}) \\ & \Pr(\text{green mutation} \mid \text{green environment}) > \\ & \quad \Pr(\text{red mutation} \mid \text{green environment}). \end{aligned}$$

Table 2.2 summarizes the four inequalities that the model of guided mutations proposes. Imagine that each of the four squares in the 2-by-2 table has a height above the surface of the page that represents how big the probability is of the mutation it describes. The model of guided mutation says that there are two peaks and two valleys in Table 2.2.

What does a model that *denies* guided mutation say about our experiment? There are many such models; each of them denies one or more

of the four inequalities displayed in Table 2.2. The simplest of all these deniers is a null model. It says that all the probabilities in Table 2.2 are equal:

$$\begin{aligned}
 \text{(N)} \quad & \Pr(\text{red mutation} \mid \text{red environment}) = \\
 & \quad \Pr(\text{red mutation} \mid \text{green environment}) \\
 & \Pr(\text{red mutation} \mid \text{red environment}) = \\
 & \quad \Pr(\text{green mutation} \mid \text{red environment}) \\
 & \Pr(\text{green mutation} \mid \text{green environment}) = \\
 & \quad \Pr(\text{green mutation} \mid \text{red environment}) \\
 & \Pr(\text{green mutation} \mid \text{green environment}) = \\
 & \quad \Pr(\text{red mutation} \mid \text{green environment})
 \end{aligned}$$

I use the term “null” because this model says there is no difference between any two of these probabilities.

What is the result of our experiment? What won't happen, if this experiment is like the many more sophisticated experiments that biologists have carried out, is that the frequencies described in Table 2.1 exhibit inequalities that “mirror” the inequalities among probabilities depicted in Table 2.2 (where the differences among these observed frequencies are statistically significant). Biologists will conclude from these non-mirroring observations that the guided model (G) is inferior to some model or other that disagrees with (G); perhaps the null model (N) turns out to be the best of these alternatives.<sup>7</sup>

It is experiments and observations of the sort just described that underlie the conviction that biologists have that mutations are “unguided.” Suppose the null model (N) is true for the experiment we've been considering. The point to notice here is that the equalities stated in (N) can be true without any of the probabilities mentioned in (N) being *causally complete*. Consider, for example, one of the probabilities discussed by the null model,  $\Pr(\text{red mutation} \mid \text{red environment})$ . There is no reason to think that the occurrence of a red mutation in our blue organisms isn't influenced by causal factors that go unmentioned in this probability. For example, maybe ambient temperature is relevant, with the consequence that

$$\begin{aligned}
 & \Pr(\text{red mutation} \mid \text{red environment}) \neq \\
 & \quad \Pr(\text{red mutation} \mid \text{red environment} \ \& \ \text{hot}).
 \end{aligned}$$

<sup>7</sup> Although fit-to-data is an important consideration in evaluating these models, there is something more – the number of adjustable parameters the different models contain. Model selection criteria like the Akaike Information Criterion take both properties of the model into account (see Sober 2008 for further discussion). Notice that the null mode (N) has fewer adjustable parameters than the guided model (G).



The null model (N) does not rule out that there may be hidden variables. So it doesn't rule out that there may be *supernatural* hidden variables.

What, then, are we to make of the considerable evidence that biologists have amassed against guided mutations? They have checked numerous organisms in numerous environments and have monitored the frequencies of numerous different mutations. In every case, a model like (G) turns out to be inferior to a model that denies that mutations are guided. It is unobjectionable to generalize from these consistent findings. So let us suppose that the experiment and the results I described are entirely typical. This means that when you take up a new organism and consider other possible mutations, you should expect that a model like (G) will not be your best model of how mutation probabilities are related to each other. Your model for this new organism and this new set of mutations will be like the old one I described for our blue organisms. The new model won't say of itself that it is causally complete any more than our model for the blue organisms did. These models, old and new, describe the effect of manipulating an organism's natural environment and how those manipulations affect (or fail to affect) mutation probabilities. None of these models rules out hidden variables. So none rules out *supernatural* hidden variables. Just as a model can be true without being causally complete, so too can a model be both true and inductively generalizable without being causally complete.

Here again, I must repeat the warning I issued at the outset: I am not saying that God intervenes in the mutation process. I am saying that scientific findings should not be misinterpreted.

### Two ways to think about conditional probabilities

Suppose you toss a coin repeatedly and use the frequency of heads to estimate the value of the conditional probability  $\Pr(\text{the coin lands heads} \mid \text{the coin is tossed})$ . Perhaps you'll use the procedure of maximum likelihood estimation, assigning to the conditional probability the value that makes the frequency data in your sample most probable; this will mean, if you got 51 heads in 100 tosses, that you'll assign to the conditional probability a value of 0.51.

Now consider a second experiment. You have 100 coins and toss each of them once. You get 51 heads. You want to estimate the probability that a coin sampled at random from these 100 will land heads. You use maximum likelihood estimation and conclude that  $\Pr(\text{the coin lands heads} \mid \text{the coin is tossed}) = 0.51$ .

How should these conditional probabilities be interpreted? One natural interpretation of the first experiment is that each toss of that single coin has a probability of landing heads of 0.51. This is the familiar idea that the coin-tossing process is *i.i.d.* (independent and identically distributed). A natural interpretation of the second experiment is that the coins may differ in their probabilities of landing heads, so what you are in fact estimating in the second experiment is the *average probability* of a coin's landing heads in that population of 100 coins. This interpretation admits the possibility that the coins may in fact have different probabilities of landing heads, but it is compatible with the *i.i.d.* assumption.

How should you interpret the probabilities that figure in the models (G) and (N) concerning our blue organisms? I think they should all be viewed as representing *average* probabilities. Suppose two blue organisms mutate to green among the 10,000 blue organisms that were placed in a green environment in our experiment. Your maximum likelihood estimate is that  $\Pr(\text{green mutation} \mid \text{green environment}) = 0.0002$ . This doesn't mean that the two mutations had exactly the same probability of occurring. The two organisms may have differed in various relevant respects. You should interpret 0.0002 as an *average* probability. In doing so, you are admitting that there may be hidden variables.

### **What if mutations sometimes were guided (in the biological sense)?**

The considerable biological data amassed so far indicate that models like (G) are inferior to models like (N). But suppose that an extraordinary species of organism were discovered that upsets the apple cart. Carefully constructed experiments reveal that these organisms adjust their mutation probabilities along beneficial lines, as (G) describes. That would be an amazing discovery. Would it show that God guides mutations?

Of course not. Although "guided mutation" has been historically connected with the idea that God guides the evolutionary process, this is not because the two ideas are logically connected. The association is a historical accident. The existence of guided mutations in the biologist's sense of that term has no more of a connection with divine intervention than does the idea that some organisms can synthesize vitamin D from sunlight and that others can regenerate lost limbs. In these last two cases, developmental biologists seek to characterize the physical mechanisms in individual organisms that subserve these functions, and evolutionary biologists look for naturalistic explanations for why these traits evolved in ancestral

populations. The same naturalistic approach would be set in motion by the hypothetical discovery of “guided” mutations.

If the existence of guided mutations doesn’t show that God exists, then the nonexistence of guided mutations doesn’t show that God does not exist. Atheists and theists should agree that the biological question is separate from the theological question.

### A Duhemian analogy

In his book *The Aim and Structure of Physical Theory*, Pierre Duhem (1954) defended the following two claims about theories in physics:

- Physical theories do not, by themselves, make predictions about what we will observe.
- Physical theories, when supplemented by auxiliary assumptions, do make predictions about what we will observe.

The auxiliary assumptions that Duhem discussed include propositions about the initial and boundary conditions of the system under study and propositions about one’s measurement devices. This pair of ideas now goes by the name “Duhem’s thesis”; it has had a deep influence on philosophy of science.<sup>8</sup> I suggest that it provides a good model for how biological findings about mutation are related to theistic claims about divine intervention in the mutation process.

Evolutionary theory does not entail that God never intervened in the mutation process, but the theory, when supplemented by auxiliary assumptions, does have implications about divine intervention. Here are some possible auxiliary assumptions. The list is not exhaustive:

(Deism) God created the universe, the laws that govern the universe, and the initial conditions of the universe, but he never intervenes in natural processes after that first moment.

(The Theology of the Unhidden God) If God ever intervened in the mutation process, then we’d have scientific evidence that mutation probabilities change in beneficial directions when the environment changes.<sup>9</sup>

<sup>8</sup> This logical thesis deserves to have the influence it has had, though it does need to be fine-tuned and generalized; see Sober (2008, 144) for discussion. An epistemological thesis is sometimes associated with the logical thesis – that evidence never confirms or disconfirms theories taken by themselves, but only has this impact on conjunctions in which the theories figure. This holistic thesis is mistaken and does not follow from the logical thesis (Sober 2004).

<sup>9</sup> I take my name for this position from Lucien Goldmann (1955), turning his book title on its head. According to Goldmann, the Jansenism of Pascal and Racine asserts that God cannot be known via

(Evidentialism) If you lack scientific evidence as to whether  $X$  is true, then you should suspend judgment about whether  $X$  is true.

(Fideism) You should believe that God guides the mutation process whether or not you have scientific evidence that he does so.

The first two of these, when added to our best scientific picture of what causes mutation, entail that God never intervened in the mutation process. The third, when conjoined with what biology tells us about mutations (properly understood), entails that we should be agnostic about divine mutational intervention. And the fourth, of course, entails that we should believe that God intervenes in the mutation process. It is beyond the scope of this chapter to consider which of these auxiliary assumptions we should adopt (or whether there are other candidates that are better). My present point is that none of these auxiliaries is part of evolutionary theory; they are – all of them – philosophical theses. My Duhemian claim is that evolutionary theory has consequences about divine intervention in the mutation process only when evolutionary theory is supplemented by further assumptions.

I mentioned at the start that some versions of interventionist theism, like Young Earth Creationism, are logically inconsistent with evolutionary theory. The idea that God sometimes intervenes in the mutation process is different.

### Concluding comments

I began this chapter by saying that Michael Ruse and I have our accommodationism in common and that neither of us is a theist. There is a third point of contact. Michael's introductory textbook in philosophy of biology (Ruse 1973) took a sympathetic view of logical positivism. I also have a lot of time for that philosophy (Sober 1999, 2000, 2008, 2010, 2011). Not that I subscribe to the testability theory of meaning. But I do think

the categories that we finite creatures possess, except that we know that God, without cause, confers the gift of grace on some human beings but not on others. Is the theology of the unhidden God the one that the biologist and atheist Jerry Coyne is endorsing when he says: "I think that *the absence of evidence for God, when there should be such evidence*, is indeed empirical evidence against God?" (Italics and underlining his: <http://whyevolutionistrue.wordpress.com/2012/05/07/can-god-create-mutations-elliott-sober-says-we-cant-rule-that-out/>.) Perhaps Coyne should write a blog about why Pascal was mistaken to think of God as he did. On the other hand, perhaps Coyne's remark is just a tautology. In any event, the slogan "absence of evidence is evidence of absence" has obvious counterexamples. I do not have evidence that there is a storm on the surface of Jupiter now, but that isn't evidence that there is no such storm (Walton 1996). See Sober (2009a) for discussion of this epistemological principle in a probability framework.

that it is important to distinguish propositions that are testable from those that are not. Young Earth Creationism is testable and false. The proposition that God undetectably intervenes in the evolutionary process is not testable. Positivism says that this proposition is meaningless, but I do not. Neither do I say that it is false; I have no way of telling what its truth value is, and I don't find Ockham's razor an appealing argument for atheism in this instance (Sober 2009b). Scientific theories don't tell you what to think about untestable propositions. That's a philosophical question that needs to be addressed as such.

It is important to distinguish the evidential grounds one has for accepting a proposition from the practical reasons one has for asserting it in public. This chapter has considered accommodationism under the first heading, but I want to close by saying something about the second. I bother to publish in defense of accommodationism in part because I want to take the heat off of evolutionary theory. The more evolutionary theory gets called an atheistic theory, the greater the risk that it will lose its place in public school biology courses in the United States; if the theory is thought of in this way, one should not be surprised if a judge decides that teaching evolutionary theory violates the constitutional principle of neutrality with respect to religion.<sup>10</sup> Indeed, the risk is more profound, since what happens in public education often has ramifications for what happens in the wider culture. Creationists have long held that evolutionary theory is atheistic; defenders of the theory are not doing the theory a favor when they agree. Atheists who think that evolutionary theory provides the beginning of an argument for disbelieving in God should make it clear that their arguments depend on additional premises that are not vouchsafed by scientific theory or data. Philosophy is not a dirty word.

#### REFERENCES

- Crow, J. and M. Kimura (1970) *An Introduction to Population Genetics Theory*. Minneapolis: Burgess Publishing Co.
- Darwin, C. (1868) *Variation of Animals and Plants under Domestication*. New York: Appleton (2nd edn., 1876).
- Duhem, P. (1954) *The Aim and Structure of Physical Theory*. Princeton University Press (first published 1914, in French).

<sup>10</sup> Here I echo Ruse (2009) when he says "[I]f science generally and Darwinism specifically imply that God does not exist, then teaching science generally and Darwinism specifically runs smack up against the First Amendment."

- Earman, J. (1986) *A Primer on Determinism*. Dordrecht: Reidel.
- Goldmann, L. (1955) *Le Dieu caché: étude sur la vision tragique dans les "Pensées" de Pascal et dans le théâtre de Racine*. Paris: Gallimard. Translated by P. Thody as *The Hidden God: A Study of Tragic Vision in the "Pensées" of Pascal and the Tragedies of Racine*. London: Routledge, 1964.
- Gray, A. (1888) "Natural Selection not Inconsistent with Natural Theology." In *Darwiniana*. Cambridge, MA: Harvard University Press, 1963, pp. 72–145.
- Renyi, A. (1970) *Foundations of Probability*. San Francisco: Holden-Day.
- Ruse, M. (1973) *The Philosophy of Biology*. London: Hutchinson.
- (2004) *Can a Darwinian be a Christian? The Relationship between Science and Religion*. Cambridge University Press.
- (2009) "Why I Think the New Atheists are a Bloody Disaster." *Biologos Forum*: <http://biologos.org/blog/why-i-think-the-new-atheists-are-a-bloody-disaster> (accessed September 30, 2013).
- Russell, R. (2008) *Cosmology from Alpha to Omega: The Creative Mutual Interaction of Theology and Science*. Minneapolis, MN: Fortress Press.
- Sober, E. (1999) "Testability." *Proceedings and Addresses of the American Philosophical Association* 73: 47–76.
- (2000) "Quine's Two Dogmas." *Proceedings of the Aristotelian Society* 74: 237–80.
- (2004) "Likelihood, Model Selection, and the Duhem–Quine Problem." *Journal of Philosophy* 101: 1–22.
- (2008) *Evidence and Evolution: The Logic behind the Science*. Cambridge University Press.
- (2009a) "Absence of Evidence and Evidence of Absence: Evidential Transitivity in Connection with Fossils, Fishing, Fine-Tuning, and Firing Squads." *Philosophical Studies* 143: 63–90.
- (2009b) "Parsimony Arguments in Science and Philosophy: A Test Case for Naturalism<sub>p</sub>." *Proceedings and Addresses of the American Philosophical Association* 83: 117–55.
- (2010) "Evolution without Naturalism." In J. Kvanvig (ed.), *Oxford Studies in Philosophy of Religion* 3. Oxford University Press, pp. 187–221.
- (2011) *Did Darwin Write the "Origin" Backwards? Philosophical Essays on Darwin's Theory*. Amherst, NY: Prometheus Books.
- Walton, D. (1996) *Arguments from Ignorance*. University Park, PA: Penn State Press.