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Are human beings part of the rest of nature?

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Abstract. Unified explanations seek to situate the traits of human beings in a causal framework that also explains the trait values found in nonhuman species. Disunified explanations claim that the traits of human beings are due to causal processes not at work in the rest of nature. This paper outlines a methodology for testing hypotheses of these two types. Implications are drawn concerning evolutionary psychology, adaptationism, and anti-adaptationism.

The issue we want to address is not whether human beings should be understood naturalistically or *super*naturalistically. Rather, our question concerns the kinds of naturalistic explanations that are needed to account for the features that human beings exhibit. If a factor C helps explain some feature E of nonhuman organisms, should we infer that C also helps explain E when E is present in human beings? The choice that interests us is between *unified* and *disunified* explanations. Do human beings fall into patterns exhibited by the rest of nature, or are we the result of fundamentally different causal processes?[1]

Although evolutionary theory is often seen as the vehicle for understanding human beings as part of the natural order, it would be wrong to assume that evolutionary explanations are automatically unified. An evolutionary explanation for why two species have a feature need not claim that they have that feature for the same reason. Fir trees are green and so are iguanas, and there is an evolutionary explanation for each of these outcomes; however, iguanas and fir trees are green for very different evolutionary reasons. In fact, within an evolutionary framework there are four possible patterns of explanation, not just two; these can be described by beginning with the three options depicted in Figure 1.

In case (1), the two species (S_1 and S_2) are similar because they inherited their shared feature from a common ancestor (A); the similarity is a *homology*. In both (2) and (3) the two descendant species obtained feature E by independent evolution; the similarity is an *analogy*.[2] Within this category of analogous similarities, one can distinguish functionally similar analogies from functionally dissimilar analogies (Sober 1993). Even though birds and bats evolved their wings independently, it still

[1] Thus the question we are considering is the mirror image of the problem of self-to-other inference that constitutes the traditional philosophical problem of other minds. See Sober (2000) for discussion.

[2] Although Figures 2 and 3 don't depict a common ancestor, we assume that one exists.

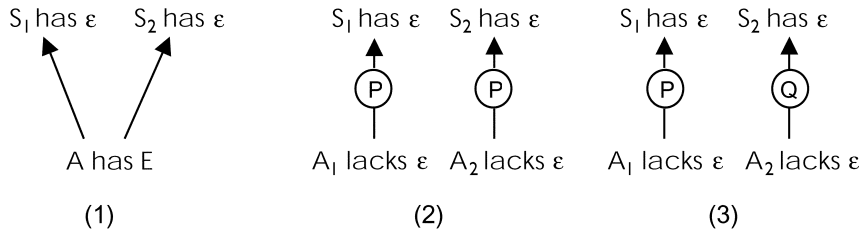

Figure 1.

may be true that the trait evolved for the same reason (P) in the two lineages – in both instances, wings evolved because they facilitated flight (case 2). The green coloration of fir trees and iguanas is different. Not only is the similarity not homologous; in addition, the reason the color evolved in the lineage leading to fir trees differs from the reason it evolved in the line leading to iguanas ($P\hat{|}$); this is case 3.[3]

In Figure 1, inheritance from a common ancestor (case 1) is represented as a possibility on a par with the two types of analogy depicted in cases 2 and 3, but in fact the category of homology needs to be subdivided. If two descendant species have trait E because their most recent common ancestor had E, it is a further question as to why the trait was maintained in the two lineages. It is possible that the trait was retained in the two lineages for the same reason (P), or for different reasons ($P\hat{|}$). Thus, case 1 in Figure 1 needs to be separated into the two scenarios depicted in Figure 2.

Inheritance from a common ancestor is often thought of as a unitary and nonselective explanation of a trait's presence; however, the fact of the matter is that a descendant can exhibit the trait possessed by its ancestor for selective as well as for nonselective reasons (Orzack and Sober 2001). Stabilizing selection can cause stasis. But what does it mean for a descendant to have a trait, not because of stabilizing selection, but simply because its ancestor had the trait? We take this to mean that the trait was retained because there wasn't sufficient time for the

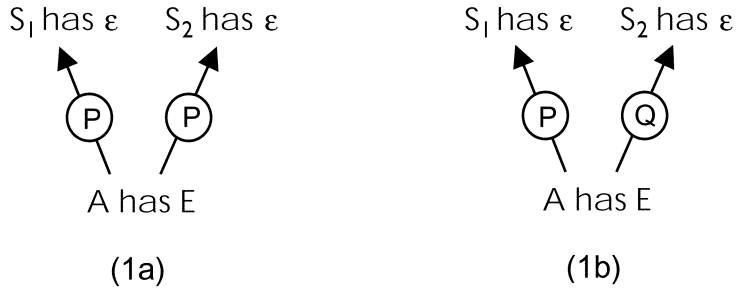

Figure 2.

[3] It is worth noting that pattern (3) subdivides into two possibilities; there may be a partial overlap between the reasons the trait evolved in the two lineages, or the reasons may be entirely disjoint.

Table 1. Why was trait E, which was found in the ancestor, retained in the descendant?

		Natural selection	
		selection for trait E	selection against trait E
Phylogenetic inertia	little time	both selection and inertia	inertia only
	lots of time	selection only	neither

descendant to evolve away from the ancestral condition. If so, it is appropriate to talk of *ancestral influence* or *phylogenetic inertia* (Harvey and Pagel 1991; Orzack and Sober 2001). Although selection and inertia are different possible causes of stasis, they are compatible; both can contribute to a trait's retention, as Table 1 indicates. Ancestral influence occurs when a lineage's initial condition affects its subsequent state; selection, on the other hand, is a process that occurs during the duration of the lineage. Both the lineage's initial condition and the processes that then set to work can affect the character states of descendants.

We so far have described how we understand the question of whether human beings are part of the rest of nature by considering how one should explain a *similarity* that unites human beings and one or more nonhuman species. Explanations (1a) and (2) are unified; explanations (1b) and (3) are disunified. However, the choice between unified and disunified explanations also arises when one wants to explain why the species under consideration exhibit *different* trait values. Rather than develop this point abstractly, we will explain it in terms of an example.

In modern industrial societies, women on average live longer than men. One might suspect that this is a recent phenomenon, a result of improved medical care that reduces the risk of dying in childbirth. In fact, the data available suggest otherwise. In 18th century Sweden, for example, women lived longer than men, and this inequality continued right up to the present, despite a steady improvement in the longevities of both sexes. The same is true of the Ache, a hunter-gatherer group now living in Paraguay. Indeed, in 20th century societies around the world, one almost always observes that women live longer than men. Is this fact about human beings to be explained in terms of some constellation of causes that is unique to our species? Or is the pattern of longevity in human beings due to factors that apply to a more inclusive set of organisms?

Allman et al. (1998) cite the facts just described and seek to explain them in terms of a general hypothesis about anthropoid primates – when one sex provides more parental care than the other, selection favors reduced mortality in the sex that makes the larger contribution. They hypothesize that selection will generate a quantitative relationship – the greater the imbalance in parental care, the more skewed the longevity should be in favor of the sex that provides more parental care. Although they don't spell out their reasoning in much detail, their idea is presumably that the sex that provides more parental care would incur a greater fitness cost by accepting an increased risk of mortality; this leads the sex that provides more parental care to be more risk-averse. In support of their hypothesis, the authors present the data in Table 2.

Allman et al. (1998) wanted to test the hypothesis that disparity in parental care causes disparity in longevity – the latter is an adaptive response to the former. They

Table 2. Survival ratios and male care of offspring in anthropoid primates

Primate	Female/Male survival ratio	Male care of offspring
Chimpanzees	1.418	Rare or negligible
Spider monkey	1.272	Rare or negligible
Orangutan	1.203	None
Gibbon	1.199	Pair-living, but little direct role
Gorilla	1.125	Protects, plays with offspring
Human (Sweden 1780–1991)	1.052-1.082	Supports economically, some care
Goeldi's monkey	0.974	Both parents carry offspring
Siamang	0.915	Carries offspring in second year
Owl monkey	0.869	Carries infant from birth
Titi monkey	0.828	Carries infant from birth

(Reproduced with permission from Allman, J., Rosin, A., Kumar, R. and Hasenstaub, A., ''Parenting and Survival in Anthropoid Primates–Caretakers Live Longer''. *Proc. Natl. Acad. Sci. USA* 95: 6866–6869, Copyright (1998) National Academy of Sciences, U.S.A.)

cite as confirmation the fact that the two variables are associated in the data, and we do not disagree. However, it is important to understand this evidential claim in the right way. Allman et al. (1998) tested their causal hypothesis against a *null hypothesis*, one that says that the two variables are causally unrelated. The former hypothesis predicts an association in the data, while the latter predicts no association.[4] This methodology is fine as far as it goes, but it has its limitations. The data do not favor the causal hypothesis that Allman et al. (1998) formulate over its converse – that differences in longevity caused differences in parental care.[5] Nor do the data rule out the hypothesis that the two variables are effects of a common cause.

The first thing to notice about this pattern of argument is that the exact survival ratio exhibited by human beings differs from that found in other species. Allman et al. (1998) are arguing that human beings are ''part of the rest of nature'', but this does not mean that the human characteristic they wish to explain must be identical with the characteristics found in other species. Rather, the study defends a unified account of the human trait value by showing how the human value falls within a larger pattern of variation. The point is that *we are not outliers*. We may be unique in our trait value (just as other species are in theirs), but the suggestion is that we are not unique with respect to the causal processes generating that trait value.

Notice also that Allman et al. (1998) do not attempt to explain the pattern of variation that exists within our species, nor, for that matter, the variation found in other species. The trait value for a species is the species *average*. It is perfectly consistent with their analysis that the ratio of female-to-male longevities should fail

[4] The argument of Allman et al. (1998) exhibits a pattern of argument that is entirely standard in evolutionary biology. Although hypotheses about natural selection purport to describe processes at work within lineages, the data sets used to test those hypotheses usually describe the character states of tip species. Why should the latter be able to confirm or disconfirm the former? For discussion, see Sober and Orzack (2002).

[5] It sometimes is possible to discriminate between the hypothesis that E is an adaptive response to C and the hypothesis that C is an adaptive response to E by seeing which trait evolved first. This procedure requires one to reconstruct the character states of ancestors in a phylogenetic tree. Cladistic parsimony is the method usually used to do this; see Sober (2002) for discussion.

to be positively related to the ratio of female-to-male parental care as one looks across populations within our species. Would this show that human beings are not ''part of nature''? Here we must recognize the limited usefulness of this way of posing the question. Not only must we relativize our question about the place of human beings in nature to a specific trait (in this instance, the fact that women live longer than men). In addition, we need to specify the pattern of variation that we wish to consider. It is entirely possible that the human *average* fits in with data about the average values found in other species, even if human *variation around that average* is generated by causal processes that differ fundamentally from the factors that generate variation within or among other species.

Let's consider what it would mean if cross-cultural variation in the longevity ratio *were* positively associated with the ratio of contributions to parental care. This could be true even if the human average were an outlier in the context of cross-species data. Furthermore, the existence of human plasticity does not automatically place us ''outside the rest of nature''. Even if, contrary to fact, ours were the only species that exhibits within-species variation in these features, it still could be true that we are part of the larger picture. What would be unique about us is our *plasticity*; but the factors that explain within-species variation in our case could still coincide with the factors that explain the pattern of between-species variation.[6]

The logical independence of these two levels of analysis – within-species and between-species – is depicted in Figure 3. We can ask how the human average relates to average values found in other species. And we can ask how variation within our species relates to those average values. The latter may seem like an ''apples and oranges'' question, since we are comparing within-species variation with between-species variation. Nonetheless, the question makes sense and has its point. In order to keep things simple, we have omitted a third question from our table, one that is logically independent of the first two – how does within-species variation in our species relate to within-species variation in other species? The two questions that are described in Figure 3 generate four possibilities, which differ with respect to whether and how the human condition is unified with the situation found in the rest of nature. We've included in each cell of the table a hypothetical data set that would support the relevant interpretation.[7]

Several features of this framework merit comment. First, it is important to see that

[6] Within-species variation could be due to genetic variation, environmental variation, or both; all three possibilities are consistent with the adaptive hypothesis (Sober 1993). There need be no commitment to ''genetic determinism''.

[7] The epistemology of choosing between unified and disunified explanations is interesting. Even if human beings are not outliers, why couldn't it be true that human trait values are the product of fundamentally unique causal processes? Conventional frequentist statistics treats the unified explanation as a null hypothesis, one that asserts that there is no difference between the human and nonhuman causal situations; testing takes the form of asking whether the data permit one to reject this null hypothesis. According to this approach, one should embrace the disunified explanation only if the data force one to do so. Frequentist statistics thus assigns a privileged status to unified explanations. For discussion of Bayesian approaches to this problem, see Forster and Sober (1994), which also locates the problem within the framework of Akaike's criterion for model selection. Some such statistical framework is needed to define what it means for a species to be an ''outlier'' – how much distance between the human trait value and the regression line for other species must there be for this to be true?

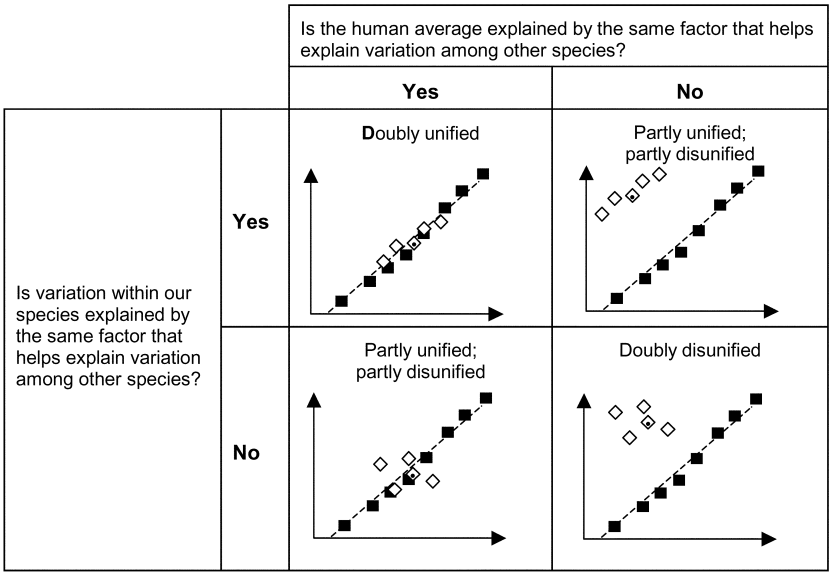

Figure 3. Explanations of patterns of variation. Each white diamond represents the average value for a human population. The white diamond with a dot in it represents the human average. Each black square represents the average value for a nonhuman species. The line is the best-fitting regression line for the non-human species.

it is specific models about the relationship of specific dependent and independent variables that get tested; the bare claim that there exists a unified (or a disunified) explanation of some effect (e.g., why women live longer than men) does not make testable predictions. A related point is that it is possible for human beings to fall into the pattern represented in one cell of Figure 3 when X and Y are the variables used, whereas the pattern changes to that depicted in another cell when a new independent variable Z is used instead of X. Finally, we note that specific models make probabilistic (not deductive) predictions about data; this means that it is perfectly possible that a given model is true and yet the data one observes fails to conform to the patterns associated in Figure 3 with that model.

What would it mean if human beings were outliers twice over (as depicted in the lower right cell of Figure 3) – suppose our average trait value does not conform to the pattern displayed by other species, and suppose that variation within our species exhibits a different pattern from that found among other species? This may be due to the fact that we human beings are influenced by nonbiological, cultural, forces that are unique to us. Or it may be that biological causes distinct from those acting on other species are at work. Our deviating from patterns found in the rest of nature does not decide this question.

We began by discussing the question of whether human beings are part of the rest of nature by examining possible explanations of the *similarities* that may unite human beings and this or that nonhuman species. We then explained how the same question can arise in explaining why human beings have trait values that *differ* from

those found in other species. The key here is not whether human beings have the same traits or different ones, but how the distribution of characters among species is explained. Similarities can arise from different causes and differences can arise from the same causes. The issue concerns causation, not whether we are similar to or different from other organisms. This question is not settled by the fact that human beings are genealogically related to other species.

We want to emphasize the role played by the concept of variation in our analysis of how the place of human beings in nature should be assessed. To decide whether human beings are part of the causal pattern found in the rest of nature, or deviate from it, one must be able to identify what that wider causal pattern is. For this to be possible, there must be variation in one's data. If a quantitative variable X causes a quantitative variable Y, then changes in X-values should be associated with changes in Y-values (once one controls for other contributing causes).[8] If smoking causes cancer, then people who smoke more should get cancer more frequently than people who smoke less, where comparisons are carried out among individuals who are otherwise the same with respect to other factors that influence cancer. The causal proposition would not be tested by a data set in which everyone smoked to the same degree. By the same token, the causal hypothesis advanced by Allman et al. (1998) – that disparities in parental care cause disparities in longevity – would not be tested by a data set in which the ratio of female-to-male investment is the same across the species considered.

The simple fact that testing causal hypotheses requires a comparison of different types of cases – of situations in which dosages of the putative causal factor are different – helps explain part of what Gould and Lewontin (1979) were getting at when they criticized the invention of ''just-so stories'' in evolutionary biology. When one's observation is the simple fact that species S has trait T, the data are too impoverished to provide a proper test of a causal explanation. It isn't that adaptive hypotheses are untestable, but rather that it takes a certain kind of data set to put them to the test. If the dichotomous trait T is universal within species S, then one needs a data set in which some species have trait T while others do not. In one sense, the explanation of impoverished data is easy – it is easy enough to invent a story that fits the data – but, in another sense, the explanation of impoverished data is impossible – the data do not permit adaptive hypotheses to be tested properly. This, we suggest, is what it means for adaptive story-telling to be ''too easy''.[9]

Adaptationism, as Gould and Lewontin (1979) understand that *ism*, contrasts with

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[8] Allman et al. (1998) do not investigate whether the similarities they observe were due partly to phylogenetic inertia rather than adaptation. Testing an adaptive hypothesis requires that one control for this possibility. See Felsenstein (1985), Orzack and Sober (2001) for discussion.

[9] Gould and Lewontin (1979) also say that if one adaptive hypothesis fails, another can be invented in its place, and that this possibility constitutes a flaw in adaptationism. The first thing to notice about this claim is that it envisions adaptive hypotheses' failing; this presupposes that a data set is being consulted that is not impoverished – it succeeds in putting the hypothesis to the test. We also note that the possibility that they describe in connection with adaptationism also is possible for the evolutionary pluralism they advocate – if one pluralistic model fails, another can be invented in its place. Since this is a feature of all research programs; it does not constitute a reason for rejecting any particular research program, though it does raise the question of when a research program ceases to be worth pursuing (Sober 1993).

evolutionary pluralism. They say they agree with Darwin that natural selection has been the *most important cause* of evolutionary change. What they disagree with is the monistic idea that natural selection has been the *only important cause*. We interpret this adaptationist hypothesis to predict that organisms should have locally optimal traits; they should exhibit traits that are fitter than any of the available alternatives (Orzack and Sober 1994; Sober 1993). Given this contrast between adaptationism and pluralism, it is important to recognize that the hypothesis that Allman et al. (1998) were testing is *not* an instance of adaptationism. They were not claiming that the disparity in mortality rates between the sexes is optimal; indeed, their paper does not even specify what the optimal disparity would be. The argument of Allman et al. (1998) was merely to show that male-female differences in longevity were influenced by natural selection. Since pluralists just as much as adaptationists are committed to the importance of natural selection, both need to avoid telling just-so stories about that process; in this sense, adaptive hypotheses are not the exclusive property (and problem) of adaptationists. Data sets that exhibit variation are a useful prophylactic device; they make it harder to invent adaptive scenarios.

In addition to throwing light on the general problem of testing adaptive hypotheses, the methodology we are suggesting also elucidates a special problem that arises in connection with human evolution. To test causal hypotheses about the place of human beings in nature, the human traits under study must be commensurable with the traits found in other species. As we saw in connection with the data used by Allman et al. (1998), it isn't essential that human beings and other organisms have exactly the same trait values. Rather, the point is that their study used quantitative variables – the female-to-male survival ratio and the female-to-male ratio of parental care – that subsume human beings and other species alike. The same point would apply if the traits considered were dichotomous. If human beings and other species can be said to have or lack trait C, and the same is true of trait E, then a data set can be obtained that allows one to evaluate whether C and E are associated. But suppose human beings are the *only* species that exhibits trait E. If so, there is an easy recipe for finding causal hypotheses that fit the data – merely find a trait C that also is unique to human beings. The result is that C and E will be perfectly associated in one's data. The trouble is that the data will not help one pry apart different causal hypotheses that focus on different uniquely human features. The hypothesis "C_1 causes E" will fit the data, but so will "C_2 causes E", "C_3 causes E", and so on. Here we find a second context in which the invention of adaptive hypotheses is too easy. If there is no variation in one's data, the data are useless. But if the variation is such that all species are the same, save one, the data are next to useless.

We think there is an important lesson here for the research program known as evolutionary psychology. Tooby and Cosmides (1990) argue that evolutionary theory predicts that the complex adaptive features found in our species (or, indeed, in any species) will be species-typical universals. There is a great deal of room to doubt whether evolutionary theory provides a principled reason to expect there to be no within-species adaptive variation (Wilson 1994). Furthermore, the argument

made by Tooby and Cosmides (1990) has not stopped other evolutionary psychologists from trying to find adaptive explanations of behavioral differences between the sexes (Buss 1994; Daly and Wilson 1988) and this, of course, is an instance of within-species variation. However, the point we want to make here is that if the trait of interest *is* universal in our species, then the only way to explain its presence is to adopt a comparative perspective. Otherwise, one is working with a single data point – an impoverished data set if ever there was one. The problem is that the features that often interest evolutionary psychologists are uniquely human – the human language faculty, the cognitive capacity to analyze what kinds of observations would falsify a conditional statement, etc. This leaves it open that evolutionary psychologists can attempt to embed their description of human beings within a wider view of the traits found in other species. For example, perhaps there are features of human language that can be related to features of communication systems used in other species.[10] The other way forward for evolutionary psychology is to focus on traits with respect to which human beings vary. What is a dead end, in our view, is the attempt to explain human universals that are unique to our species without relating those features to trait values found in other species.

Although our protocol for testing hypotheses concerning the place of human beings in nature has focused on adaptive hypotheses, our proposed methodology is not limited to hypotheses that make claims about natural selection. It isn't just adaptive hypotheses that can be elaborated as ''just-so'' stories. Hypotheses that postulate nonadaptive processes also require data sets that are not impoverished. For example, consider the hypothesis that the human language faculty did not evolve because it facilitates communication, but was merely a byproduct of the evolution of a big brain, which evolved for other adaptive reasons.[11] We submit that this proposition is untestable if one looks just at human beings. And if one considers a range of species within which the language faculty and a big brain are both unique to our species, the byproduct hypothesis is testable, but now a new difficulty arises, one that we described previously. The invention of *non*-adaptive hypotheses also can be ''too easy''.

We have emphasized that the data sets used to test adaptive hypotheses must contain variation. Is there an alternative methodology, one in which a single observation of a trait that is universal within a species suffices? We are skeptical. Granted, if an adaptive hypothesis specifies the optimal trait value, and asserts that organisms have attained their optima, it is possible to make a single observation and determine whether the model's prediction is correct. However, as we have emphasized, adaptive hypotheses are very often not of this form. What these weaker

[10] This type of analysis is developed by Pinker and Bloom (1990), Pinker (1994).

[11] Thus Gould (1991, 62) : ''the traits that Chomsky (1986) attributes to language – universality of the generative grammar, lack of ontogeny, . . . , highly peculiar and decidedly nonoptimal structure, formal analogy to other attributes, including our unique numerical faculty with its concept of discrete infinity – fit far more easily with an exaptive, rather than an adaptive, explanation. The brain, in becoming large for whatever adaptive reasons, acquired a plethora of cooptable features. Why shouldn't the capacity for language be among them?''

hypotheses assert is that species have evolved in the direction of more optimal trait values. These hypotheses predict that lineages have *changed* in certain ways. Hypotheses about the direction of change cannot be tested by a single snapshot. Even when a species is ''close'' to the trait value that an optimality model says is optimal, the question remains of whether the lineage leading to that species has evolved towards that trait value or away from it (Sober and Orzack 2002).

Our demand for comparative data may seem gratuitous when it seems obvious that a species' trait value is an adaptive response to some problem that the species faces. It may seem obvious that the polar bear's thick fur evolved as an adaptive response to cold weather. There seems to be an obvious ''fit'' between the warm coat and the icy temperature. Why do we have to look at bears that live in warmer climates to see if they have thinner coats? The polar bear's fur and the temperature of the bear's environment resemble a key and the lock it opens. Shouldn't it be self-evident that the one was made as a solution to the problem posed by the other?[12] There is reason to be cautious here, however, since every biologist can recount examples in which intuitively ''obvious'' adaptive scenarios turned out to be disconfirmed by data. Without a correlation between fur thickness and ambient temperature, the hypothesis that polar bears have thick fur as an adaptive response to ambient temperature remains a mere plausible conjecture.[13]

In closing we want to describe a kind of problem that the methodology we have proposed does not solve. If an adaptive hypothesis predicts a correlation, it can be tested against a null hypothesis that predicts no correlation. And if a nonadaptive hypothesis predicts a correlation, it too can be tested against a null hypothesis that predicts no correlation. But how can adaptive and non-adaptive hypotheses be tested against each other? If they predict *different* correlations, the procedure is straightforward. But what if they predict *the same* correlation?[14] This is an interesting question, but it differs from the one we set out to address. Notice that the adaptive and the non-adaptive hypotheses we now are considering are both *unified* – both seek to explain human trait values by situating them in a causal framework that subsumes other species as well. Our concern in this paper has been to discuss how unified and disunified hypotheses should be compared, not to make assessments within the category of unified explanations.

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[12] Lewontin (1982) skepticism of the idea that adaptations are ''solutions'' to pre-existing ''problems'' posed by the environment is relevant here.

[13] In this connection, see Sober (1997) discussion of how one might test Godfrey-Smith (1996) hypothesis that mental representation is an adaptation for coping with environmental complexity.

[14] The study of Allman et al. (1998) provides an example. Their hypothesis is that disparities in parental care cause disparities in longevity. How would one test this against the claim that the two traits are pleiotropic consequences of the same set of genes?

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