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The matching problem for evolutionary psychiatry

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ABSTRACT

Evolutionary psychiatry suggests that mental disorders can be explained in evolutionary terms (a) as failures of psychological mechanisms to produce the adaptive effects for which they were naturally selected, (b) as mismatches between naturally selected psychological mechanisms and contemporary environmental pressures, or (c) as naturally selected psychological mechanisms whose effects continue to be adaptive. In this paper, I present a philosophical critique of evolutionary psychiatry that draws on Subrena Smith's matching problem for evolutionary psychology. For evolutionary psychiatry hypotheses to be empirically supported, proponents of evolutionary psychiatry must demonstrate (1) that the contemporary psychological mechanisms involved in mental disorders resemble the psychological mechanisms of our evolutionary ancestors, (2) that the contemporary psychological mechanisms are phylogenetically descended from the ancestral psychological mechanisms, and (3) that the ancestral psychological mechanisms were naturally selected because their effects had adaptive benefits. However, for many mental disorders, evolutionary psychiatry lacks the methodological resources to demonstrate these conditions. Therefore, many evolutionary psychiatry hypotheses are empirically untestable and remain indefinitely underdetermined by data.

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Introduction

Evolutionary psychiatry suggests that mental disorders can be explained in evolutionary terms. The scope of evolutionary psychiatry is characterized by Randolph Nesse as follows:

> Evolutionary psychiatry is the subfield of evolutionary medicine that addresses mental disorders. The term invites misunderstandings, because it sounds like a new treatment method, perhaps one that is alternative or somehow radical. But evolutionary psychiatry is simply the field that uses the principles of evolutionary biology to better understand, prevent and treat mental disorders. It brings in a missing basic science, that joins

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> genetics, physiology, learning theory, cognitive science, neuroscience and psychodynamics, to better understand and treat mental disorders. (Nesse, 2023, p. 178)

This *prima facie* sounds like a compelling proposal. After all, we are evolved organisms whose bodies and brains have been influenced by natural selection. Accordingly, there has been increasing enthusiasm among psychiatrists and philosophers for evolutionary explanations of mental disorders (Abed & St John-Smith, 2016; Garson, 2022; Murphy, 2005; Nesse, 2023; Stevens & Price, 1996).

My aim in this paper is to quell some of this enthusiasm. While I do not dispute the broad claim that evolutionary processes have influenced our cognitive and affective capacities in ways that may make us susceptible to certain forms of psychological and behavioral distress, I argue that specific evolutionary psychiatry hypotheses about the origins of certain mental disorders rest on foundations that are methodologically unsound. The philosophical critique I provide draws on Subrena Smith's (2019) matching problem, which was initially intended as a methodological challenge to evolutionary psychology. For evolutionary psychiatry hypotheses to be empirically supported, proponents of evolutionary psychiatry must demonstrate (1) that the contemporary psychological mechanisms involved in mental disorders resemble the psychological mechanisms of our evolutionary ancestors, (2) that the contemporary psychological mechanisms are phylogenetically descended from the ancestral psychological mechanisms, and (3) that the ancestral psychological mechanisms were naturally selected because their effects had adaptive benefits. However, for many mental disorders, evolutionary psychiatry lacks the methodological resources to demonstrate these conditions. Therefore, many evolutionary psychiatry hypotheses are empirically untestable and remain indefinitely underdetermined by data.

Evolutionary psychiatry hypotheses

A helpful classification of evolutionary psychiatry hypotheses about mental disorders is provided by Dominic Murphy (2005), who distinguishes three explanatory approaches:

(a) Breakdown – Mental disorders are failures of psychological mechanisms to produce the adaptive effects for which they were naturally selected;
(b) Mismatch – Mental disorders are naturally selected psychological mechanisms whose effects were adaptive for our ancestors

but are no longer adaptive for us in the contemporary environment;

(c) Persistence – Mental disorders are naturally selected psychological mechanisms whose effects were adaptive for our ancestors and continue to be adaptive for us in the contemporary environment.

All these explanatory approaches have an adaptationist component. An adaptation is defined by Elliott Sober as follows:

> Characteristic *c* is an adaptation for doing task *t* in a population if and only if members of the population now have *c* because, ancestrally, there was selection for having *c* and *c* conferred a fitness advantage because it performed task *t*. (Sober, 2000, p. 85)

Evolutionary psychiatry hypotheses are adaptationist insofar as they suggest that mental disorders involve psychological mechanisms that were naturally selected because of their adaptive benefits for our ancestors. Of course, breakdown, mismatch, and persistence hypotheses disagree about precisely how these psychological mechanisms are involved. Specifically, they differ over whether mental disorders comprise failures of these selected mechanisms, effects of these selected mechanisms that are no longer adaptive, or effects of these selected mechanisms that are still adaptive. Nonetheless, the three explanatory approaches suppose that the relevant psychological mechanisms were naturally selected because they were adaptive for our prehistoric ancestors.

An example of a breakdown hypothesis is Jerome Wakefield's harmful dysfunction analysis of mental disorder, which is characterized as follows:

> A condition is a disorder if and only if (a) the condition causes some harm or deprivation of benefit to the person as judged by the standards of the person's culture (the value criterion), and (b) the condition results in the inability of some internal mechanism to perform its natural function, wherein natural function is an effect that is part of the evolutionary explanation of the existence and structure of the mechanism (the explanatory criterion). (Wakefield, 1992, p. 384)

Here, Wakefield assumes an etiological account of function, according to which the function of a trait of an organism is an effect of the trait that was evolutionarily adaptive for the organism's ancestors and, hence, explains the inheritance of that trait across generations up to the present organism (Millikan, 1989; Neander, 1991). For example, Wakefield (2007), suggests that we possess mechanisms for regulating sadness and loss that were naturally selected because their regulatory effects were evolutionarily adaptive. He then suggests that "depressive disorders involve failures of sadness and loss-response regulating mechanisms" (Wakefield, 2007, p. 152). Similarly, Anthony Stevens and John Price suggest:

> These response patterns, master programmes, propensity states, response strategies, evolved psychological mechanisms, and prepared tendencies are held responsible for crucial, species-specific patterns of behaviour that evolved because they maximized the fitness of the organism to survive, in the environment in which it evolved … Psychopathology intervenes when these strategies malfunction as a result of environmental insults or deficiencies at critical stages of development. (Stevens & Price, 1996, p. 9)

Again, the claim here is that mental disorders involve failures of psychological mechanisms that were naturally selected because they were adaptive for our prehistoric ancestors.

By contrast, mismatch hypotheses do not suggest that psychiatric disorders involve breakdowns of naturally selected psychological mechanisms. Rather, they suggest that the mechanisms are still producing their effects, but that these effects are no longer adaptive in our contemporary environment. Various mismatch hypotheses of depressive disorders have been proposed. For example, Price and colleagues suggest that depressive symptoms were adaptive for our ancestors who resided in small hierarchical groups because these symptoms helped to avoid conflicts with dominant members of these groups, but that such symptoms are no longer adaptive for us in our contemporary society (Price et al., 1994). Likewise, Nesse claims that depressive symptoms may have been adaptive for our ancestors who engaged in projects such as foraging because they inhibited the urges to move too quickly onto different and potentially risky projects when temporary setbacks were encountered:

> The start-up costs for a new enterprise are huge, there is often no certainty that another enterprise can be found at all, and the attractiveness of alternatives may be illusory … In this situation, pessimism, lack of energy, low self-esteem, lack of initiative, and fearfulness can prevent calamity even while they perpetuate misery … When depression is instead seen as a state shaped to cope with unpropitious situations, it is clear how it could be useful, both to decrease investment in the current unsatisfying life enterprise and also to prevent the premature pursuit of alternatives. (Nesse, 2000, p. 17)

He then suggests that such symptoms may no longer be adaptive for us in our contemporary culture where "enterprises tend to be huge, vulnerable, and irreplaceable" (Nesse, 2000, p. 18). Earlier scholars have also suggested that depressive symptoms may have been adaptive strategies that regulated how our ancestors allocated emotional and material resources to their plans and goals (Klerman, 1974; Klinger, 1975; Wender & Klein, 1982).

Persistence hypotheses also suggest that psychiatric disorders are the effects of psychological mechanisms that were adaptive for our ancestors. However, unlike mismatch hypotheses, persistence hypotheses claim that these effects are still adaptive for us in our contemporary environment. Some scholars suggest that depressive cognitions were adaptive for our ancestors because they were helpful for solving complex social problems that involve competing

goals (Andrews & Thompson, 2009; Hollon et al., 2021). Moreover, they suggest that these depressive cognitions may still sometimes be adaptive for us in the present day because they help us focus on the sources of our problems without being distracted by hedonic pursuits. For example, Steven Hollon and colleagues write:

> The analytical rumination hypothesis suggests that depression is an adaptation that evolved to serve a purpose in our ancestral past and may still be doing so today. It further suggests that depression evolved to keep people focused on the source of their distress until they could come up with a solution to resolve the relevant problem. (Hollon et al., 2021, p. 9)

Similarly, Justin Garson (2022) proposes that depressive symptoms and psychotic states are adaptive responses that are associated with epistemically beneficial insights into aspects of our realities that are usually obscured in our ordinary mental states.

These three explanatory approaches yield competing hypotheses of specific mental disorders. With regards to depressive disorders, the breakdown hypothesis is contradicted by the mismatch and persistence hypotheses, insofar as the latter two claim that depressive disorders are the ongoing effects of naturally selected psychological mechanisms whereas the former claims that depressive disorders are failures of the naturally selected psychological mechanisms to produce their effects. Mismatch and persistence hypotheses contradict each other with regards to whether depressive disorders are still adaptive in the contemporary environment. It is often hoped that further empirical evidence will help us to adjudicate between these competing hypotheses and justify evolutionary psychiatry approaches to mental disorders. For example, Murphy writes:

> Evolutionary hypotheses may at least have heuristic value in the development of testable predictions about behavior and testable assumptions about the functions of the mind/brain. And if we can link the functions of the mind/brain to facts about behavior, development and society, we may be able to develop more high-powered predictions . . . (Murphy, 2005, p. 763)

However, in what is to follow, I argue that proponents of evolutionary psychiatry are not epistemically situated to acquire the relevant sorts of empirical evidence to justify their hypotheses about mental disorders.

Conditions for successful evolutionary adaptationist explanations

In a recent philosophical critique of evolutionary psychology, Smith (2019) notes that certain conditions must be met for an evolutionary adaptationist explanation of a contemporary trait to be successful:

(1) Concordance – It must be demonstrated "*which* ancestral structures are related to *which* contemporary ones" (Smith, 2019, p. 42). That is to say, a given contemporary trait must be shown to produce the same sort of effect as some ancestral trait.
(2) Strong vertical homology – Concordance by itself is insufficient for an adaptationist explanation, as traits may produce similar effects for a variety of reasons, such as shared environmental influences and developmental constraints. Hence, it must also be demonstrated "that the contemporary module has the same function as the ancestral one in virtue of its being descended from the ancestral module" (Smith, 2019, p. 42). The contemporary trait must be a genetically inherited feature that has a continuous phylogenetic lineage going back to the ancestral trait.
(3) Ultimate explanation – For the explanation to be adaptationist, it must be demonstrated "that the function of a contemporary module is one that an ancestral module was selected for performing" (Smith, 2019, p. 42). That is to say, it must be shown that the trait was naturally selected because the effect it produced was conducive to survival and reproductive fitness.

Of course, adaptationist explanations are not always appropriate. Some traits may not be adaptations at all, but instead may be evolutionarily neutral byproducts or ontogenetic effects of culture and the environment (Gould & Lewontin, 1979; Oyama, 2000). Nonetheless, where an adaptationist explanation is appropriate, the above conditions must be met for the explanation to be successful.

As noted earlier, evolutionary psychiatry hypotheses are adaptationist insofar as they suppose that mental disorders involve psychological mechanisms whose effects were adaptive for our ancestors. Although breakdown, mismatch, and persistence hypotheses disagree over precisely how these psychological mechanisms are involved, all of them suppose that the relevant mechanisms have been conserved through natural selection because they were conducive to survival and reproduction for our ancestors in the prehistoric environment (Murphy, 2005). Hence, the above three conditions for what makes a successful adaptationist explanation also apply to evolutionary psychiatry hypotheses.

For biological traits, the above conditions can often be met with evidential support from (i) the fossil record, (ii) the comparative method, and (iii) optimality modeling. The fossil record can provide evidence that the same sort of trait that is possessed by a contemporary organism was also possessed by an ancestral organism. This can help to demonstrate concordance (Kaplan, 2002). The comparative method involves examining the contemporary organism's closest extant relatives to establish whether the same sort

of trait in the contemporary organism is also possessed by these closely related species (Sterelny & Griffiths, 1999). In conjunction with the fossil record, the comparative method can help to demonstrate concordance and strong vertical homology by providing evidence that the trait has been conserved in multiple descendants of the ancestral organism. Optimality modeling involves the construction of a model to assess the effects that possessing the trait and lacking the trait have on an organism's survival and reproductive fitness in a given environment (Orzack & Sober, 1994). This can support an ultimate explanation of the trait by providing evidence that the trait was adaptive for the ancestral organism.

An example of a successful evolutionary adaptationist explanation is that of the vertebrate heart. The contemporary vertebrate heart has a contractile mechanism that produces the effect of pumping blood around the body. There is evidence from the fossil record that ancestral vertebrates possessed hearts that resemble the hearts of contemporary vertebrates, which supports concordance (Trinajstic et al., 2022). The comparative method reveals that hearts with contractile mechanisms are conserved across multiple taxa that are descended from ancestral vertebrates, which supports strong vertical homology (Jensen et al., 2013; Stevenson et al., 2017). Optimality modeling has suggested that hearts with certain contractile properties pump blood and utilize energy more efficiently, which supports an ultimate explanation of how hearts with similar contractile properties were evolutionarily adaptive (Han et al., 2022; Waldorp et al., 2020). All this evidence supports the evolutionary adaptationist hypothesis that the contemporary vertebrate heart has a contractile mechanism that produces the effect of pumping blood around the body because having a heart that produced such an effect was evolutionarily adaptive for the ancestral vertebrate from which the contemporary vertebrate is descended. Furthermore, this evidence supports a breakdown hypothesis regarding heart failure, according to which heart failure involves the failure of the heart's naturally selected contractile mechanism to produce its adaptive effect of pumping blood.

However, for many of the psychological traits that are involved in mental disorders, the above methods are not available. As Smith notes, the fossil record is unproductive because psychological processes "leave no unambiguous material evidence" (Smith, 2019, p. 47). We only have fragmentary information about the behaviors of our prehistoric ancestors and the information that we do have is too general to support detailed inferences about their cognitive processes and emotional lives. Furthermore, brains tend not to fossilize well because neural tissue decomposes quickly. The casts of cranial cavities may provide views of the external surfaces of ancestral brains, but they do not provide details of the internal cerebral structures from which inferences about our ancestors' cognitive processes and emotional lives could be made (Jerison, 1975). Hence, as Paul Sheldon Davies

notes, "much of the evidence involved has literally long since died and rotted away" (Davies, 1999, p. 75).

The comparative method is also unproductive for evolutionary psychiatry because many of the relevant behaviors are poorly conserved across our closest extant relatives. As noted by Jonathan Kaplan (2002), this partly reflects the large evolutionary distance between us and our closest extant relatives, as the most recent common ancestor humans share with chimpanzees was six million years ago. It also reflects the profound ways in which psychological and behavioral characteristics are influenced by social and cultural circumstances. Some mental disorders involve capacities that seem to be unique to humans and are not shared by other organisms. For example, depressive disorders are marked by certain social and moral emotions such as guilt and shame (Czéh et al., 2016; Krishnan & Nestler, 2011), while psychotic disorders involve certain epistemic and linguistic capacities that are associated with communal meaning making (Crow, 1997; Howard et al., 2019). Moreover, social behaviors are diverse among our closest extant relatives. Chimpanzees, bonobos, gorillas, orangutans, and other primates exhibit significant interspecies and intraspecies variations with regards to their social organizations and ways of interacting, such that behaviors can differ between different communities of the same species (Rawlings et al., 2023; Whiten, 2017). Therefore, comparisons between us and our closest extant relatives do not yield features that are sufficiently stable to support evolutionary adaptationist inferences about mental disorders in humans. To be clear, this does not suggest exceptionalism about humans, but rather acknowledges that the minds of organisms from different species are diverse. Different creatures have different experiences.

Optimality modeling is problematic for evolutionary psychiatry for three related reasons. First, our contemporary environment is vastly different from the prehistoric environment of our ancestors, and so the effect of a psychological trait in the contemporary environment may be very different from the effect of a concordant psychological trait in the prehistoric environment (Smith, 2019). Second, we have very little information about the specific environmental conditions wherein key aspects of the evolution of ancestral humans took place, and so we do not have the relevant detail to model the effect of a psychological trait on evolutionary fitness in the prehistoric environment (Kaplan, 2002). Third, we are highly neuroplastic organisms whose psychological and behavioral characteristics are heavily influenced by our environmental conditions, and so the fact that a given psychological trait had developed in the contemporary environment does not guarantee that a concordant psychological trait would have developed in the prehistoric environment (Sleutels, 2013). Therefore, we are not epistemically situated to assess whether certain psychological traits would have been adaptive for our prehistoric ancestors.

In the case of evolutionary psychology, the unavailability of the usual methodology of evolutionary biology leads to what Smith (2019) calls the matching problem. This is the inability to demonstrate that contemporary psychological traits are phylogenetically descended from ancestral psychological traits which were naturally selected because they were adaptive in the prehistoric environment. In what is to follow, I discuss in further detail how the matching problem also presents a major challenge to evolutionary psychiatry.

The matching problem

As noted earlier, for evolutionary psychiatry hypotheses to be empirically justified, they must demonstrate the following:

(1) Concordance – The psychological mechanisms involved in mental disorders concord with and produce the same sorts of effects as the psychological mechanisms of our ancestors.
(2) Strong vertical homology – The contemporary psychological mechanisms are phylogenetically descended from the ancestral psychological mechanisms.
(3) Ultimate explanation – The psychological mechanisms were naturally selected because their effects were conducive to the survival and reproductive prospects of our ancestors.

In evolutionary biology, these are usually demonstrated with evidence from the fossil record, the comparative method, and optimality modeling. However, as argued above, these methods are not available for many of the psychological traits that are relevant to mental disorders, and so evolutionary psychiatry cannot meet the above conditions.

Given the lack of a substantial fossil record for psychological traits, proponents of evolutionary psychiatry are not epistemically situated to ascertain what the psychological mechanisms of our prehistoric ancestors were like and what effects they had. The information we do have about the behaviors and brains of our prehistoric ancestors is too vague and too fragmentary to support detailed hypotheses about their cognitive processes and emotional lives (Davies, 1999; Jerison, 1975). This is further compounded by the fact that the material and social environment of our prehistoric ancestors was very different from our contemporary environment, which could result in our ancestors having very different psychological traits from us. Indeed, even across different extant cultures, cultural differences in values, norms, and practices have been shown to be associated with differences in the emotional lives of people (Mesquita & Karasawa, 2002). Therefore,

evolutionary psychiatry cannot demonstrate that our prehistoric ancestors had psychological mechanisms which are concordant with the contemporary psychological mechanisms that are involved in mental disorders.

Sometimes, proponents of evolutionary psychiatry suppose that the ancestral psychological mechanisms can be inferred from our contemporary psychological mechanisms. For example, Nesse notes that there is substantial overlap between the contemporary psychological mechanisms involved in depression and anxiety, which he suggests is because "natural selection has partially differentiated several negative affects to deal with different kinds of unpropitious situations" (Nesse, 2000, p. 18). He also notes that depressive episodes tend to be triggered by adverse events that are humiliating or entrapping, which he considers to be evidence that depression was an ancestral mechanism that served "to decrease investment in the current unsatisfying life enterprise and also to prevent the premature pursuit of alternatives" (Nesse, 2000, p. 17). Similarly, Hollon and colleagues note that depressive symptoms tend to be "precipitated by negative or stressful experiences that can include interpersonal conflict" and are associated with neuronal mechanisms that direct attention to the sources of the interpersonal problems (Hollon et al., 2021, p. 2). They then suggest that the same sorts of mechanisms must have been possessed by our prehistoric ancestors because they "served in our ancestral past to keep people focused on complex interpersonal problems until they could arrive at a resolution" (Hollon et al., 2021, p. 1).

However, the above approach does not demonstrate that contemporary psychological mechanisms are concordant with ancestral psychological mechanisms, but merely assumes that they are concordant. This amounts to what Jan Sleutels (2013) calls "the Flintstones fallacy", which is when the traits of modern minds are projected onto ancient minds, even though ancient minds may have been very different from modern minds. The fact that mental disorders involve certain psychological mechanisms is insufficient to establish that our prehistoric ancestors possessed similar psychological mechanisms. As mentioned earlier, we are highly neuroplastic organisms whose psychological characteristics are heavily influenced by our environmental conditions (Pascual-Leone et al., 2005). The problem for evolutionary psychiatry is that the modern environmental conditions wherein our psychological characteristics developed are very different from the environmental conditions of our prehistoric ancestors. Accordingly, the psychological characteristics which we have developed in our contemporary environment may be very different from the psychological characteristics which our ancestors developed in their prehistoric environment. As Sleutels notes, many of our contemporary psychological mechanisms may have been "contrived in relatively recent history as a product of contingent cultural

practices exploiting the considerable bandwidth of human neuroplasticity" (Sleutels, 2013, p. 74). It is, therefore, possible that some mechanisms that are relevant to some mental disorders may be ontogenetic products of the contemporary environment which were not possessed by our prehistoric ancestors (Greenfeld, 2013; López-Ibor & López-Ibor, 2014; Pérez-Álvarez et al., 2016).

An example provided by Smith (2019) of an ontogenically acquired psychological mechanism is the visual word form area, which is a cortical region that is associated with reading. Written language only became established less than six millennia ago (Schmandt-Besserat, 1997), which is a timescale too short for any significant genetic evolutionary change to have resulted from natural selection. This suggests that the visual word form area is highly unlikely to have come about from genetic evolutionary change due to some selective advantage of reading. Rather, it is likely to be an ontogenetically acquired mechanism whose development is occasioned by an environment wherein written language is ubiquitous (Dehaene & Cohen, 2011; Heyes, 2018). Therefore, the fact that we possess certain mechanisms that produce certain behaviors is insufficient to justify the claim that our prehistoric ancestors possessed the same sorts of mechanisms or performed the same sorts of behaviors.

The inability to demonstrate concordance would also entail the inability to demonstrate strong vertical homology, as the former is a necessary condition for the latter. However, for the sake of argument, let us assume that the contemporary psychological mechanisms involved in mental disorders are concordant with certain ancestral psychological mechanisms. Even then, I argue that proponents of evolutionary psychiatry cannot demonstrate strong vertical homology.

For the condition of strong vertical homology to be met, proponents of evolutionary psychiatry must demonstrate that the contemporary psychological mechanisms are phylogenetically descended from the ancestral psychological mechanisms. They must show that a given contemporary psychological mechanism is a genetically inherited trait which has a continuous lineage going back to the corresponding ancestral psychological mechanism. However, there are various reasons why a contemporary trait may resemble an ancestral trait besides genetics. Proponents of developmental systems theory, such as Paul Griffiths, Russell Gray, and Susan Oyama, emphasize that phenotypes are shaped through contingent and dynamic interactions of multiple developmental factors besides genes, including material resources, environmental conditions, social institutions, and cultural practices (Griffiths et al., 1994; Oyama, 2000). Many of these factors can be inherited nongenetically or reconstructed across generations so that they contribute to phenotypic similarities across generations. Again, consider Smith's (2019) example of the visual word form area. Although

written language only appeared recently in the history of our species, social institutions and cultural practices have enabled conditions conducive to the development of reading to persist across generations. And so, even if a contemporary psychological mechanism turns out to be concordant with some ancestral psychological mechanism, it is possible that this concordance is due to the ontogenetic influences of similar environmental resources and not the inevitable outcome of genetics.

Strong vertical homology might be supported if the comparative method yields evidence that the relevant traits have been conserved in multiple descendants of a common ancestor. However, as noted above, the comparative method is unproductive for the psychological mechanisms involved in mental disorders, because many of the relevant behaviors are poorly conserved across our closest extant relatives. Furthermore, some features associated with mental disorders appear to be unique to humans, such as the complex moral emotions involved in depressive disorders and the linguistic capacities involved in psychotic disorders (Crow, 1997; Czéh et al., 2016; Krishnan & Nestler, 2011; van den Heuvel et al., 2019). Therefore, proponents of evolutionary psychiatry do not have the epistemic resources to demonstrate that the contemporary psychological mechanisms involved in mental disorders are related to corresponding ancestral mechanisms through phylogenetic descent.

The inability to demonstrate concordance between given contemporary trait and an ancestral trait would also seem to preclude the prospect of an ultimate explanation, as the proliferation of the trait across generations is precisely what such an ultimate explanation is supposed to explain. Again, for the sake of argument, let us assume that the contemporary psychological mechanisms involved in mental disorders are concordant with certain ancestral psychological mechanisms. Even then, I argue that proponents of evolutionary psychiatry cannot provide an empirically supported ultimate explanation.

For an ultimate explanation to be empirically supported, proponents of evolutionary psychiatry must demonstrate that relevant psychological mechanisms involved in mental disorders were naturally selected because their effects were conducive to the survival and reproductive prospects of our ancestors. However, as noted above, the method of optimality modeling that is usually used to assess adaptiveness in evolutionary biology is unreliable with regards to the psychological mechanisms involved in mental disorders. Given how vague and fragmentary our knowledge of the prehistoric environment of our ancestors is, proponents of evolutionary psychiatry do not have the relevant detail to model the effect of a given psychological mechanism on evolutionary fitness in the prehistoric environment (Kaplan, 2002). Furthermore, given how different the prehistoric environment was from the contemporary environment and given how

sensitive psychological development is to environmental contingencies, the effect of a such a psychological mechanism on evolutionary fitness in the ancestral environment cannot be reliably inferred from the observed effect of a similar psychological mechanism in the contemporary environment. Therefore, proponents of evolutionary psychiatry are not epistemically situated to establish whether the psychological mechanisms involved in mental disorders would have been adaptive, maladaptive, or neutral in the prehistoric environment. This suggests that they cannot justify the claim that psychological mechanisms were naturally selected because they their effects were conducive to the survival and reproductive prospects of our ancestors.

And so, the matching problem indicates that evolutionary psychiatry hypotheses are unable to demonstrate three key features of successful evolutionary adaptationist explanations. Due to methodological limitations, they cannot demonstrate that the contemporary psychological mechanisms involved in mental disorders are concordant with ancestral psychological mechanisms, that the contemporary psychological mechanisms are phylogenetically descended from the ancestral psychological mechanisms, or that the relevant psychological mechanisms were naturally selected because they were adaptive in the prehistoric environment. I now consider some of the deeper epistemic implications of the above for evolutionary psychiatry.

Underdetermination of theory by data

The matching problem is a significant challenge for evolutionary psychiatry, not only because it suggests that its hypotheses are empirically unsupported, but also because it suggests that they are to significant extents empirically untestable. As noted earlier, evolutionary psychiatry hypotheses tend to fall into three sorts, namely breakdown, mismatch, and persistence hypotheses (Murphy, 2005), which can yield competing hypotheses of specific mental disorders. It might be hoped that further empirical evidence could help us to select between these competing hypotheses. However, the matching problem suggests that such empirical evidence is not forthcoming. Given the lack of a substantial fossil record, the unavailability of the comparative method, and the unreliability of optimality modeling for the relevant psychological traits, proponents of evolutionary psychiatry are not epistemically situated to yield the sorts of evidence that would disconfirm certain hypotheses or provide differential support for one hypothesis over others. Therefore, some evolutionary psychiatry hypotheses may indefinitely remain underdetermined by data.

Underdetermination of theory by data refers to the idea, associated with Pierre Duhem (1906), that the available empirical evidence is insufficient to select between empirically equivalent hypotheses. Consider, for example,

what would be required to select between breakdown, mismatch, and persistence hypotheses of depression. A breakdown hypothesis claims that depression comprises the failure of a psychological mechanism to produce the effect for which it was naturally selected (Stevens & Price, 1996; Wakefield, 1992), whereas mismatch and persistence hypotheses claim that depression comprises the ongoing effect for which the psychological mechanism was naturally selected (Andrews & Thompson, 2009; Hollon et al., 2021; Nesse, 2000; Price et al., 1994). To select between these competing claims, proponents of evolutionary psychiatry must establish what sort of ancestral psychological mechanism is supposed to have been naturally selected and what adaptive effect it is supposed to have produced. The breakdown hypothesis might suggest that the relevant ancestral psychological mechanism was a "sadness and loss-response regulating" mechanism which was performing its adaptive effect when it was staving off depressive behavior (Wakefield, 2007, p. 152), whereas the mismatch and persistence hypotheses suggest that the relevant ancestral psychological mechanism was performing its adaptive effect when it was producing depressive behavior. However, due to the matching problem, proponents of evolutionary psychiatry cannot establish which of these, if any, is correct. They do not have the methods to demonstrate what the relevant psychological mechanisms of our prehistoric ancestors were like or what sorts of effects would have been adaptive.

Mismatch and persistence hypotheses about depression both claim that depression was an effect of a psychological mechanism that was adaptive in the ancestral environment, but they differ over whether such an effect is still adaptive in the contemporary environment. To some extent, the effect of a trait on survival and reproduction in a given environment can be tested. For example, a study by Nicholas Jacobson (2016) examined the associations between different psychiatric diagnoses, fertility rates, and interactions between parents and children in a national sample. The results were mixed, with some psychiatric diagnoses being associated with higher fertility rates and others being associated with lower fertility rates in the sample population. However, even these results are insufficient to provide differential support for mismatch and persistence hypotheses. First, Jacobson acknowledges that the effects of traits depend on the developmental and environmental contexts, and so a given psychiatric diagnosis may be associated with decreased fertility in one contemporary cultural setting but be associated with increased fertility in another contemporary cultural setting. Second, while we may be able to measure the correlations between certain psychological mechanisms and fertility rates in a given contemporary environment, we cannot, due to the matching problem, demonstrate whether corresponding psychological mechanisms were correlated with fertility rates in similar ways in the

prehistoric environment, or indeed whether our prehistoric ancestors possessed these psychological mechanisms at all. Therefore, data about how mental disorders are correlated with fertility rates in the contemporary environment do not tell us if mental disorders comprise breakdowns, mismatches, or persisting adaptations, because we still cannot establish that the relevant contemporary psychological mechanisms are phylogenetically descended from corresponding ancestral psychological mechanisms that were adaptive in the prehistoric environment.

As well as being unable to select between breakdown, mismatch, and persistence hypotheses, proponents of evolutionary psychiatry are unable to exclude non-adaptationist explanations. An example of a non-adaptationist hypothesis is offered by Somogy Varga (2011), who suggests that the psychological mechanisms involved in mental disorders may have been adaptively neutral byproducts of evolved structures. This recalls Stephen Jay Gould and Richard Lewontin, who note that the effect that a trait currently produces may not be the reason it evolved (Gould & Lewontin, 1979). Hence, it is possible that certain cognitive structures were naturally selected because they enabled domain-general learning and that mental disorders were byproducts of these structures that did not affect survival or reproductive fitness. Another example of a non-adaptationist hypothesis is the view that the psychological mechanisms involved in some mental disorders are ontogenetic products of our modern social and material environment. As noted earlier, we are highly neuroplastic organisms whose psychological and behavioral characteristics are profoundly influenced by our environmental conditions, which are very different from the environmental conditions of our prehistoric ancestors. Hence, rather than involving psychological mechanisms that were phylogenetically descended from corresponding psychological mechanisms of our ancestors, some scholars have hypothesized that some mental disorders involve relatively novel psychological mechanisms that develop through interactions with the modern environment (Greenfeld, 2013; López-Ibor & López-Ibor, 2014; Pérez-Álvarez et al., 2016).

The matching problem suggests that proponents of evolutionary psychiatry cannot empirically adjudicate between adaptationist explanations and non-adaptationist explanations. As noted above, the methods are not available to demonstrate that the psychological mechanisms involved in mental disorders are phylogenetically descended from corresponding ancestral psychological mechanisms that were adaptive in the prehistoric environment. Therefore, the data are just as consistent with mental disorders being adaptively neutral byproducts of evolved structures or ontogenetic products of modern environmental conditions. The ontogenetic hypothesis may even be more plausible and easier to test than evolutionary adaptive explanations, because nongenetic factors can produce observable changes on much

shorter time scales than genetic factors. For example, the finding that urbanicity is associated with increased rates of depressive disorders and psychotic disorders comprises evidence of the effects of modern environmental conditions on our psychological mechanisms (van Os, 2018).

Of course, it could be contended that all psychological theories are underdetermined by data to certain degrees. For example, cognitive-behavioral, attachment, and psychoanalytic theories of mental disorders are underdetermined by data insofar as they invoke psychological states and processes that are not directly observable, yet these theories have inspired effective therapies. Nonetheless, I argue that there are still important differences between evolutionary psychiatry and some of the above psychological theories. While cognitive-behavioral and attachment theories do posit some unobservable psychological processes in their explanations, they do also generate specific hypotheses which make novel predictions about presently observable states of affairs that are empirically testable. Notably, cognitive-behavioral theory has informed the hypothesis that interventions on certain cognitive and behavioral processes can result in sustained changes in certain affective and physiological responses (Clark et al., 2006). Similarly, attachment theory has informed the hypothesis that parental deprivation in childhood contributes to the subsequent development of psychopathy and antisocial behavior, which has been empirically confirmed through retrospective and prospective studies (Gao et al., 2010; Waller et al., 2012). Here, as with other scientific theories, the empirical confirmation of the specific hypothesis provides a justification for accepting the unobservable processes posited by the general theory.

By contrast, the problem with evolutionary psychiatry is that its specific hypotheses are not merely hypotheses about presently observable states of affairs, but are hypotheses about the evolutionary origins of certain psychological mechanisms. Given the matching problem, proponents of evolutionary psychiatry lack the methods to ascertain these evolutionary origins, and so cannot empirically confirm or disconfirm their hypotheses. Instead, they have to rely on untestable assumptions about the psychological properties of our prehistoric ancestors. This results in circular reasoning, whereby the properties of modern minds are assumed to have also been possessed by ancient minds and then these assumed properties of ancient minds are invoked to explain the properties of modern minds, without any independent evidence that ancient minds actually possessed these properties (Sleutels, 2013). Hence, evolutionary psychiatry hypotheses are not testable in ways that certain cognitive-behavioral and attachment hypotheses are. Whereas, for example, the greater effectiveness of cognitive therapy over exposure therapy for the alleviation of social anxiety comprises evidential support for one specific cognitive-behavioral hypothesis over another (Clark et al., 2006), empirical observations about our contemporary

psychological mechanisms do not differentially support breakdown, mismatch, persistence, or non-adaptationist hypotheses.

The comparison with psychoanalytic theory is perhaps more apt, given that psychoanalytic theory has also come under philosophical scrutiny for being empirically unsupported. Famously, Karl Popper (1962) argued that psychoanalysis is unfalsifiable, whereas Adolf Grünbaum (1984) argued that it has been discredited. These criticisms have cast doubt over the scientific status of psychoanalysis. Accordingly, instead of defending it as a science, Sebastian Gardner (1993) suggests that psychoanalysis is an extension of ordinary psychology. Under this view, psychoanalysis can yield useful therapies and provide meaningful explanations, but these explanations are more akin to ordinary psychological explanations than to scientific explanations. The trouble for evolutionary psychiatry is that it presents its explanations as scientific explanations based on "the principles of evolutionary biology" (Nesse, 2023, p. 178). However, due to the matching problem, evolutionary psychiatry explanations cannot meet the conditions that are required of evolutionary biology explanations. And so, while they may make compelling stories, evolutionary psychiatry hypotheses about mental disorders indefinitely remain underdetermined by data.

Objections and replies

Objection 1: undue pessimism

It could be objected that I am being too pessimistic about how much we know about our prehistoric ancestors. For example, John Tooby and Leda Cosmides note:

> Our ancestors nursed, had two sexes, hunted, gathered, chose mates, used tools, had color vision, bled when wounded, were predated upon, were subject to viral infections, were incapacitated from injuries, had deleterious recessives and so were subject to inbreeding depression if they mated with siblings, fought with each other, lived in a biotic environment with felids, snakes, and plant toxins, etc. (Tooby & Cosmides, 1997)

Hence, although there may be no unambiguous fossil evidence for specific cognitive and behavioral traits, we still know some general facts about the lives of our prehistoric ancestors from which we can make inferences.

However, in reply, I argue that the information we do have about our prehistoric ancestors is too general to support specific hypotheses about specific mechanisms that are relevant to mental disorders. Caring for infants, using tools, hunting, gathering, having color vision, being vulnerable to predators, and being susceptible to viral infections are features that admit a wide and diverse range of social organizations and norms for behaving, which could be support the development of a variety of different

possible psychological mechanisms. That is to say, the above features vastly underdetermine the sorts of psychological mechanisms that our ancestors could have developed. Furthermore, even if it is shown that a certain feature was adaptive for our prehistoric ancestors, it would still be possible that such a feature was conserved not through genetic inheritance, but through cultural processes. For example, Marion Godman (2018) proposes that gender systems are historical kinds that are reinforced across generations through social norms, practices, and institutions, which both accounts for the diversity of gender systems across cultures and why gender systems are so persistent within cultures. Therefore, the information that we do have about our prehistoric ancestors is insufficient to demonstrate the conditions of concordance, strong vertical homology, and ultimate explanation.

Objection 2: shared mechanisms

While many features of mental disorders involve psychological capacities that are restricted to humans, such as the complex moral emotions involved in depressive disorders and the linguistic capacities involved in psychotic disorders (Crow, 1997; Czéh et al., 2016; Krishnan & Nestler, 2011; van den Heuvel et al., 2019), there are also features of mental disorders that involve mechanisms which are shared by other organisms. For example, anxiety disorders involve activity of the sympathetic nervous system (Thayer et al., 1996) and depressive disorders involve inflammation (Maes, 1995). These are physiological mechanisms which evolved in vertebrates long before humans diverged from other apes, and so are conserved across many different extant taxa that were descended from ancestral vertebrates.

Indeed, the above does offer valuable knowledge about some of the evolved physiological systems that can be involved in certain mental disorders. Nonetheless, I argue that this is insufficient to support the sorts of adaptationist explanations that are conjectured by evolutionary psychiatry. First, the above physiological mechanisms, while they may be involved in some mental disorders, neither exhaust the mental disorders nor are specific to the mental disorders. Mental disorders are complex conditions that involve dynamic and reciprocal interactions between psychological, biological, and social processes. An evolutionary explanation of inflammation would not be an evolutionary explanation of depression, but would merely be an evolutionary explanation of inflammation, because depression involves much more than just inflammation and because inflammation can occur in many other contexts aside from depression. Rather, a comprehensive explanation of a mental disorder such as depression would have to account for the characteristic psychological and social features in addition to the associated physiological mechanisms. However, as noted earlier, some of these psychological and social features appear

restricted to humans and are poorly conserved across our nearest extant relatives, and so cannot be studied through the comparative method.

Second, the fact that a given physiological mechanism is involved in a mental disorder underdetermines whether its involvement is adaptive or maladaptive in this specific context. Sympathetic nervous system activity and inflammation are general mechanisms that are involved in all sorts of processes besides anxiety and depression. Some of these processes are adaptive, such as the maintenance of energy metabolism and the responses to exercise, injury, and infection, whereas some of these processes are maladaptive, such as hypertension, cardiovascular disease, and autoimmune conditions. Hence, the mere involvement of such a physiological mechanism in a bodily process does not tell us whether this involvement comprises a breakdown, a mismatch, or a persisting adaptive effect. Rather, to establish whether the sorts of sympathetic nervous system activity and inflammation that occur in the specific contexts of anxiety and depression comprise breakdowns, mismatches, or persisting adaptive effects, we would need to establish whether these mechanisms also occurred in the same sorts of contexts in our prehistoric ancestors and, if so, whether they were adaptive in these contexts. However, given the lack of fossil record and the unavailability of the comparative method, we are not epistemically situated to answer those questions.

Objection 3: psychiatric genetics

Another potential objection is that evolutionary psychiatry could be empirically supported by research in psychiatric genetics. As noted by Pieter Adriaens, evolutionary psychiatrists commonly assume that for a given mental disorder there is "an orderly and limited number of major impact genes that code for the susceptibility to this disorder" (Adriaens, 2007, p. 518). If this turns out to be the case, then it would support the possibility that mental disorders are phylogenetically inherited traits that were amenable to genetic natural selection.

Research in quantitative behavioral genetics, which relies on patterns of certain traits in twins, siblings, other family members, and genetically unrelated people, has suggested that major depressive disorder has a heritability of 37% (Sullivan et al., 2000). This means that 37% of the variation in depressive symptoms in the study population was correlated with genetic variation in that population. To be clear, heritability is not a measure of the degree to which a trait is genetically caused, but is a measure of the degree to which phenotypic variation correlates with genetic variation in a given population (Lewontin, 1974). Hence, a high heritability does not imply insensitivity to environmental changes, as the heritability for a given trait may differ across different environmental

conditions. Nonetheless, a heritability of 37% does suggest that differences in the vulnerability to depression in that specific study population partly correlate with genetic differences.

However, I argue that the findings of psychiatric genetics are insufficient to support evolutionary psychiatry hypotheses. First, the assumption that there are "an orderly and limited number of major impact genes" that code for vulnerabilities for mental disorders has been empirically discredited (Adriaens, 2007, p. 518). Research in molecular genetics, which examine genome-wide associations between genes and phenotypic traits, has found no reliable associations between specific genes or sets of genes and depressive disorders (Border et al., 2019). Rather, it has been suggested that there are over a hundred different genetic variants that are weakly involved in depression, none of which are necessary or sufficient for the development of depression and many of which are not specific to depression (Howard et al., 2019). Furthermore, such research in molecular genetics has yielded a heritability estimate of 8.9%, which is considerably lower than the 37% estimated by the research quantitative behavioral genetics. Accordingly, some scholars have suggested that the observed concordances of some mental disorders among family members could partly be due to other mechanisms as well as genetic inheritance, such as cultural, behavioral, epigenetic inheritance (Crow, 2008; González-Pardo & Pérez-Álvarez, 2013).

Second, even if it turns out that a certain genotype is associated with the development of depression in a given environment, this would be insufficient to support a specific evolutionary psychiatry hypothesis. As molecular geneticists have pointed out, it may be possible to discern the influence of an allele on an extant trait in a specific environment and to find evidence that this allele has been conserved across generations, but this evidence would still fail to demonstrate that the allele was conserved because it had a specific adaptive effect (Barrett & Hoekstra, 2011; Nielsen, 2009). Philosophers of biology have emphasized that the ways in which genetic and environmental factors interact to produce phenotypic traits are contingent and context sensitive (Griffiths et al., 1994; Lewontin, 1974; Oyama, 2000). The effect of any given developmental resource, whether it is a gene or an environmental factor, is dependent on the state of the rest of the developmental system, which comprises the organism and the interactions with the environment wherein the organism is embedded. Hence, a certain phenotypic outcome may be associated with a genotype in a certain environment but a different phenotypic outcome may be associated with the same genotype in a different environment. Given that our modern environment is very different from the prehistoric environment of our ancestors, it is possible that a certain pattern of genetic variation may be correlated with susceptibility to mental disorder in our modern environment but may not have

been correlated with any such susceptibility in the prehistoric environment. Indeed, even on a smaller timescale, Bolton and Bhugra (2021) note that there have been increasing incidences of mental disorders among young people in recent decades despite the genetic properties of people being the same, which indicates that social and environmental changes are major difference makers in what phenotypic outcomes are associated with these genetic properties. Therefore, due to the contingency and context sensitivity of trait development, phenotypic concordance cannot be straightforwardly inferred from genetic similarity on its own. We cannot rule out the possibility that the genes which contingently happen to be involved in mental disorders in our modern environment were conserved because they were associated with entirely different adaptive effects in our ancestors or because they were adaptively neutral in our ancestors.

Objection 4: the prevalence of mental disorder

A related objection is that the uniform prevalence of mental disorder across different geographical regions suggests that mental disorder has been a part of the evolutionary history of our species for a sufficient length of time for it to have been influenced by genetic natural selection. For example, in his defense of an evolutionary account of schizophrenia, Jonathan Burns suggests that "the fact that this disorder is found universally implies that the schizophrenic genotype dates to at least 150–100,000 years ago when the migration of *Homo erectus* out of Africa occurred" (Burns, 2004, p. 832). This is taken to lend plausibility to an evolutionary explanation of mental disorder.

I offer two responses to this objection. First, the prevalence of mental disorder is much more geographically heterogeneous than evolutionary psychiatrists assume. Psychotic disorders and depressive disorders have been shown to be more prevalent in urban areas marked by social fragmentation and deprivation (van Os, 2018). Schizophrenia has complex epidemiological patterns across different countries and across different communities within countries (Adriaens, 2007). Hence, the assumption that mental disorder has a geographically uniform prevalence is not empirically supported. This does not undermine the claim that there may be genetic factors that are associated with susceptibility to mental disorder in the present population, but it does suggest that the associations between these genetic factors and susceptibility to mental disorder are highly contingent on the environmental conditions that shape development. The uniform prevalence claim supposes that the "schizophrenia genotype" is associated with the same phenotype across all environmental conditions, prehistoric and modern. However, this is discredited by the complex epidemiological patterns of schizophrenia, which show that environmental

conditions are major difference makers in what phenotypic outcomes are associated with a given genotype. Again, in view of this contingency and context sensitivity, it is possible that the genetic factors which happen to be associated with schizophrenia susceptibility in the modern environment were not associated with such susceptibility in the prehistoric environment. For example, these genetic factors may have had different phenotypic effects in our prehistoric ancestors and may have only recently become associated with schizophrenia through their interactions with modern cognitive mechanisms shaped by the modern environment. And so, given the complex and varied epidemiological patterns of schizophrenia, proponents of evolutionary psychiatry do not have sufficient evidence to support the claim that schizophrenia has been around for over a hundred thousand years.

Second, even if a psychological trait does turn out to have a uniform prevalence, then this would not necessarily entail that the trait was genetically selected. Again, consider Smith's (2019) example of the visual word form area, which is a cortical region associated with reading. Given that written language was only established less than six millennia ago (Schmandt-Besserat, 1997), which is a timescale too short for any significant genetic evolutionary change to have resulted from natural selection, it is highly unlikely that the visual word form area is a genetically selected trait. Rather, it is likely to be an ontogenetically acquired trait whose development and widespread prevalence in our population are due to the ubiquity of written language across many cultures (Dehaene & Cohen, 2011; Heyes, 2018). And so, evolutionary psychiatry's assumption about uniform prevalence indicating a genetic basis underestimates the extent to which social and environmental factors are shared across cultures in an increasingly globalized postindustrial and postcolonial world. It is entirely possible that the geographically uniform prevalence of a mental disorder could be due to uniform aspects of the contemporary environment that are shared across cultures.

Conclusion

I have shown herein that a version of Smith's (2019) matching problem presents a serious challenge to evolutionary psychiatry. According to evolutionary psychiatry, mental disorders involve psychological mechanisms that have been conserved through natural selection because they were adaptive for our prehistoric ancestors. Competing evolutionary psychiatry hypotheses suggest that mental disorders are (a) breakdowns of these mechanisms, (b) mismatches between these mechanisms and the contemporary environment, or (c) persisting adaptive effects of these mechanisms. In order to support the above empirically, evolutionary psychiatry must demonstrate (1) that the contemporary psychological mechanisms involved in mental

disorders are concordant with the psychological mechanisms of our prehistoric ancestors, (2) that the contemporary mechanisms are phylogenetically descended from the prehistoric mechanisms, and (3) that the mechanisms were conserved through natural selection because they were conducive to survival and reproduction for our prehistoric ancestors. However, due to the lack of unambiguous fossil evidence, the unsuitability of the comparative method, and the untenability of optimality modeling for psychological traits, evolutionary psychiatry cannot demonstrate these three conditions. Therefore, evolutionary psychiatry lacks the epistemic resources to establish whether breakdown, mismatch, or persistence hypotheses are correct, or indeed if any of them are correct.

The matching problem does not challenge the general claim that evolutionary processes have influenced our cognitive and affective capacities in ways that may make us susceptible to certain forms of psychological distress. This general claim is plausible. It also does not challenge the claim that some features of certain psychiatric conditions may sometimes have epistemic benefits and social meanings in our present environment. Rather, the matching problem indicates that specific evolutionary psychiatry hypotheses about the evolutionary origins of specific mental disorders rely on assumptions that are empirically untestable. Accordingly, evolutionary psychiatry hypotheses may indefinitely remain underdetermined by the data. Given that we are versatile organisms whose cognitive and affective capacities are highly sensitive to the social, cultural, and material settings wherein we develop, it may be more constructive for the purpose of understanding mental disorders to study how our present psychological characteristics have been shaped by our modern environment than to speculate about the past psychological characteristics of our prehistoric ancestors. Evolutionary psychiatry may offer compelling stories, but caution is warranted regarding how much credence is granted to these stories as scientific explanations.

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