The Products of Evolution: Conceptual Distinctions, Evidentiary Criteria, and Empirical Examples

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Evolution refers to a change in allele frequencies in a population over time. Change in allele frequencies can result from four different forces: mutation, genetic drift, gene flow, and natural selection (e.g., Bergstrom and Dugatkin, 2012; Futuyma and Kirkpatrick, 2017). Mutation is the random change in alleles that constitute the raw material on which selection operates. Genetic drift is the random, chance-driven change in allele frequencies from one generation to the next. Gene flow, sometimes called admixture or migration, is the movement of genes from one population to another. Natural selection is the non-random filtering of randomly mutated genes as a function of differential reproductive success. These four evolutionary forces yield three kinds of products: adaptations, byproducts, and noise. In this chapter, we discuss these three products of evolution.

ADAPTATIONS

An adaptation is a functionally organized biological system that evolved to solve an adaptive problem (Tooby and Cosmides, 1992; Williams, 1966). Adaptations can be morphological, physiological, or psychological. Their most salient feature is that they exhibit functional design (Dennett, 1996; Tooby and Cosmides, 2015; Williams, 1966) – that is, adaptations have a function. For example, the porcupine's quills serve the function of protection, as does the African bombardier beetle's (*Stenaptinus insignis*) ability to project chemical explosions out of its body at potential attackers (Eisner and Aneshansley, 1999). The larvae of the Alcon blue butterfly *(Phengaris alcon)* emit chemical signals designed to manipulate ants into caring for them and feeding them, sometimes

to the exclusion of the ants' own offspring (Thomas and Elmes, 1998). The emerald cockroach wasp (*Ampulex compressa*) parasitizes the common household cockroach (*Periplaneta americana*) by injecting it with a precise neurochemical cocktail in two different locations in its brain, turning it into a 'zombie' (Piek et al., 1984). These neurochemical cocktails are specifically designed to rob the cockroach of its ability to *initiate* movement, but *not* actually paralyze it. This is crucial, because the tiny wasp needs to drag the much larger roach to its nest, a feat that would be impossible if the roach were paralyzed. The wasp's solution is to inject the roach with a cocktail that nullifies its willingness to *initiate its own movement,* but does not actually paralyze it – this way, when the wasp begins to drag the roach toward the nest, the roach's legs automatically move in concert, enabling the wasp to drag it. Once it reaches the nest, the wasp lays an egg on the roach's abdomen and buries it alive. When the wasp egg hatches, the larva that emerges eats the roach's internal organs in a precise sequence that keeps the roach alive (and thereby the food fresh) for as long as possible, while also producing another chemical cocktail designed to sanitize the roach and protect itself from pathogenic infection (Herzner et al., 2013). These are examples of adaptations: 'special problem-solving machinery' (Williams, 1985: 1) designed by selection to solve a specific survival- or reproduction-related problem.

Of the four evolutionary forces (selection, mutation, migration, and genetic drift), only selection is capable of producing adaptations. The other three forces can cause evolution – a change in allele frequencies in a population over time – and they can also affect the features of adaptations. But natural selection is the only known causal process capable of crafting an adaptation in the first place; it is the only evolutionary force capable of producing a functional fit between an organism and its environment. In the following sections, we discuss misconceptions about adaptations, key features of adaptations, and how to test adaptation-based hypotheses.

Misconceptions About Adaptations

Common misconceptions about adaptations include the ideas that (1) adaptations must be present at birth, (2) adaptations are genetically determined, (3) adaptations must be *currently adaptive*, and (4) adaptations cannot have maladaptive effects. We tackle these misconceptions briefly before presenting the key features of adaptations.

Do adaptations need to be present at birth?

A common misconception about evolved adaptations is that they must be present at birth (e.g., see Al-Shawaf et al., 2018b). This is incorrect: selection builds adaptations that emerge at the developmental phase in which they are needed, not ones that are necessarily present at the moment of birth (Al-Shawaf et al., 2018b). Walking and language are two human adaptations that are not present at birth, but emerge later in development. So are beards, breasts, and teeth. Hatchlings of many bird species cannot see or fly, and yet vision and flight are both avian adaptations – they are not present at birth, but they emerge later in development, during the ontogenetic phase in which they are needed (see, e.g., Alcock, 2013; Al-Shawaf et al., 2018b; Williams, 1966).

Are adaptations genetically determined?

Adaptations, like all other features of the body, brain, and mind, are produced by the joint interplay of genes and environment (e.g., Buss, 1995; Dawkins, 1976; Tooby and Cosmides, 1992). There is a tendency to conflate 'evolved' with 'genetically determined', but this conflation is mistaken. Adaptations are sensitive to contextual and environmental input (Al-Shawaf et al., 2019). Evolutionary thinking highlights the central causal role of the environment in

the emergence of adaptations. Specifically, environmental pressures drive the evolution of adaptations in the first place, are critical for the normal development of adaptations during an organism's life, and are needed to activate adaptations in the immediate present (e.g., Buss, 1995; Confer et al., 2010; Lewis et al., 2017). Even something as 'basic' as the visual system requires environmental input in order to develop normally (e.g., Wiesel, 1982). Far from adopting a 'genetic determinist' view, an evolutionary approach emphasizes the importance of the environment at every phase of an adaptation's emergence (Al-Shawaf et al., 2019; see also Boyer and Bergstrom, 2011; Tooby and Cosmides, 2015).

Do adaptations need to be currently adaptive?

Whether an organismic feature is an adaptation is distinct from whether it is *currently adaptive*. Adaptations may *not* be currently adaptive for many reasons. These include the fact that selection is subject to time lags, and, for humans, cannot keep up with some of the rapid changes in our environment since the agricultural revolution 12,000 years ago (Dawkins, 1999; Tooby and Cosmides, 1992; but see Cochran and Harpending, 2009 for important exceptions). Adaptations that were beneficial to humans during most of our evolutionary history as hunter-gatherers can be positively harmful in the modern age, leading to outcomes such as obesity, heart disease, and drug addictions (Al-Shawaf and Zreik, 2018; Nesse and Williams, 1994).

Later in this chapter, we discuss evidentiary standards for establishing whether something is an adaptation. For now, we simply note that being *currently adaptive* (i.e., currently linked with reproductive success) is not one of them.

Can adaptations have maladaptive effects?

It may sound surprising, but adaptations can have maladaptive effects for several reasons (Del Giudice, 2018). Below, we discuss (1) malfunctioning adaptations, (2) evolutionary time lags or mismatches, (3) maladaptive outcomes due to evolutionary conflicts, (4) adaptively biased mechanisms that make maladaptive mistakes as part of their evolved design, and (5) other constraints on natural selection. For more in-depth discussions, see Nesse (2015), Crespi (2000, 2014), and Del Giudice (2018).

Malfunctioning adaptations

Adaptations can malfunction for numerous reasons, ranging from degradation due to pathogens and parasites to environmental circumstances that occur outside the adaptation's normal range of sensitivity. One example of a malfunctioning adaptation is theory of mind in individuals on the autism spectrum. In people with autism, this adaptation for understanding others' thoughts and emotions is impaired (Baron-Cohen, 1996, but see Gernsbacher and Yergeau, 2019). As Nesse has pointed out, when faced with a disorder or disease, the correct question is rarely 'why did this disease evolve?', but rather 'why did selection fashion the body or mind in ways that left us vulnerable to this disease?' (Nesse, 2019).

Maladaptive outcomes due to environmental mismatches

Adaptations can also have maladaptive effects due to environmental mismatches (see Dawkins, 1999; Symons, 1992). Preferences for sugar and calorically dense foods were adaptive during human evolution, when these resources were scarce. However, in modern environments where these resources are available at any time of day and in nearly unlimited quantities, these ancestrally adaptive preferences motivate people to consume unhealthy amounts of sugar and excess calories, leading to maladaptive outcomes such as obesity and Type 2 diabetes (see, e.g., Symons, 1992).

The prevalence of pornography in modern environments captures another evolutionary mismatch. During human evolution, before the advent of pixels on a screen, the retinal

projection of a naked and interested conspecific would have represented a real sexual opportunity. Because pixels on a screen were not part of the environment in which humans evolved, our species has not had sufficient evolutionary time to evolve neurocognitive mechanisms that render these virtual simulacra ineffective.

In short, phenomena such as widespread consumption of refined sugar and pornography are maladaptive effects produced by adaptations due to evolutionary mismatch (for more information, see Al-Shawaf and Zreik, 2018; Dawkins, 1999).

Maladaptive outcomes due to evolutionary conflicts, including intragenomic conflict

Different organisms do not have perfectly overlapping fitness interests, so adaptations can produce maladaptive outcomes due to unavoidable conflicts between individuals (Del Giudice, 2018; Trivers, 1974). For example, mother and fetus engage in evolutionarily predictable conflicts during pregnancy because their fitness interests partially diverge (Trivers, 1974). Similarly, conflict within the genome between maternally imprinted and paternally imprinted genes ('intragenomic conflict') can lead to maladaptive outcomes for the developing fetus (Haig, 1993, 1997). Disequilibria in these intragenomic conflicts have been implicated in certain psychological disorders, including autism and schizophrenia (Badcock and Crespi, 2006; Byars et al., 2014; Crespi and Badcock, 2008; Wilkins, 2011).

Maladaptive outcomes due to 'adaptively biased mechanisms' that produce errors as part of their design

Some adaptations even produce errors *as part of their design*. For example, research on error management theory has shown that inferential mechanisms can evolve to be 'adaptively biased' rather than perfectly accurate, as long as the net costs of a mechanism that errs on the side of caution are lower than the net costs of a maximally accurate mechanism (the latter would make fewer errors, but a larger proportion of them would be in the more costly direction – and selection minimizes net costs, not crude error rate; Haselton and Buss, 2000; Haselton and Nettle, 2006). This logic has led researchers to discover predictable errors in mating cognition (Haselton and Buss, 2000; Henningsen and Henningsen, 2010), environmental navigation (Jackson and Cormack, 2007, 2008), physiological defenses (Nesse, 2001, 2005), auditory perception (Neuhoff, 1998, 2001), and many other domains of psychology (Haselton and Nettle, 2006). These individual errors are often maladaptive, but they are normal and expected outputs of the 'adaptively biased' mechanisms that produce them.

Maladaptive outcomes due to constraints on natural selection

Adaptations do not evolve because they perform perfectly; they evolve because they perform better, on average, than alternative variants extant in the population at the time (Williams, 1966). This means that adaptations are often adaptive, on average, but nonetheless imperfect. Moreover, adaptations are necessarily imperfectly designed because there are constraints on the ability of selection to craft optimally designed mechanisms – and this, too, can lead to maladaptive effects. These constraints include time lags, phylogenetic or historical constraints, lack of genetic variation, unavoidable tradeoffs, environmental accidents, antagonistic pleiotropy, imperfections due to genic-level selection having deleterious effects at the individual level, and tradeoffs between survival and reproduction (see Al-Shawaf and Zreik, 2018; Dawkins, 1999; Nesse and Williams, 1994).

Fuller discussions of these constraints can be found elsewhere (see, e.g., Al-Shawaf and Zreik, 2018; Dawkins, 1999), but here we offer one example of unavoidable tradeoffs. Wild dogs (*Lycaon pictus*) prey upon gazelles (*Gazella thomsoni*) in the African Serengeti. These gazelles could evolve longer

legs, enabling them to run faster and escape their predators, but longer leg bones would be more brittle and more likely to break. As a consequence, the gazelles face an unavoidable tradeoff: more robust bones but higher likelihood of being caught by a predator, or longer, more gracile bones that enable faster escape but are more likely to break. The key point is that organisms face unavoidable tradeoffs, and these tradeoffs make it impossible for selection to optimize all the relevant parameters at once. This leads to non-optimal outcomes that are maladaptive on at least one of these parameters. For these and other reasons, natural selection is best conceptualized as a 'meliorizing' force, not an 'optimizing' one (Dawkins, 1999).

Key Features of Adaptations

Adaptations are typically universal at the information-processing level, but may not be universal at the behavioral output level

To think clearly about psychological adaptations, it is essential to distinguish between behavior and the underlying information-processing mechanisms that produce behavior. This distinction is key because most evolutionary approaches suggest that the underlying neurocognitive mechanisms that produce behavior will be universal, but that the behaviors themselves will not necessarily be.

Psychological adaptations can be understood in terms of three components: (1) inputs, which can be external (e.g., ecological, sociocultural) or internal (e.g., body temperature, immune function), (2) algorithms that process those inputs, and (3) outputs (including behavior) produced by those algorithms and decision rules (Lewis et al., Chapter 6, this volume). Evolutionary approaches to psychology typically suggest that the *information-processing structure* of the adaptation will be universal, not the behavior that the adaptation produces (Al-Shawaf and Lewis, 2017; Symons, 1992; Tooby and Cosmides, 1992). For example, people from different cultures may speak different languages (behavioral output), but appear to be equipped with the same universal evolved language learning abilities (neurocognitive mechanisms). In this way, cross-cultural variation in behavior does not conflict with an evolutionary perspective; rather, it can often be predicted *a priori* on the basis of evolutionary thinking (for example, see Al-Shawaf, 2019; Gangestad and Buss, 1993; Gangestad et al., 2006; Schmitt, 2005). This same distinction between the neurocognitive or information-processing level of analysis and the behavioral output level of analysis also sheds light on how universal psychological adaptations can yield individual differences in behavior (Lewis et al., in press).

Learning is driven by evolved learning adaptations

A commonly held view is that learning and evolution are conflicting explanations for behavior. If something is learned, it's not evolved, and vice versa – at the very least, some things are 'more learned', whereas others are 'more evolved'. This way of thinking is neither accurate nor useful.

Organisms learn by virtue of evolved learning mechanisms instantiated in their brains. Evidence suggests that humans, for example, have evolved learning mechanisms for language, fear of snakes and spiders, and avoiding incest (e.g., Lieberman et al., 2007; LoBue et al., 2010). Humans are not born speaking a language or already fearing snakes – nor are they born feeling disgust at the notion of incest. We must learn these things, but we accomplish this learning by virtue of evolved learning mechanisms designed for these purposes. This is also why different species will learn different things given the same inputs: they are equipped with different evolved learning mechanisms, and what they come away with given any set of inputs depends on the nature of the evolved learning mechanisms instantiated in their brains (e.g., Al-Shawaf et al., 2019). For example,

if rats and quail are both exposed to sour blue water and subsequently fall ill, the rats will preferentially avoid sour water in the future, whereas the quail will preferentially avoid blue water (Wilcoxon et al., 1971). Because birds attach greater importance to visual cues, whereas rats attach greater importance to olfactory and gustatory cues, the two species ultimately learned different things despite having been exposed to the same stimulus and the same illness.

Selection is even responsible for *how much learning* is involved in a given outcome (e.g., Alcock, 2013; Frankenhuis and Panchanathan, 2011; Symons, 1979). For example, when a species faces a problem predictably and the problem is invariant, selection minimizes the amount of learning necessary for members of that species to solve that problem. By contrast, when a species faces an unpredictable and changing problem, or one that is complex enough that information acquisition during an individual organism's lifetime is necessary, selection crafts a solution to the problem that requires learning (Symons, 1979). Additionally, animals sample their environments more (i.e., they spend more time learning) when their Bayesian priors are less informative and when environmental cues are moderately informative. When environmental cues are either minimally or very highly informative, organisms derive less benefit from extensive environmental learning (Frankenhuis and Panchanathan, 2011; see also Fenneman and Frankenhuis, 2020).

In sum, evolution and learning are not in explanatory conflict with one another for three reasons: (a) selection crafts evolved learning mechanisms, (b) different species learn different things because they are equipped with different evolved learning mechanisms, and (c) depending on environmental pressures, selection produces psychological solutions that vary in the amount of learning they require. Additionally, the two explanations are not even at the same level of analysis: learning is at the proximate level, whereas evolution is at the ultimate level (Tinbergen, 1963). To understand how and why organisms learn what they do, we must understand the nature of their evolved learning mechanisms and the selection pressures that crafted them (Lorenz, 1973; Symons, 1979).

Exaptations are a class of adaptations

Many readers are undoubtedly familiar with the term 'exaptations', introduced by Stephen Jay Gould (Gould, 1991; see also Gould and Lewontin, 1979; Gould and Vrba, 1982). Although Gould used the word exaptation in different, sometimes conflicting ways (Buss et al., 1998), an exaptation is a biological feature that either (1) first served one function, or (2) served no function at all, and was later co-opted by natural selection to serve a new function. In other words, an exaptation is a former byproduct (we discuss byproducts later in the chapter) or a former adaptation for X, which, under new selection pressures, acquired a new function and became an adaptation for Y. For example, evidence suggests that feathers may have initially evolved for thermoregulation, and only later acquired the function of flight (Ostrom, 1974, 1979). Another example might be the parental bonding system, which may have later become co-opted for adult pair bonding (Shaver et al., 1988).

Exaptations are sometimes discussed as if they pose a threat to the importance of natural selection or the usefulness of adaptation-based hypotheses. They do not. Exaptations are a normal consequence of the way natural selection works. In fact, most, if not all, adaptations are exaptations: they have gone through many changes in their features and their functions throughout the millennia in arriving at their current state (Darwin, 1859; Dennett, 1996). Since selection is a tinkerer, not an engineer (Jacob, 1977), many adaptations likely had a different function (and different features) at some point in the past, making them exaptations. In Dennett's words, 'every adaptation is one sort of exaptation or

the other – this is trivial, since no function is eternal; if you go back far enough, you will find that every adaptation has developed out of predecessor structures each of which either had some other use or no use at all' (Dennett, 1996: 281). In short, exaptations are an important product of the evolutionary process. However, they are a subclass of adaptations, not a mysterious product that falls outside the traditional classification of adaptations, byproducts, and noise.

Hypotheses About Adaptations Are Testable

Psychologists who are not deeply familiar with evolutionary psychology have sometimes alleged that adaptationist hypotheses are inherently untestable. By contrast, this statement is demonstrably false to those who have even a passing familiarity with evolutionary psychology (for discussion, see Al-Shawaf et al., 2018b; Buss et al., 1998; Haig and Durrant, 2000; Ketelaar and Ellis, 2000; Lewis et al., 2017; Sell et al., 2002). What is responsible for the discrepancy between these views?

Adaptationist hypotheses may be informed *by the past, but they yield* predictions *about the present day*

One possible reason for the discrepancy has to do with the historical element implicit in any evolutionary hypothesis. Some writers have correctly noted that (1) we do not have complete knowledge of the conditions present during our ancestral past as hunter-gatherers, (2) evolutionary psychological hypotheses necessarily contain an implicit historical element, and (3) cognition and behavior do not fossilize. Together, these three premises are thought to yield the conclusion that evolutionary psychological hypotheses about adaptations are untestable. But this conclusion does not follow from the premises, for the following reason.

Adaptationist hypotheses are informed by what we know of our past, but *they yield predictions about the present day.* To test them, we do not need to travel to the past, nor do we need to have perfect and complete knowledge of the past – we simply need to test the predictions they yield about how modern humans will behave in the present (Al-Shawaf et al., 2018b). The key point is that while there is a historical component in *generating* adaptation-based hypotheses in the first place, there is no historical component in the *predictions* they yield. And crucially, those predictions can be tested in the immediate present.

In other words, as long as evolutionary hypotheses yield predictions that can be tested in the present, they are eminently testable. However, one might reasonably ask whether we know enough about our ancestral past to generate these hypotheses in the first place.

Do we know enough about our ancestral past to generate hypotheses?

Our knowledge about ancestral humans is limited, but we know with certainty that

> our ancestors, like other Old World primates, nursed; had two sexes; chose mates; had color vision calibrated to the spectral properties of sunlight; lived in a biotic environment with predatory cats, venomous snakes, and spiders; were predated on; bled when wounded; were incapacitated from injuries; were vulnerable to a large variety of parasites and pathogens; and had deleterious recessives rendering them subject to inbreeding depression if they mated with siblings. (Tooby and Cosmides, 2005: 23–4)

Each of these seemingly obvious, quotidian observations can be used to generate novel hypotheses about human psychology and behavior. The observation about deleterious recessives has led to new findings about the psychological mechanisms that govern incest avoidance (Lieberman et al., 2007). The observation about parasites and pathogens has spawned dozens of studies yielding a host of interesting new findings about the

link between disgust and food neophobia, mating, pregnancy, and immune function, among others (e.g., Al-Shawaf et al., 2015c; Al-Shawaf et al., 2018a; Curtis et al., 2011; Fessler et al., 2005). The fact about predatory cats, snakes, and spiders has led to numerous hypotheses and findings about our fear mechanisms, including discoveries of biased learning mechanisms geared toward predator avoidance in children (Barrett, 2015; Barrett and Broesh, 2012). Very basic knowledge of features of our ancestral environment, together with a consideration of the cost asymmetries involved in the different kinds of error one can make when facing a decision, has led to novel findings about human visual perception (Jackson and Cormack, 2007), auditory perception (Neuhoff, 1998, 2001), and social cognition (Haselton and Buss, 2000; Haselton and Nettle, 2006) – and there are many more examples. The key point is this: (a) although it is true that our knowledge of ancestral humans is incomplete, we actually know more than many initially realize, (b) mundane and obvious facts, like the (single) fact that we are susceptible to pathogenic infection, can lead to (dozens of) hypotheses, and (c) these hypotheses yield empirical predictions that can be tested in the modern day (Al-Shawaf et al., 2018b; Lewis et al., 2017). Because of this, we can indeed use evolutionary reasoning to generate predictions that can be tested in the present day.

Are adaptationist hypotheses 'just-so stories', evaluated on the basis of plausibility alone?

It has been famously claimed that evolutionary psychological hypotheses about adaptations are evaluated on the basis of plausibility alone (Gould and Lewontin, 1979: 581). The empirical evidence, however, shows that this is false (Alcock, 2001, 2018a; Lewis et al., 2017) and relies on inaccuracies and misrepresentations of how adaptationist hypotheses are evaluated (Alcock, 2018b; Borgia, 1994; Tooby and Cosmides, 1997). Hypotheses about psychological adaptations are never evaluated on the basis of plausibility alone. Rather, they – like all psychological hypotheses – are evaluated based on the cumulative body of the evidence (Al-Shawaf et al., 2015a; Alcock, 2018b; Lewis et al., 2017). Good discussions of this issue already exist in the literature, so rather than repeating those points, we direct the reader to these papers for fuller discussion: Al-Shawaf et al., 2018b; Confer et al., 2010; Ketelaar and Ellis (2000); and Lewis et al. (2017).

Most evolutionary psychologists argue that adaptations are specialized problem-solving machines, and that specific evidentiary criteria are required to invoke adaptation (Williams, 1966, 1985). The central evidentiary criterion is that of *special design* or *improbable utility*: a hypothesized adaptation must appear so well-designed to solve a particular adaptive problem that it is exceedingly unlikely to have arisen by chance (Williams, 1966). The reason this is considered the key evidentiary criterion is that every adaptation hypothesis is, at its core, a probability statement that a collection of parts or features is so well-designed for a particular function that it must have been crafted by selection rather than having emerged as a byproduct of another adaptation or arisen by chance (e.g., Tooby and Cosmides, 1990a, 1992). Consequently, demonstrating adaptation requires evidence of special design or improbable utility (Tooby and Cosmides, 1992; Williams, 1966).

To establish that something is an adaptation, researchers must demonstrate evidence of special design or improbable utility

This reasoning suggests that *special design* is the gold standard for assessing whether or not a feature is an adaptation. The umbrella criterion of special design includes the more specific subcriteria of economy, efficiency, precision, complexity, and reliability. Although these terms were not explicitly

defined in their original presentation, we might consider the following acceptable working definitions: economy refers to how economically the mechanism works (e.g., without excessive cost to the organism), efficiency refers to how well the adaptation solves the problem, precision refers to how specifically or precisely matched the mechanism is to the adaptive problem, complexity refers to how many component parts the hypothesized adaptation has, and reliability refers to how predictably the proposed adaptation develops and how predictably it solves the problem in question. Some of these conditions are not individually necessary – for example, some adaptations can be (relatively) simple, so complexity is not a necessary condition. Nonetheless, the more a psychological mechanism meets this set of criteria, the more likely it is to be an adaptation. At present, we know of no formal, quantitative operationalizations of these terms – this may represent a challenging but useful direction for future theoretical work.

Evidence of functional design can come from numerous sources (e.g. cross-species, cross-cultural, experimental), and take numerous forms (e.g., psychological, physiological, behavioral). Psychological and behavioral scientists interested in assessing whether or not a certain feature meets the evidentiary criteria for adaptation can therefore use numerous sources and types of evidence to do so (for more detail on types of evidence, ruling out alternative explanations, and cautionary notes, see Lewis et al., 2017).

What kind of evidence is *not* needed to establish that something is an adaptation?

It may also be useful to consider what is *not* needed to demonstrate adaptation. Three common misconceptions are that researchers must (1) discover the specific genes underlying the trait in question, (2) show that the trait in question is present at birth, and (3) show that the trait is currently adaptive. These criteria are misplaced for the following reasons.

Misconception 1: Researchers must discover the genes underlying the trait in question. All adaptations have a genetic basis, but this does not mean that it is necessary to pinpoint the specific genes underlying an adaptation in order to show that it is an adaptation (Lewis et al., 2017). Analogously, all adaptations are underlain by brain mechanisms, but nobody argues that we need to identify the specific brain areas or pathways subserving a trait in order to show that that trait is an adaptation. Discovering the specific brain areas involved is necessary if our goal is to develop a complete science of psychology and a comprehensive understanding of the adaptation in question. But if our goal is to demonstrate that something is an adaptation, identifying the specific brain areas involved will not accomplish that goal. Similarly, all adaptations are underlain by genes, and a comprehensive understanding of an adaptation must include knowledge of the genes that underlie it, but it does not follow from this that you must identify which genes are involved in order to show that it is an adaptation in the first place.

Despite this key caveat, molecular genetic evidence can be important in showing that certain alleles have undergone positive selection, thereby pointing to the presence of a possible adaptation (e.g., Barrett and Schluter, 2008; Johnson and Voight, 2018). However, because many genes have multiple different phenotypic effects (pleiotropic effects; e.g., Williams, 1957), this kind of evidence often leaves open the question of *why* these alleles were selected (which phenotypic effect they were selected for). This suggests that molecular genetic evidence can be helpful, but not definitive, in pointing toward the specific function of a hypothesized adaptation.

Identifying the genes underlying a hypothesized adaptation is also important because an understanding of the relevant genetics contributes to the two proximate levels of Tinbergen's 'four questions' (ontogeny and mechanism; Tinbergen, 1963).

For the above reasons, identifying the genes underlying a hypothesized adaptation

is important – and is key to a comprehensive understanding of the adaptation in question – but is not necessary to establish *that a feature is an adaptation in the first place*. Only evidence of special design or improbable utility can speak to the issue of whether a feature is an adaptation in the first place (e.g., Lewis et al., 2017; Tooby and Cosmides, 1992; Williams, 1966).

Misconception 2: Researchers must show that the feature in question is present at birth. As we noted above, adaptations often emerge during the ontogenetic phase in which they are needed. 'Presence at birth' is therefore not an appropriate evidentiary criterion for evaluating whether a feature is an adaptation. Accordingly, one does not need to show that a feature is present at birth, or that it develops very early in an organism's life, in order to demonstrate that it is an adaptation. For the same reason, evidence that a trait emerges later in life, or involves learning, does not constitute evidence against the trait being an adaptation (e.g., Al-Shawaf et al., 2018b; Al-Shawaf et al., 2019; Symons, 1979).

Misconception 3: Researchers must show that the feature in question is currently adaptive (currently correlated with fitness). It is commonly thought that adaptations should be currently linked with reproductive success. At first glance, this seems correct. And if it were, then a key method of testing whether or not a feature is an adaptation would be measuring actual reproductive outcomes – such as how many offspring are sired by individuals with and without the trait.

This seems intuitive, but in fact this kind of evidence – evidence of current reproductive success – tells us only about *current adaptiveness*, not about whether a feature is an *adaptation* (Symons, 1990, 1992; Tooby and Cosmides, 1990a). Whether a feature is *currently* adaptive is interesting and important information. However, it does not address the issue of whether that feature is an adaptation because (a) adaptations can be currently maladaptive (e.g., Del Giudice, 2018), (b) non-adaptations can be currently adaptive, and (c) the central question about adaptation is not one of current utility – it is one of whether the feature's structure exhibits an improbably good fit with the structure of the adaptive problem it purportedly evolved to solve (Symons, 1990, 1992; Tooby and Cosmides, 1990a).

In other words, *adaptation* and *current adaptiveness* can be decoupled in both theory and practice. If researchers want to determine whether something is an adaptation, the best way to do this is to conduct an engineering analysis of its constituent elements to see if they exhibit evidence of special design for solving a particular adaptive problem. Whether they happen to be currently linked with reproductive success is interesting, but not, strictly speaking, relevant to the question of whether the trait is an adaptation. As anthropologist Donald Symons once argued, if you want to demonstrate that vision is an adaptation, the appropriate test is not to check whether people with 20/20 vision have more offspring than people with 19/20 vision – for one thing, many other factors which have nothing to do with vision affect a complex outcome like number of offspring, making it hard to isolate the contributions of vision. Instead, a more theoretically grounded approach would be to analyze the machinery of the visual system to determine whether it fits the statistical structure of the problem it supposedly evolved to solve – whether its component parts exhibit such economy, reliability, efficiency, and complexity that it is exceedingly unlikely to have gotten that way without having been crafted by selection (Symons, 1989).

For these reasons, pinpointing specific genes, presence at birth, and current adaptiveness are not appropriate evidentiary standards for invoking adaptation. Thankfully, a theoretically grounded alternative set of standards exists in the notion of special design or improbable utility (Symons, 1990; Williams, 1966). This set of criteria, along with Williams' (1966) helpful admonition that researchers should not infer the presence of adaptation if the phenomenon in question can be explained by lower-level laws of physics or chemistry, provides a useful set of constraints

for researchers wondering when it is appropriate to make claims about adaptation. Interested readers may wish to consult resources that discuss these ideas at greater length, including Williams' classic *Adaptation and Natural Selection* (1966); Tooby and Cosmides' 'The Psychological Foundations of Culture' (1992) and 'The Past Explains the Present' (1990a); Steven Pinker's foreword in the *Handbook of Evolutionary Psychology* (2015[2005]); Donald Symons' 'Adaptiveness and Adaptation' (1990), 'A Critique of Darwinian Anthropology' (1989), and 'On the Use and Misuse of Darwinism in the Study of Human Behavior' (1992); as well as a recent practically oriented guide ('Evolutionary Psychology: A How-To Guide'; Lewis et al., 2017) that discusses how to translate these ideas into a rigorous empirical research program.

BYPRODUCTS

Byproducts, as the name suggests, are concomitants or side effects of adaptations. Unlike adaptations, byproducts did not evolve to solve an adaptive problem and do not have a biological function (e.g., Tooby and Cosmides, 1992). They exist because they are coupled with adaptations, not because they helped solve a problem of survival or reproduction themselves (Williams, 1966). They can arise because of pleiotropy (genes with multiple effects), genetic linkage, as a side effect of developmental mechanisms, or because adaptations can have effects other than their proper evolved functions (Andrews et al., 2002; Barclay and van Vugt, 2015).

For example, reading and writing are byproducts of evolved adaptations for speaking and understanding language. This is likely why humans everywhere learn to speak and understand language with little formal instruction as long as they are exposed to it, whereas tasks such as reading and writing require long hours of formal instruction and sustained effort (Pinker, 1997).

In order to identify a byproduct, one ideally has to identify which adaptation(s) it is a byproduct of, as well as why it is coupled with that adaptation – an issue to which we return later in this section.

Morphological and Physiological Byproducts

The redness of blood, the whiteness of bones, and male nipples are morphological or physiological byproducts. None of these features has a specific function or evolved to solve an adaptive problem. Rather, the redness of blood is a byproduct of the iron in the blood's hemoglobin – the hemoglobin serves an oxygen-carrying function, but the redness itself serves no function and is merely a side effect of the hemoglobin (e.g., Symons, 1995). The whiteness of bones is also a byproduct – their color does not offer any survival or reproductive advantage. Rather, this whiteness is a side effect of the calcium that fortifies them and protects them against breakage (Symons, 1995); in other words, the incorporation of calcium into our endoskeleton is an adaptation, but the color is a byproduct. Male nipples are thought to be developmental byproducts of female nipples, for whom they do serve a function (Symons, 1979). In other words, nipples have a biological function in females, but males may only have them because they are byproducts of a shared developmental pathway with females (Symons, 1979).

Psychological and Behavioral Byproducts

Men have significantly more paraphilias (sexual fetishes) than women (Laws and Marshall, 1990; O'Donohue and Plaud, 1994). Researchers (e.g., Al-Shawaf et al., 2015a) have argued that this higher incidence of fetishism among men is likely a functionless byproduct of (a) males' easier-to-cross thresholds of sexual arousal combined with

(b) biased sexual learning mechanisms. In other words, as in many mammals, human males have lower thresholds for sexual arousal and display a greater eagerness for casual sex than females (e.g., Buss, 2018; Lippa, 2009). Men are also more likely to *learn sexual responses to non-sexual stimuli* – for example, after many pairings of erotic stimuli with non-erotic items such as colored squares or women's boots, some men will exhibit a sexual response to the colored squares or the boots alone (McConaghy, 1974; Rachman and Hodgson, 1968). Male rats and Japanese quail show similar effects of sexual conditioning, and these effects are typically weaker or absent in females (Crawford et al., 1993; Pfaus et al., 2001).

Racial prejudice may be another example of an evolutionary byproduct. Given the continuous nature of human variation and the presumably restricted range of ancestral humans, it is unlikely that our ancestors encountered individuals that belonged to a different 'race' than their own (Kurzban et al., 2001; Lewis et al., 2017). And yet, despite this fact – and the corollary that therefore there could *not* have been selection for categorization along the lines of race – racial prejudice and xenophobia are among humans' most damaging proclivities. Why is this? Kurzban and colleagues (2001) provide a byproduct-based answer to this puzzle. They suggest that racial categorization arises as a byproduct of evolved learning mechanisms. These proposed mechanisms evolved to attend to the local environment for cues that are statistically associated with patterns of cooperation and conflict in order to categorize people by group membership. Adaptations for tracking group membership would have served multiple useful functions for ancestral humans, including facilitating cooperation for tasks such as coalitional hunting, social alliances, and intergroup warfare. Kurzban and colleagues suggest – and provide evidence that – these adaptations for tracking group membership can be 'tricked' into categorizing people along racial lines even though they did not evolve to respond to race per se (Kurzban et al., 2001). This line of research also suggests that race can be 'erased', meaning you can prevent this kind of 'mistaken' categorization by providing other salient cues for the coalition-tracking mechanisms to use instead (Kurzban et al., 2001; Navarrete et al., 2010). This example of byproduct research highlights the conceptual and practical utility of differentiating adaptations from byproducts.

Another example of byproducts comes from Belyaev and Trut's seminal fox domestication experiment. Over the course of several decades beginning in 1959 and continuing to this day, Belyaev and Trut attempted to domesticate foxes (*Vulpes vulpes*) by artificially selecting for tameness. Despite *only* selecting for tameness, within a few generations, their foxes exhibited several other new traits: floppy ears, piebald coloration, shorter tails and legs, altered head shape, and other new characteristics. These traits emerged as byproducts of selection for tameness – they were not directly selected for, but arose as hormonal and developmental side effects of selection for tameness (Dugatkin and Trut, 2018; Trut, 1999).

A final prominent example of byproduct work centers on why humans believe in supernatural entities. The most widely endorsed position is a byproduct view: humans do not have adaptations for religious belief, but rather are equipped with adaptations that produce belief in gods and invisible entities as an incidental side effect (Atran and Norenzayan, 2004; Boyer, 2001). According to this view, religious and supernatural belief are evolutionary byproducts of adaptations such as agency-detection mechanisms that are biased toward false positives (Atran and Norenzayan, 2004), theory of mind mechanisms (Willard and Norenzayan, 2013), and the parent-child attachment system (Kirkpatrick, 2009). Other researchers disagree, arguing that the human mind is equipped with adaptations specifically designed to produce religious belief (e.g., Alcorta and Sosis, 2005; Bering and

Johnson, 2005). Our purpose here is not to adjudicate between the different hypotheses on this issue, but rather to highlight a prominent empirical program based on byproduct hypotheses, and to emphasize the kinds of evidence a byproduct claim requires. We discuss the latter issue later in this chapter.

Obligate vs. Facultative Byproducts

The *obligate vs. facultative* distinction is frequently applied to adaptations (see Schmitt, 2015). It may be useful to apply the distinction to byproducts as well. Many morphological and physiological byproducts are obligate; that is, they are an unavoidable side effect of the adaptations to which they are coupled. The whiteness of bone, the redness of blood, and the bellybutton are all like this – they are inextricably coupled with their respective adaptations. By contrast, many psychological examples of byproducts are facultative; that is, they are *potential* side effects of the adaptations that produce them, but are not necessarily coupled with their adaptations. Reading, writing, and discriminating along racial lines are like this – they are likely side effects of adaptations that evolved for other purposes, but those adaptations are not guaranteed to produce them.

This distinction has led some to argue that the adaptation-byproduct distinction may not be useful in psychology, or that a different distinction may be required (Park, 2007). These discussions have been useful, but we suggest that the dispute is more terminological than conceptual. For example, based on the above reasoning that morphological byproducts tend to be inextricably coupled with adaptations, but many psychological ones are not, Park (2007) suggests that we should instead conceptualize the issue in terms of *adaptive vs. non-adaptive* effects of adaptations. By this, he means something similar to the distinction in biology between *proper function* and *effect* (the 'proper function' of an adaptation is the effect it evolved to have, whereas an 'effect' of an adaptation is any other effect it has, even though it did not evolve *because* of that effect). We suggest that in psychology, most byproducts are not inherently coupled with adaptations – they are not guaranteed side effects; they are potential or likely side-effects of adaptations. The adaptation-byproduct distinction can accommodate these psychological byproducts just fine, especially if we conceptualize them as facultative byproducts rather than obligate byproducts. Calling facultative byproducts 'non-adaptive effects' is not quite right because (a) a byproduct can be adaptive or have adaptive effects despite not being an *adaptation* (see below), and (b) an adaptation can have non-adaptive effects – but these non-adaptive effects are not the same as byproducts. We therefore suggest that it would be simpler, and closer to the intended meaning, to borrow the obligate–facultative distinction from adaptations and use it to differentiate between obligate and facultative byproducts whenever the distinction is helpful or illuminating.

Byproducts Can Be Maladaptive

Byproducts did not evolve to solve an adaptive problem, and therefore have no evolved function. They exist merely because they are coupled with adaptations. In the present day, however, byproducts can be adaptive, neutral, or maladaptive. Byproducts have occasionally been defined as having no effect on current fitness (e.g., Buss et al., 1998), but this does not necessarily follow from the fact that they do not have a biological function. Above, we argued that evolutionary thinking requires a firm distinction between adaptation and current adaptiveness. Crucially, this same distinction also applies to byproducts: they should be defined by their history of selection (or lack thereof) and their evolved function (or lack thereof) – not by their current effects on fitness. Consequently, despite not being *adaptations*, byproducts can still

be adaptive or maladaptive – they can still have positive or negative effects on fitness.

For example, sickle cell anemia is a maladaptive byproduct of selection for genes that protect against malaria (Allison, 1954). The allele for sickle cell anemia underwent positive selection because it is adaptive when in a heterozygous state – it protects against malaria. However, in a homozygous state this same allele can lead to sickle cell anemia, a potentially fatal disease (Allison, 1954). Tay Sachs disease provides another example. Tay Sachs is an autosomal recessive neurodegenerative disorder, and it is often fatal. The genes for Tay Sachs seem to persist because they protect against Tuberculosis in the heterozygous state. This state of affairs is called heterozygous advantage, and it highlights how selection can produce byproducts that are actively harmful or maladaptive (Allison, 1954; Al-Shawaf and Zreik, 2018; Nesse and Williams, 1994).

Some evidence suggests that there may be psychological analogues in conditions like schizophrenia and bipolar disorder (Del Giudice, 2018). Genes that give rise to schizophrenia or bipolar disorder may be selected due to their beneficial effects in low doses, but if too many of these alleles are present in a single body, they can lead to maladaptive effects that fall outside the normal range of variation for that trait (Del Giudice, 2018; Nesse, 2004). This phenomenon is sometimes known as a cliff-edge effect (Nesse, 2004, 2009; Vercken et al., 2012). There are many other potential causes of schizophrenia, including pathogenic infection, other exacerbating environmental effects, and intragenomic conflict between maternally and paternally imprinted genes (Nesse, 2015). Our point is not to provide a definitive explanation for the etiology of schizophrenia; rather, we wish to highlight the idea that positively selected genes can lead to maladaptive byproducts if those genes (in combination with environmental stressors) give rise to maladaptive levels of a trait that would have been adaptive at lower levels (Nesse, 2004). The broader point is this: even though byproducts did not evolve to solve an adaptive problem, this does not mean that they must have zero effect on fitness – they may indeed be neutral, but they can also be adaptive or maladaptive.

Differentiating Byproducts from Adaptations: Evidentiary Criteria

What kind of evidence is needed to establish that a feature is a byproduct?

The key test of whether a feature is an adaptation or a byproduct centers on whether that feature exhibits evidence of special design (Symons, 1989, 1990; Williams, 1966; see Lewis et al., 2017, for more on how to generate, test, and interpret byproduct hypotheses). Researchers who wish to distinguish between adaptations and byproducts should therefore look for evidence of improbable utility in solving an adaptive problem – something only adaptations will evince. However, a few secondary considerations may be relevant. First, in the case of facultative byproducts like reading, it can be instructive to note how reliably developing the feature is and/or how much formal training is required to acquire it. We noted earlier that spoken language (adaptation) develops more reliably and with less instruction than written language (byproduct). This criterion is only applicable in some cases, however, as obligate byproducts (like bellybuttons) will typically exhibit the same developmental patterns and universality as adaptations. Second, in the case of developmental byproducts such as male nipples, researchers have suggested that the byproduct may appear smaller in size, less complex, more variable, lacking specific features, or vestigial, especially relative to the sex for whom the feature is an adaptation (females; e.g., Puts and Dawood, 2006). In cases like this, it can be valuable to show empirically that there *is* a common genetic or developmental pathway shared by both male

and female nipples. A third reasonable consideration in distinguishing between adaptations and byproducts is whether a trait or feature could *possibly* have been selected for in ancestral environments. For example, Kurzban and colleagues (2001) argued on theoretical grounds that it would have been difficult or impossible for racial prejudice to have been selected for in ancestral environments due to the continuous nature of human variation and the limited dispersal of ancestral humans. These kinds of considerations about evolvability constraints can help to differentiate between adaptations and byproducts by providing an *a priori* argument for the (im)plausibility of past selection for a particular trait. We expand on the kind of evidence required to test byproduct claims below.

Byproduct claims require just as much evidence as claims of adaptation – and possibly more

There is a common misconception that if you cannot demonstrate that something is an adaptation, you should automatically infer that it is a byproduct (Alcock, 1998; Buss et al., 1998). This reasoning is tempting, but inappropriate. While it is true that adaptation is a special and onerous concept whose invocation requires that key evidentiary criteria be met, this does not mean that a byproduct is a quick-and-easy null hypothesis for which no evidence is required. Just like claims of adaptation, byproduct claims must meet their own evidentiary standards.

Ideally, to establish that a feature is a byproduct, it is necessary to accomplish three things: (1) identify which adaptations it is supposedly coupled with, (2) articulate why, and provide evidence that, the byproduct accompanies those adaptations, and (3) demonstrate that it does not appear to exhibit evidence of special design for solving an adaptive problem (see Kurzban et al., 2001, for an example of this). This means that establishing that a feature is a byproduct typically requires the same amount of evidence as establishing that a feature is an adaptation – or possibly more, in the sense that a clear byproduct demonstration requires demonstrating the adaptation that supposedly produces the byproduct *plus* additional pieces of evidence (Andrews et al., 2002; Buss et al., 1998; Goetz and Shackelford, 2006; Tooby and Cosmides, 1992). If studies fail to reveal conclusive evidence for either the byproduct or adaptation hypotheses, it is best not to automatically infer that the feature in question is a byproduct, but rather remain agnostic until further data are available to adjudicate between competing hypotheses.

NOISE

In addition to adaptations and byproducts, evolution also produces noise – sometimes referred to as 'random effects' (e.g., Krasnow and Truxaw, 2017; Tooby and Cosmides, 1992). Noise can be thought of as variation in the structure and function of adaptations or byproducts that was not selected for and did not evolve for any specific reason (Tooby and Cosmides, 1992). It can be genetically or environmentally driven. Examples of noise may include whether one's bellybutton is an 'innie' or an 'outie', whether one's earlobes are attached or unattached, or how noisily one drinks. Here, we delineate two different kinds of noise produced by evolution: selectively neutral noise generated by mutation and subsequently not winnowed out by selection, and deleterious noise maintained by mutation–selection balance.

Types of Noise

Selectively neutral noise

Selectively neutral noise refers to quantitative variation in the minor details of an adaptation or byproduct that has no effect on fitness or is unlinked to the adaptive aspects of design features (Buss et al., 1998; Goetz

and Shackelford, 2006). For example, the exact size and shape of one's bellybutton, whether it's an 'innie' or an 'outie', and how fast one's fingernails grow could qualify as selectively neutral noise if they have no effect on fitness and did not evolve to solve an adaptive problem. Neutral noise, then, does not show evidence of special design, did not evolve to solve an adaptive problem, and was not filtered out by selection because it has (or more properly, *had*) no effects on fitness.

Deleterious noise

Some forms of noise do have an impact on fitness. The process of mutation constantly introduces new genetic variants into a population. Although selection works to winnow out the deleterious variants, mutation keeps introducing new variants into the population without cease. Consequently, selection is sometimes unable to completely expunge these deleterious genetic variants before new ones arise (e.g., Bergstrom and Dugatkin, 2012). This process, called mutation–selection balance, is thought to partly explain variation in intelligence (Penke et al., 2007), as well as certain disorders such as familial adenomatous polyposis (Bergstrom and Dugatkin, 2012).

This kind of variation (detrimental heritable variation maintained by mutation–selection balance) does not solve an adaptive problem, does not have a biological function, and does not show evidence of special design. We therefore suggest this qualifies as a form of noise. Although some scholars have characterized noise as necessarily having no effect on fitness (e.g., Shackelford and Liddle, 2014), the same distinction between adaptation and adaptiveness introduced earlier and applied to byproducts can also be applied to noise. According to this view, adaptations, byproducts, and noise should all be defined not by their current effects on fitness, but rather by their history of selection (or lack thereof) and evidence of special design (or lack thereof). This reasoning suggests that deleterious genetic variation maintained by mutation–selection balance should be categorized as noise despite its current effects on fitness[1].

Causes of Noise

Noise can have several causes, including mutation, genetic drift, gene flow (also known as migration), and random environmental effects. In other words, noise arises because of the stochastic elements of evolution (Tooby and Cosmides, 1992).

Mutation can introduce noise into a population, and mutation–selection balance can maintain that noise. As discussed above, if the new alleles are neutral with respect to fitness, they will not be winnowed out by selection, thereby providing a way for noise to remain in the population. If, by contrast, they have negative effects on fitness, selection will cull them – but not necessarily at a rate that ensures they completely disappear from the population (e.g., Bergstrom and Dugatkin, 2012).

Noise can also be produced by genetic drift, which is the random, chance-driven change in allele frequency from one generation to the next (e.g., Futuyma and Kirkpatrick, 2017). By chance alone, some features can become more common in a population than others, even though these features were neither selected for or against (Bergstrom and Dugatkin, 2012). Two important subtypes of drift are founder effects and bottlenecks.

Founder effects occur when some subset of a population – say, one that has a disproportionately high percentage of redheads – founds a new colony for reasons unrelated to being a redhead. In this case, the next generation of the new colony will also have a disproportionate number of redheads, despite no positive selection for red-headedness. In this case, genetic drift has led to a change in allele frequency despite no history of selection on the alleles in question.

Bottlenecks occur when, for example, there is an environmental catastrophe and, by chance, some genetic variants survive better

than others, resulting in a post-catastrophe population that exhibits different allele frequencies than the pre-catastrophe population. This is an outcome of chance, not driven by any functional advantage of the alleles that now constitute a greater proportion of the population. For example, imagine that an earthquake causes the death of a large subset of a population, sparing those who happened to be elsewhere at the time. By chance, the group that was elsewhere happens to have an unusually large number of moles on their bodies. If variation in mole prevalence is partly heritable, then the next generation of individuals will have more moles on their bodies than the average individual in the pre-earthquake population – not because the moles were protective against disease or danger, but rather due to the chance effects of who happened to be there when the fatal earthquake struck. This is another process by which genetic drift (in this case, a bottleneck) can lead to noise in a population. These kinds of considerations are important because investigators seeking an adaptive explanation for the increased prevalence of moles in this population will not find one.

Gene flow, sometimes called admixture or migration, can also produce noise. Gene flow refers to the movement or 'flow' of genes from one geographic area to another. For example, if some organisms migrate from one region to another (perhaps in search of mates or food), gene flow has occurred. This kind of migration can lead to noise because the resulting variation between groups may not be adaptively patterned. For example, imagine that relative to those who do not migrate, those who migrate are more likely than average to be double-jointed or have faster-growing nails. This will result in a new population of colonists that has different characteristics than the original population. However, these differences are not produced by adaptation and are not adaptively tied to the ecology of the new location – they are noise. Interpreting these differences as evidence of adaptation would therefore be a mistake.

One caveat here is that it is also possible for gene flow to have an adaptive basis. For example, individuals with higher levels of extraversion and risk-taking may be more likely to migrate than those with lower levels of these traits, in which case the migration is not entirely arbitrary. This may be the case: although the direction of causation is still unclear, studies suggest that nomadic populations have a greater proportion of the 7R allele of the DRD4 gene, which is associated with novelty-seeking (Chen et al., 1999; Eisenberg et al., 2008). Consequently, it is worth keeping in mind that while gene flow can produce evolutionary noise, it is also the case that the patterns of variation produced by gene flow may sometimes reveal an underlying logic that is non-arbitrary.

Finally, noise can be driven by environmental effects such as pathogens, parasites, stochastic developmental effects, and unexpected environmental inputs into evolved mechanisms (e.g., Tooby and Cosmides, 1992). These environmental effects can lead to variation that is not adaptively patterned and does not have a biological function – noise. This does not mean that *all* environmentally-driven variation is noise, however. Much environmentally-driven variation is adaptively patterned, arising from evolved psychological mechanisms responding systematically to environmental inputs. Examples of this kind of environmentally driven, adaptively patterned variation include evoked culture (Gangestad et al., 2006), reactive heritability (Tooby and Cosmides, 1990b; Lukaszewski and Roney, 2011), adaptation to local regional climates and dietary practices (e.g., Fan et al., 2016), and other adaptively patterned individual differences (e.g., Lewis et al., Chapter 6, this volume).

Evidentiary Standards for Invoking Noise

How can researchers distinguish between random noise and adaptively patterned

variation produced by adaptations? The primary criterion is again that of special design: evidence that the variation in question seems arranged to solve an adaptive problem, and whose patterning is so improbably useful that it is exceedingly unlikely to have arisen by chance.

For example, the human disgust system produces effects that are remarkably well-suited to solving the adaptive problem of avoiding infection: it is preferentially triggered by more pathogenic items compared to less pathogenic ones (Curtis et al., 2004), releases pro-inflammatory cytokines (Schaller et al., 2010), produces avoidant motor behaviors (Mortensen et al., 2010), leads to decreases in state extraversion and openness to experience (Mortensen et al., 2010), reduces desire for casual sex (Al-Shawaf et al., 2018a), is downregulated in short-term matters (Al-Shawaf et al., 2015b), responds to the odor of sickness in conspecifics (Olsson et al., 2014), might be upregulated during periods of immunosuppression (Fessler et al., 2005), appears to respond more strongly to groups that are likely to carry unfamiliar pathogens (Faulkner et al., 2004; Makhanova et al., 2015; but see van Leeuwen and Petersen, 2018), and is downregulated when caring for one's kin (Case et al., 2006). This pattern conforms to an engineering analysis of what you might expect from a system specifically designed to reduce the likelihood of infection. This pattern is too improbably useful at solving the problem of avoiding infection to be the result of chance. It is therefore unlikely to be noise, but rather the product of an adaptation (Al-Shawaf et al., 2015a; Curtis et al., 2004).

By contrast, consider variation in features such as the length of one's nails, whether one's earlobe is attached or unattached, or how noisily one drinks water. Variation in these features is presumably selectively neutral, and probably was so over long periods of human evolution as well. Additionally, these features are not obviously tied to solving a particular adaptive problem. There is no compelling reason to believe that a systematic empirical investigation of variation in these traits would uncover adaptive patterning indicative of special design to solve a particular problem. Note that the same lack of adaptive patterning is likely true for *some* of the variation in disgust – even if disgust itself is an evolved adaptation, some proportion of the quantitative variation in the emotion is likely driven by chance and is not characterized by adaptive patterning. Such variation, like variation in earlobe connectedness or how noisily one drinks, is a good candidate for noise.

The key phrase in this analysis is 'good candidate'. These suggestions should be regarded as working hypotheses to be tested, not definite conclusions. As with all questions about adaptations, byproducts, and noise, the above suggestions are empirical questions that can only be settled by testing the patterns of variation in these features and investigating whether they exhibit evidence of special design. Of course, it can be difficult or impossible to answer this question if one doesn't know what adaptive problem to investigate in the first place. For this reason, patterns that initially appear to be noise may later turn out to be the product of an adaptation once a researcher has identified the correct adaptive problem. As is always the case in science, hypotheses and conclusions about noise remain tentative and open to revision (as do hypotheses and conclusions about adaptations and byproducts).

We suggest that noise should be the null hypothesis for researchers testing hypotheses about adaptations – if no special patterning is detected, infer that variation is noise, and only if patterning suggestive of special design is detected, consider the 'special and onerous concept' of adaptation (Williams, 1966: 4). This approach may lead to under-identifying (missing) some adaptations, but of course this can always be overturned by subsequent investigations. Furthermore, because byproduct hypotheses also require that a specific and stringent set of evidentiary criteria be met, noise seems to be the most appropriate choice for a null hypothesis.

CONCLUSION

In sum, the evolutionary process produces three outcomes: adaptations, byproducts, and noise.

Adaptations are morphological, physiological, or psychological mechanisms that evolved to solve an adaptive problem. They are distinguished by evidence of special design – that is, they are patterned in such a way that suggests a tight fit with an adaptive problem. Adaptations do not need to be present at birth, are not genetically determined, and are not in conflict with learning – indeed, learning mechanisms are themselves evolved adaptations.

Adaptations evolved because they served a function, but they do not need to be *currently adaptive.* In fact, they can have *maladaptive* effects for many reasons, including constraints on selection, environmental mismatches, malfunctioning adaptations, interindividual conflict, errors by design, and the on-average nature of selection. Importantly, adaptations are typically universal at the information-processing level, not the behavioral level, so behavioral variation across groups or cultures does not automatically constitute evidence against adaptation. Indeed, evolutionary reasoning often predicts that adaptations will lead to systematically patterned cultural variation in behavior, as is the case with 'evoked culture' (e.g., Al-Shawaf and Lewis, 2017; Gangestad et al., 2006).

By contrast, byproducts exist because they are incidental side effects of adaptations. They did not evolve to solve an adaptive problem and do not have a function. Examples of morphological and physiological byproducts include the whiteness of bones and the redness of blood. Examples of psychological byproducts include reading and writing, discrimination along racial lines, and belief in supernatural beings. Unlike adaptations, byproducts do not evince evidence of special design. Testing byproduct hypotheses requires that one show an apparent lack of special design, identify the adaptations that supposedly lead to the byproduct, and provide evidence that these adaptations actually do lead to the byproduct as an incidental concomitant.

Noise is variation that is not adaptively patterned, does not appear to solve an adaptive problem, and is not a byproduct. Examples of noise likely include whether one's earlobe is attached or unattached, whether one's bellybutton is an innie or an outie, and possibly other inconsequential variation such as how noisily one drinks water or the number of moles on one's body. Some definitions of noise stipulate that noise must be selectively neutral, but we have argued that noise can have effects on fitness and still qualify as noise as long as it meets the above criteria – especially the criteria of not having evolved to solve an adaptive problem and not showing evidence of special design.

Hypotheses about adaptations, byproducts, and noise are testable (Simpson and Campbell, 2015). One way to proceed is using the top-down method, in which researchers generate *a priori* hypotheses on the basis of theory, use these hypotheses to derive specific predictions, and subsequently test these predictions in empirical studies. Another way to proceed is using the bottom-up method, in which researchers observe an interesting phenomenon, generate a hypothesis to explain it, derive new predictions from that hypothesis, and subsequently test these predictions in empirical studies. The 'just-so' charge (also known as post hoc storytelling) falls flat for hypotheses that were generated *a priori* using the top-down approach. By contrast, hypotheses generated using the bottom-up approach can lapse into just-so storytelling, but – as in all sciences – *only* if the researcher stops halfway through the process and decides to believe the hypothesis he or she just concocted without deriving and testing any new predictions emanating from it (Al-Shawaf et al., 2018b). This can happen, but is relatively rare (for discussions of how to rigorously apply evolutionary thinking to psychological research, see Lewis et al., 2017). By contrast, if the researcher derives novel predictions from the newly

concocted hypothesis and conducts empirical tests of these novel predictions, then he or she has proceeded in the normal scientific manner – and the just-so charge falls flat again.

Evolutionary thinking has led to great advances in our understanding of human psychology and behavior over the last 40 years (Al-Shawaf et al., 2018b; Buss, 1995; Confer et al., 2010). The three products of evolution (adaptations, byproducts, and noise) are distinguishable on conceptual and empirical grounds – distinctions that play a key role in explaining known findings and predicting new ones. We hope that this chapter motivates researchers to carefully differentiate these three products of evolution, and contributes in some small way to the light that evolutionary thinking continues to shed on the psychological and behavioral sciences.

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Note

1 For the same reason, random variation that contributes positively to fitness – and thus is likely to undergo selection, but currently has no function – should also qualify as (positive) noise.

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