Chapter 1

Introduction to Evolutionary Psychiatry

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Abstract

This introductory chapter serves multiple purposes. Its primary aim is to introduce psychiatrists and other mental health professionals who are new to Darwinian thinking to some of the basic concepts and terminology of evolutionary science in order to ease their progress through the remaining chapters of this volume. Another aim is to provide a distillation and update of some significant theoretical and other developments in a variety of evolutionary disciplines relevant to psychiatry and psychology that would be of benefit to all readers, including existing evolutionists. Given the constraints of space, there will inevitably be significant omissions. We have elected to cover the basics of standard evolutionary theory, as well as some of the basic principles of evolutionary psychology and medicine. We also briefly survey some of the recent developments in the evolutionary literature on cultural evolution and related fields. We recognise that a balance needs to be struck between covering as wide an area as possible without the chapter becoming a glossary of terms. Readers unfamiliar with specialised evolutionary terms are advised to consult the glossary on the Evolutionary Psychiatry Special Interest Group at the Royal College of Psychiatrists' website: www.epsig.org (click on 'About us' then 'Resources').

Keywords

EEA, evolutionary medicine, evolutionary psychiatry, evolutionary psychology, Tinbergen

Key Points

- Darwinian theory is the organising framework for all life sciences.
- Evolutionary thinking can transform our understanding of causality in medicine and psychiatry through the application of Tinbergen's four questions.
- Without evolution, our understanding of the causes of disease is necessarily incomplete.
- The evolutionary perspective can help us understand human vulnerability to disease and disorder.
- Evolutionary psychiatry complements and augments mainstream psychiatry and does not seek to replace it.
- Evolution can also help us understand human uniqueness and especially the role of cumulative culture and gene–culture co-evolution in shaping the human body and mind.

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1.1 Introduction to Evolutionary Theory

1.1.1 Background

Charles Darwin made two distinct and revolutionary proposals in 1859. The first was that all living organisms shared a common ancestor and the second was that natural selection was the mechanism through which all the diversity of life on Earth arose (Nesse and Stein, 2019).

These insights set in motion one of the greatest scientific revolutions in history. Whereas other major scientific paradigm shifts occurred in the physical sciences (e.g. those of Copernicus, Newton, Einstein and Heisenberg), they had few conspicuous implications outside their specialist fields. Darwinism, however, challenged deeply entrenched assumptions in multiple fields of enquiry and belief, ranging from biology to geology, as well as having profound meta-scientific consequences in its challenge to creationism, essentialism and anthropocentrism

(Mayr, 1971). Yet despite being part of the life sciences, psychiatry (as well as much of medicine) has remained largely pre-Darwinian in its approach. In this book, evolutionary scholars of various disciplines, including psychiatrists, philosophers, anthropologists and psychologists, aim to rectify this, not by rejecting or replacing current mainstream psychiatry, but through the addition of the evolutionary perspective, which should provide the discipline with a more contemporary, sound scientific foundation.

Psychiatry is the branch of medicine that deals with mental disorders that manifest themselves through disturbances in cognition, emotions and behaviour. However, the failure of psychiatry to make significant progress in understanding the aetiology of mental disorders has been described as a 'crisis' by leading evolutionists (Brüne et al., 2012) – a fact that has also been acknowledged in an article in *Science* that stated that there have been no major breakthroughs either in the treatment of schizophrenia for 50 years or in the treatment of depression for 20 years (Akil et al., 2010). Evolutionists would contend that this is partly because mainstream psychiatry focuses exclusively on proximate causation and favours mechanistic explanations of disease and disorder. However, unlike medicine, where human physiology provides clear reference points for normal functioning, psychiatry has attempted to identify disorder and dysfunction without a coherent theory of normal human psychology (Nesse, 2016). Also, even on the rare occasions when the vital questions of function and the role of evolution are considered by mainstream psychiatrists, they stop well short of exploring the full implications of such a radical shift in thinking and approach (e.g. Kendler, 2008). Evolutionary psychiatrists argue that Darwinian theory can serve as the essential, missing basic science for psychiatry (Nesse, 2019).

Psychiatry's pre-Darwinian state may be changing very gradually with the development of evolutionary models for a number of psychiatric disorders and the publication of a number of influential evolutionary psychiatric texts over the past couple of decades (Baron-Cohen, 1997; Brüne, 2015; Del Giudice, 2018; Gilbert and Bailey, 2000; McGuire and Troisi, 1998; Nesse, 2019; Stevens and Price, 2000).

In its development, evolutionary psychiatry has benefited from work in two closely related fields. The first field is evolutionary medicine, which has seen a massive expansion since the publication of Nesse and Williams' (1994) foundational work (preceded by an article by Williams and Nesse (1991) and an American Association for the Advancement of Science symposium on evolutionary medicine in 1993) followed by many others (e.g. Gluckman et al., 2009; Trevathan et al., 2008). The other field is the now highly accomplished and rapidly expanding domain of evolutionary psychology. This was heralded as an academic discipline by the publication of the highly influential *Adapted Mind* (Barkow et al., 1992) followed by the publication of many influential texts and specialised academic journals, as well as the voluminous scientific output of numerous university departments across the Western world. Furthermore, evolutionary anthropologists have had a significant impact on these academic strands, especially on the development of evolutionary medicine (Trevathan et al., 2008).

In this book, we provide reasons as to why evolution is ideally placed to guide psychiatrists in determining what the phenotypic end products of neurobiological systems are (these are the genetically based, behavioural and psychological traits that have been shaped by selection). Importantly, the evolutionary emphasis on function can provide the scientific basis for a non-reductionist expansion of the concept of the biological to encompass the psychological, social and cultural domains (Abed and St John-Smith, 2021). Hence, in contrast with mainstream biological psychiatry's narrow 'decontextualized' view of mental disorder as brain disorder (or brain circuit disorder) (Insel and Cuthbert, 2015), evolutionists consider the environmental context to be of paramount importance in determining the existence and nature of mental disorder (Nesse, 2019).

Thus, evolutionists consider Darwinian theory to be the fundamental organising framework or meta-theory underpinning the whole of the life sciences and not simply one perspective to be considered alongside many others. Evolutionary psychiatry is the application of modern evolutionary theory to the scientific understanding of mental health and disease. The goal of evolutionary psychiatry then is to understand why people get sick as well as how they get sick.

The remainder of this introductory chapter will provide a survey of some of the fundamentals

of evolutionary science relevant to the understanding of health and disease in humans.[1]

1.1.2 Evolution, Natural Selection and Adaptation

What is evolution? Evolution may be defined as any net directional change or any cumulative change in the characteristics of organisms or populations over many generations – in other words, descent with modification. When individuals in a population vary in ways that influence their genetic contribution to future populations, the average characteristics of the population will change.

It is essential to understand that biologists recognise many ways in which evolution can occur, evolution by natural selection being just one of them, although it is often held to be the most important. Other basic evolutionary processes include genetic drift, mutation, migration and sexual and social selection.

Natural selection can lead to speciation, where one species gives rise to a new and distinctly different species. This is one of the processes that drives evolution and helps to explain the diversity of life on Earth. Natural selection is the process through which populations of living organisms adapt and change. Natural selection, however, involves no foresight, planning or goal. Hence, any heritable (genetically based) phenotypic trait that confers a reproductive advantage in competition with alternatives within a population will spread, and given enough time the trait may become fixed as a species-wide characteristic. The measure of reproductive success is referred to as 'fitness'. Repeated cycles of natural selection lead to the preservation of successful variants and the elimination of less successful ones, leading to the appearance of design and the shaping of traits that increase the organism's fitness. These are referred to in the evolutionary literature as 'adaptations'. Although Darwin was unaware of the existence of genes or how variation came about, we now know that variation arises as a result of mutations, which are copying errors in the DNA sequence that occur during cell division (NIH, 2020). When mutations occur in germ-line cells as opposed to somatic cells they can be transmitted to offspring.

The basic Darwinian ideas (variation, inheritance and natural selection) were enhanced in the twentieth century by what was called the 'modern synthesis'. This involved the incorporation of the modern science of genetics, which included the concepts of genes, mutation and Mendelian inheritance, into evolutionary theory.

The modern synthesis led to the insight that while the primary mechanism that generates variation is random (mutations), the success or failure of the different variants depends on the fitness they confer and is not at all random. Thus, natural selection shapes adaptive and functional systems that aid survival and reproduction through favouring certain phenotypic traits over others and leads to the spread of the underlying genes within the population. Nevertheless, the same evolutionary processes that shape functional adaptations, paradoxically and inevitably, produce maladaptations (Brady et al., 2019) as well as vulnerabilities to disease and disorder (Nesse, 2019) (see Box 1.1). However, before tackling the evolutionary causes of the persistence of disease and disorder, we will first explore how evolutionary thinking can transform our understanding of causality followed by a brief discussion of a range of other important evolutionary concepts.

Box 1.1 Evolutionary pathways for the persistence of disease and disorder (adapted from Crespi (2016) and Gluckman et al. (2009))

- Mismatch
- Life history factors
- Overactive defence mechanisms
- Co-evolutionary considerations: consequences of the arms race against pathogens
- Constraints imposed by evolutionary history
- Trade-offs
- Sexual selection and its consequences
- Balancing selection and heterozygote advantage
- Demographic history and its consequences
- Selection favours reproductive success at the expense of health
- Deleterious alleles
- Extremes of adaptations

[1] Readers interested in a more detailed introduction to evolutionary science relevant to medicine may wish to consult *Principles of Evolutionary Medicine* (Gluckman et al., 2009) or later editions.

1.1.3 Tinbergen's Causal Framework

One of the most significant implications of evolutionary theory is in the understanding of causality in the biological sciences. In his seminal paper on the subject, Nikolaas Tinbergen, Nobel Laureate and co-founder of the science of ethology, proposed a causal system that is now known as 'Tinbergen's four questions' (Tinbergen, 1963). Building on the distinction between proximate (mechanistic) and ultimate (evolutionary) causation made by Mayr (1961), Tinbergen proposed that a complete understanding of any biological system, trait or organ requires an understanding of all four categories of its causation (Table 1.1). These are the mechanisms that make it work (physiology, structure), the developmental processes that form the system during the lifetime of the organism, the phylogenetic history of the system and the function that the system served the organism in its natural environment. In Table 1.1, boxes (1) and (2) correspond to the proximate causes and boxes (3) and (4) correspond to the ultimate causes according to Mayr's classification. It is important to note that all four causes apply simultaneously to all biological phenomena and are not alternatives to each other, and that neglecting any of these four causal elements necessarily results in an incomplete understanding of the given system or trait.

As diseases and disorders are phenomena affecting biological systems, they should plainly benefit from the application of Tinbergen's system by asking 'why' questions that supplement the more traditional 'how' questions (see Chapter 2 for a detailed discussion of some clinical applications).

Focusing exclusively on the proximate (as is currently the case in mainstream psychiatry) is akin to a technician's view of a machine, whereas considering ultimate causation as well is more like an engineer's view (Nesse, 2019). Evolutionists consider that a clinician skilled in the recognition of distressing emotional states who also understands why we have such emotions and how emotional systems interact with people's current lives is likely to have a deeper understanding of the patient's distress and is able to take greater account of the circumstances that may be contributing to the patient's current state (Abed and St John-Smith, 2021). In addition, importantly, evolutionary considerations have the potential for influencing research agendas through testing hypotheses regarding what is the normal function of the system that is giving rise to psychopathology; questions that are seldom asked by mainstream psychiatry (Brüne, 2015).

1.1.4 Darwinian Fitness and Inclusive Fitness

Fitness is a central concept in evolutionary theory. Darwinian fitness is a measure of reproductive success and can be defined either with respect to a genotype or to a phenotype in a given environment. This is measured by the average contribution to the gene pool of the next generation that is made by an individual of the specified genotype or phenotype. Where fitness is affected by differences between various alleles of a given gene, the relative frequency of those alleles will change across generations through selection, and alleles with greater positive effects on individual fitness will become more common over time.

As alluded to earlier, the integration of modern genetics with Darwinian theory led to the 'modern synthesis' and the formulation of the concept of 'inclusive fitness' (Hamilton, 1964). According to Hamilton's formulation, fitness should be measured not only through the number of direct descendants who carry copies of one's genes, but also through the number of non-descendant kin who also carry copies of the

Table 1.1 Tinbergen's four questions (adapted from Nesse, 2013)

	Developmental/historical	Characteristics of the trait/system
Proximate causation	(2) Ontogeny: how does the trait develop during the lifetime of the organism?	(1) Mechanism: how does it work?
Evolutionary or ultimate causation	(3) Phylogeny: what is the phylogenetic history of the trait? (*Why* is the trait/system the way it is?)	(4) Adaptive function: how has the trait or system contributed to the organism's inclusive fitness in its natural environment? (*Why* does the trait/system exist?)

same genes. It follows that behaving altruistically towards kin can improve one's overall fitness or inclusive fitness (the sum total of descendant and non-descendant kin who carry copies of one's genes) provided that the fitness cost to the altruist is lower than the fitness gain to kin multiplied by the coefficient of relatedness (this is also known as Hamilton's rule). This provides a basis for the understanding of the evolution of altruism and of the conditions that would give rise to competition and cooperation (Del Giudice, 2018). 'Kin selection' is the term that is used for the evolutionary strategy that increases inclusive fitness through the application of Hamilton's rule.

1.1.5 Evolution and the Concept of Psychological Mechanisms

Natural and sexual selection are the only known causal processes capable of producing complex functional mechanisms (also known as adaptations). An adaptation may be defined as an inherited characteristic that came into existence as a feature of a species through natural selection because it facilitated survival and reproduction during the period of its evolution (Tooby and Cosmides, 1992). Solving a recurrent adaptive problem is the function of any given adaptation. There must be genes for any adaptation because they are axiomatically required for the passage of the adaptation from parents to offspring. Therefore, evolutionary psychologists/psychiatrists start from the position that all brain neurobiological mechanisms/systems have been shaped through a long process of selection within a particular set of environmental conditions (see Section 1.1.8) (Buss, 2009).

Psychological mechanisms are viewed as specialised neurobiological systems shaped by selection to solve recurrent problems of survival and reproduction faced by ancestral humans over evolutionary history (Tooby and Cosmides, 1992). An understanding of the function and phylogeny of evolved mechanisms thereby provides unique insights into both their adaptive output as well as how and why these mechanisms can misfire, leading to maladaptive responses (e.g. in novel environmental conditions; see Section 1.2.1). Examples of evolved psychological mechanisms include: fear, attachment, security, status, mating and caregiving (Del Giudice, 2018).

An illustrative example of the derailing of an evolved mechanism is the way in which the cuckoo chick exploits the innate parental feeding mechanism of certain bird species. The hatching cuckoo chick provides a supernormal stimulus that triggers a (parental) feeding response through its huge gaping beak despite being in the nest of a different species (such as a great reed warbler), which induces the warbler to feed the cuckoo chick to the detriment of its own offspring (e.g. Tanaka et al., 2011). Similarly, evolved psychological mechanisms in humans can be derailed and produce maladaptive responses when exposed to novel environmental conditions, leading to mental disorder in some individuals (see Section 1.2.1).

1.1.6 Parental Investment Theory and Parent–Offspring Conflict

Parental investment is the investment that parents make in an offspring that increases that offspring's chances of surviving. By definition, such investment imposes a cost on the parents as measured by their ability to invest in other offspring, current and future. Components of fitness include the well-being of existing offspring, parents' future reproduction and inclusive fitness through aid to kin (Hamilton, 1964; Trivers, 1972). Parental investment may be performed by both males and females (biparental care), females alone (exclusive maternal care) or males alone (exclusive paternal care). Care can be provided at any stage of the offspring's life, from prenatal (e.g. egg guarding and incubation in birds and placental nourishment in mammals) to postnatal (e.g. food provisioning and protection of offspring).

Parental investment theory predicts that, on average, the sex that invests more in its offspring, including the size of gametes, gestation, lactation and child rearing, will be more selective when choosing a mate, and the less-investing sex will engage in more intra-sexual competition for access to mates. This theory has been influential in explaining sex differences in sexual selection and mate preferences throughout the animal kingdom, including humans. Trivers (1974) extended parental investment theory to explain parent–offspring conflict: the conflict between optimal investment from the parent's versus the offspring's perspective.

A further complication in nurturing occurs with parent–offspring conflict. This is a biological process that can start from the moment of conception. This conflict, which occurs exclusively in sexually reproducing species, is based on the fact that while the mother (or father) is related to their offspring by 50%, the foetus is 100% related to itself. This is used to signify the evolutionary conflict arising from differences in optimal parental investment to an offspring from the standpoint of both the parent and the offspring (Trivers, 1974).

Similarly, each sibling is only 50% related to any of their full siblings, and so they have a propensity to attempt to acquire more than their fair share of parental investment and more than the parents are willing to provide. However, parent–offspring conflict is functionally and statistically counterbalanced by the processes related to inclusive fitness and thus limited by the close genetic relationship between parent and offspring as additional parental investment obtained by one offspring at the expense of its siblings can decrease the number of its surviving siblings and reduce inclusive fitness. This leads to the prediction that, all other things being equal, parent–offspring conflict will be stronger among half-siblings than among full siblings. These observations and models may have significant effects relevant to child psychiatry (see Chapters 14 and 15).

1.1.7 Phenotypic Plasticity, Canalisation and Differential Susceptibility

Plasticity is an evolutionary adaptation to environmental variation that is reasonably predictable and occurs within the lifespan of an individual organism as it allows individuals to 'fit' their phenotype to different environments. Phenotypic plasticity describes the possibility of modifying developmental trajectories in response to specific environmental cues and also the ability of an individual organism to change its phenotypic state or activity (e.g. its metabolism) in response to variations in environmental conditions (Garland and Kelly, 2006).

Phenotypic plasticity can evolve if Darwinian fitness is increased by changing the phenotype. However, the fitness benefits of plasticity may be limited by the trade-off of the costs of plastic responses (e.g. synthesising new proteins, adjusting expression ratios of isozyme variants, maintaining sensory machinery to detect changes) as well as the predictability and reliability of environmental cues. Canalisation is the converse of plasticity and refers to developmental stability that resists both genetic and environmental disruption or perturbation. Canalisation mechanisms are vitally important and ensure that an organism's traits demonstrate robustness and develop reliably. However, their drawback is that they limit plasticity (Haltigan et al., 2021; Waddington, 1942).

Another interpretation of psychological findings that are traditionally discussed according to the diathesis–stress model is differential susceptibility (Belsky, 1997). Both models suggest that development can be differentially susceptible to experiences or qualities of the environment. Whereas the diathesis–stress model suggests a distinct and mostly negativity-sensitive response, Belsky describes a group that is sensitive to adverse experiences but also to positive experiences. These models may be complementary if some individuals are dually or uniquely positivity-sensitive while others are uniquely negativity-sensitive.

Bakermans-Kranenburg and van IJzendoorn (2006) were the first to test the differential susceptibility hypothesis as a function of genetic factors, examining the moderating effect of the dopamine receptor D4 seven-repeat polymorphism (DRD4–7R) on the association between maternal sensitivity and externalising behaviour problems in 47 families. Children with the DRD4–7R allele and 'insensitive mothers' displayed significantly more externalising behaviours than children with the same allele but with 'sensitive' mothers. Children with the DRD4–7R allele and sensitive mothers had the fewest externalising behaviours of all, whereas maternal sensitivity had no effect on children without the DRD4–7R allele.

Research has also demonstrated that possessing at least one s-allele of the serotonin transporter gene *HTTLPR* confers an increased risk of developing depression when facing adverse events. However, the same variation is linked to superior cognitive performance in several domains and increases social conformity (Homberg and Lesch, 2011).

These examples serve as evidence against simple genetic determinism and also provide indications that naïvely aspiring to alter genes alone

in order to treat disorders may not be in an individual's best interest as differing circumstances alter the harmfulness or benefits of such a gene.

1.1.8 The Ancestral Environment or Environment of Evolutionary Adaptedness

The concept of the environment of evolutionary adaptedness (EEA) was first proposed by John Bowlby (1969) of attachment theory fame. Broadly speaking, the EEA refers to the overall ancestral human environment during which the distinctive traits of modern humans were shaped. It is sometimes referred to incorrectly as if it were a single, uniform time and place. However, it is more appropriately conceptualised as 'a statistical composite of the adaptation-relevant properties of the ancestral environments encountered by members of ancestral populations, weighted by their frequency and fitness consequences' (Tooby and Cosmides, 1990: 386–387). The EEA is therefore a compound idea representing the sum of a population's exposure, over a given time frame, to external conditions and stimuli, threats and opportunities, including nutrients, social pressures, threats from parasites, predators and competitors as well as climate and general habitat (Gluckman et al., 2009). Thus, it may be considered as a 'composite of environmental properties of the most recent segment of a species' evolution that encompasses the period during which its modern collection of adaptations assumed their present form' (Tooby and Cosmides, 1990: 388). It is important to note that 'different adaptations will have different EEAs. Some, like language, are firmly anchored in approximately the last two million years; others, such as infant attachment, reflect a much lengthier evolutionary history' (Durrant and Ellis, 2003: 10).

Critics of the concept of the EEA have argued that we do not know much about how our remote ancestors lived, and they claim that this makes the concept of the EEA a highly speculative and unscientific premise (Hagen, 2016). Critics such as Gould (1997), Buller (2005) and Laland and Brown (2011) also objected to the use of the concept of the EEA because they assumed that we are unable to specify the living conditions of our ancestors with sufficient precision. There is no doubt that some of these concerns are legitimate and should be seriously considered. However, if their assertions are true, such that we can never know anything about how our ancestors lived and will never be able to do so, then an evolutionary approach could not ascertain the exact function of any somatic or brain system. As all functions are adaptations shaped by selection in response to past environments, discovering facts about past environments remains an important part of the evolutionary endeavour and a prerequisite to understanding current function and dysfunction.

The assertion that we cannot know much about the past is nowadays no longer tenable and contradicts a wide range of academic disciplines whose focuses are entirely on investigating the past. These fields include archaeology, palaeontology, palaeoanthropology, history and cosmology, which now include not only research into fossils and artefacts, but also sequencing the DNA of ancient and extinct species (Hagen, 2020). This has allowed enormous progress and clearly and decisively demonstrates that scientific research aimed at discovering facts about the past is capable of producing rigorous, testable and falsifiable models of past environments (e.g. Dunbar, 2014). Without knowledge of the past, evolutionary science simply cannot progress, and hence a concept of the EEA or ancestral human environment is essential. This does not mean that statements about human evolutionary history should be accepted blindly or uncritically. All such claims should be stated as hypotheses that can be supported or falsified by the evidence, and a similar level of scientific rigour should also apply to hypotheses about human psychological adaptations and their functions.

1.2 The Evolutionary Pathways for the Persistence of Disease and Disorder

As we have seen, natural selection produces bodies and brains with assortments of adaptations shaped over thousands of generations to enhance reproductive success (fitness) but not necessarily well-being or happiness. The explanation for the conundrum of why evolution has left humans so vulnerable to disease and disorder has itself been evolving ever since it was first posed by the

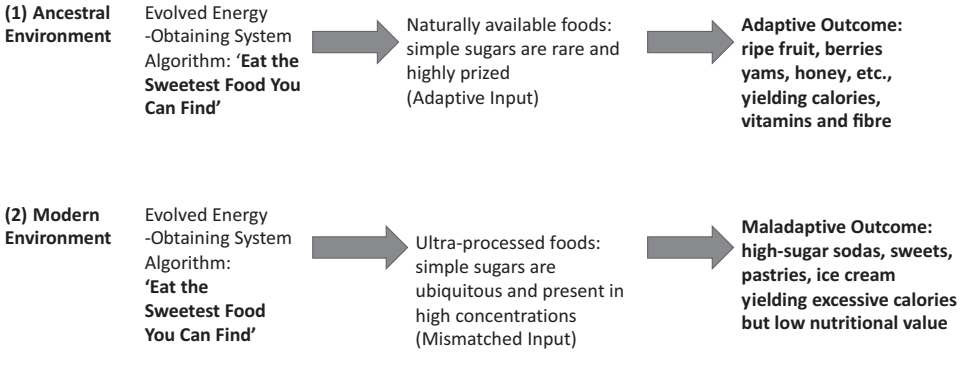

Figure 1.1 Illustration of nutritional mismatch in the modern environment (adapted from Li et al., 2018)

founders of modern evolutionary medicine (Nesse and Williams, 1994). Accordingly, a range of pathways have been proposed by which evolutionary processes can lead to the existence and persistence of disease or disorder, as presented in Box 1.1.

Some of these pathways are more relevant than others to psychiatry, and they are not mutually exclusive. Several may be implicated concurrently or sequentially in the origin of mental disorders. They represent a list of ultimate/evolutionary causes of our vulnerability to disease and disorder, including mental disorder.

1.2.1 Mismatch

Mismatch is arguably one of the most important insights of evolutionary medicine and is indispensable to the understanding of a range of diseases and disorders prevalent in the modern environment, such as the increased prevalence of coronary artery disease, hypertension, obesity, type 2 diabetes, depression, alcoholism and eating disorders, to name a few (Nesse and Williams, 1994; Pollard, 2008). The idea of mismatch is based on the fact that adaptations are shaped by selection within a given environment. If the environment changes rapidly and radically, some biological systems run the risk of becoming mismatched to the new environment. This is also referred to as 'genome lag' (Li et al., 2018). Given that the modern human environment has undergone a radical change from that of our ancestors in a number of ways, this has led to some systems becoming mismatched to this novel environment, giving rise to dysfunctional outcomes, including a range of mental disorders (see Chapter 2 for further discussion). Examples of mental disorders arising/increasing in the modern environment include eating disorders (Rantala, 2019; Russell, 2000) and drug and alcohol addictions (Nesse, 2005) (see Figure 1.1 for an illustration of nutritional mismatch). However, while humans may be mismatched to certain aspects of the modern environment (e.g. the constant abundance of nutrients and especially of ultra-processed foods), we are well matched to the majority of modern conditions, as humans are clearly thriving and not becoming extinct (Hagen, 2020).

1.2.2 Life History Theory

Life history theory (LHT) deals with species' typical solutions to problems associated with survival and reproduction that change over an individual's lifespan (Brüne, 2015). Hence, LHT provides a framework for understanding how organisms allocate time and energy in achieving core biosocial goals across their lifespan. Life history strategies involve a series of trade-offs that shape important biological developments, including the timing of sexual maturity and the number and quality of offspring, as well as the length of lifespan (Stearns, 1992). The application of LHT demonstrates that these trade-offs yield a spectrum of life history strategies, and the trade-offs include somatic versus reproductive effort, present versus the future and quality versus quantity of offspring. The 'fast' end of the spectrum is characterised by a shorter lifespan, faster growth, earlier maturation and reproduction and a larger number of offspring, while those at the 'slow' end of the life history spectrum show the opposite characteristics (Del Giudice, 2018). The idea of a

fast–slow spectrum of life history has been proposed as a framework for understanding individual differences, including vulnerability to mental disorders (Del Giudice, 2018). Differences in life history strategies are partly under genetic control, but it appears that the nature and quality of an individual's early environment may also be important (Belsky et al., 1991; Ellis et al., 2011). The application of LHT to trait variations between individuals as opposed to between species has recently come under critical scrutiny (e.g. Zietsch and Sidari, 2020). As a result, this area of research is undergoing considerable revision regarding both its methodology and its theoretical assumptions (Del Giudice, 2020; Young et al., 2020).

1.2.3 Overactive Defences

Defences such as the mood- and anxiety-regulating systems can become overactive or dysregulated, resulting in harmful outcomes and leading to defence activation disorders (Del Giudice, 2018; Nesse, 2019). Examples of defences in general medicine include pain, diarrhoea, vomiting and pyrexia, for which similar principles apply. Importantly, all defences – whether in biologically evolved or human-made systems – have a common design feature such that they are designed to allow false alarms (also known as false positives), as these are far less costly than failure to activate (false negatives) when the risk is present (usually with catastrophic results; imagine, for example, ingesting a toxin and failing to vomit). This is referred to as the 'smoke detector principle' and explains why all bodily defences (including aversive emotions) can activate excessively (Nesse, 2019). The excessive tendency for false alarms that characterises all defence systems is akin to a strategy of 'better safe than sorry' (Blumstein, 2020) and explains why it is usually safe to block a defence once it is established that the response is not necessary or even counterproductive.

1.2.4 Co-evolutionary Considerations (Arms Races between Pathogens and Hosts)

Humans as hosts have been and continue to be engaged in an unending arms race with rapidly evolving pathogens such as bacteria and viruses (Ewald, 1994). This means that increasingly innovative host defences (e.g. increasingly sophisticated immune responses) are matched by even more novel ways of evading such defences. Also, increasing numbers of pathogens have become resistant to antimicrobial therapy, which poses an increasingly serious hazard to human health. In this arms race, pathogens, as rapid replicators, have the advantage because of their much faster capacity to evolve (Nesse, 2005). The recent Covid-19 pandemic is a vivid example of a newly evolved virus jumping species and spreading globally through the human population, taking a massive toll in terms of human life and livelihoods, and there seems no doubt that there will be other such pandemics in the future. This is undoubtedly a massive problem for medicine in general, but examples in mental health appear more limited. For example, obsessive-compulsive disorder (OCD) can arise as a result of streptococcal-induced autoimmune disease (Swedo et al., 1994), and there have been ongoing suggestions of a link between *Toxoplasma gondii* and schizophrenia (Fuglewicz et al., 2017) (see Chapter 10).

1.2.5 Constraints Arising from Evolutionary History

Unlike a human designer, evolution cannot go back to the drawing board and start afresh. This is called 'path dependency' and explains poor 'designs' such as why human eyes have blood vessels that occlude portions of the retina (Nesse, 2005), why the light receptors in the human retina face the wrong way (Lents, 2020), the tortuous path of the recurrent laryngeal nerve and why our bipedal skeleton – a modified version of a quadrupedal design plan – creates myriad vulnerabilities from ubiquitous back problems to birth canals too narrow to admit a foetal head (Pavličev et al., 2020; Taylor, 2015). It is also why phylogenetic history and the EEA matter so much. Evolution must work with what has gone before; complex systems are not created out of nothing. Evolution has been described as a tinkerer, shaping adaptations (from available biological systems) that work just well enough for survival and reproduction (Jacobs, 1977). Hence, evolution is a process that shapes adaptations through historical constraints, multiple trade-offs and (genetic) errors (Nesse, 2005). Evolutionary

thinking, therefore, explains the flaws, quirks and tortuous complexity that is ubiquitous in biological systems, all of which can create vulnerabilities to dysfunction and disorder.

1.2.6 Trade-Offs

It is necessary to appreciate that all biologically evolved adaptations, traits and systems represent trade-offs, as increasing one trait is often at the expense of worsening the performance of another. For example, increasing resistance to infections increases the risk of autoimmune diseases. Also, this explains why improving energy conservation and famine resistance increases the risk of obesity when food becomes plentiful and why the reciprocal trade-off between body size/muscle bulk and speed of movement has an optimum balance such that increasing one can lead to a decrease in the other.

1.2.7 Sexual Selection and Its Consequences

Sexual selection was described by Darwin (1871) to explain the evolution of traits that do not aid survival and may even be detrimental to it. The canonical example of a sexually selected trait is the peacock's tail, which serves no survival purpose but is an attractor of peahens. Sexually selected traits are those that improve reproductive success through increased attractiveness to the opposite sex. Sexual selection occurs in all sexually reproducing organisms, including humans, and usually involves the display of costly and extravagant traits that are difficult to fake and can therefore act as honest markers of good health and high-quality genes. Sexual selection tends to shape traits that are gender divergent and to specifically target the preferences of the opposite sex. The evolution of sexually selected traits can create particular kinds of vulnerabilities to mental disorders, which are often skewed in their sex ratios. Examples of mental disorders where sexual selection may play an important role include eating disorders (Abed, 1998) (see Chapter 11), sexual dysfunction and schizophrenia (Del Giudice, 2017).

1.2.8 Balancing Selection and Heterozygote Advantage

In diploid species such as humans, the two alleles can be identical (homozygote) or different (heterozygote). The classical example in medicine of a heterozygote advantage is sickle cell anaemia, where the heterozygote state confers immunity to malaria (which is a major advantage in parts of the world where malaria is endemic), whereas the homozygote state causes sickle cell anaemia, a serious and debilitating disease (Gluckman et al., 2009). In this example, the benefits of the heterozygote state are counterbalanced by the deleterious effect of the homozygote state. Other examples of heterozygote advantage in medicine are more speculative (e.g. cystic fibrosis). There are currently no examples of this process relevant to mental health.

1.2.9 Demographic History and Its Consequences

Human migrations out of Africa took place around 70,000 years ago onwards. They took place in successive waves and in doing so human populations frequently passed through impediments or bottlenecks (due to famine, disease, etc.) that caused significantly reduced genetic diversity (Henn et al., 2012). Such scenarios also include small populations that become isolated through chance events and continue living in small, isolated communities where otherwise-rare mutations can become unusually prevalent as a result of a 'founder effect' (Gluckman et al., 2009). Such chance events are also referred to as genetic drift. Examples of rare harmful genes becoming prevalent as a result of the founder effect include Tay–Sachs disease, which exclusively affects Ashkenazi Jews, and Gaucher's disease, which is found disproportionately in French Canadians. Interestingly, recent findings suggest that globally, human populations, in their migration out of Africa, have been subject to serial bottlenecks (and founder effects) that increased the further away they travelled from the African continent, as a result of which genetic diversity successively declined (Henn et al., 2012). This phenomenon may also explain the high prevalence rates of Huntington's disease in Venezuela, Colombia, Peru and Brazil (Kay et al., 2017).

1.2.10 Selection Favours Reproductive Success over Health

The basic tenet of Darwinian theory is that selection works through reproductive success and not