Chapter 1
Applying Evolutionary Thinking in Medicine: An Introduction

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Lay Summary Evolutionary thinking is beginning to infiltrate medical practice and has the potential to transform how clinicians explain human diseases. Evolutionary medicine takes a long-term view of why humans suffer from various diseases and addresses the reasons behind these. Proponents of this relatively new field argue that clinicians need to understand basic concepts in evolutionary biology and that these should be embedded in the training students receive in medical schools. Historically, in the late nineteenth and early twentieth centuries, medical writings did include evolutionary concepts, but this approach fell out of favour following the excesses of the Second World War. Evolutionary medicine emerged again in the 1990s and has slowly been building momentum around the world with journals, societies, books, and papers expanding in number and visibility. Although biologists and other scientists have been the main proponents, a growing number of physicians and medical students are becoming involved as the field reaches a new maturity.

1.1 A Shift in Perspective in Approaching Medical Issues

What is evolutionary thinking in medicine? In brief, it is the application of basic evolutionary principles derived from the science of biology to understand human susceptibility to disease [1–4]. But it is so much more than this! An evolutionary approach to health and diseases addresses how past and present pathogens, with which we now coexist, behave and change over time [5], is concerned with how individual development within specific environmental contexts can shape suscep-

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tibilities over the life course [6–8], considers how major changes in human lifestyle such as urbanisation and industrialisation have altered the epidemiological nature of illnesses that can afflict us [9, 10], takes into account phylogenetic history in relation to our vulnerability to specific conditions such as lower back pain [11], and attempts to understand human lifespan and mortality within a broader context of why ageing should exist at all among species [12], among many other topics [3–18]. More recently, evolutionary medicine is reaching out to embrace veterinary practitioners since the two disciplines have much to learn from each other [19, 20].

1.1.1 Novel Questions

So, what are the basic principles that inform an evolutionary approach to medicine? One of the first distinctions between this and clinical medicine is that the former addresses "ultimate" (i.e. evolutionary) questions about health and disease as opposed to "proximate" (i.e. mechanistic) ones [3]. In this respect, Randy Nesse has frequently referred to one of the great biological thinkers, Nikolaas Tinbergen, who contributed to the development of the field of ethology or animal behaviour. Tinbergen developed a set of four questions dealing with mechanism, ontogeny, phylogeny, and function with which to address evolutionary questions about behaviour [3, 21, 22]. These are suggested as highly useful when comparing the kinds of questions asked by medical doctors as opposed to evolutionary biologists, but both ultimate and proximate questions are viewed as complementary. Tinbergen's first question asks about the "proximate" cause of a trait (similar to the kinds of questions a medical doctor might ask), the second addresses immediate developmental issues, the third deals with the development or evolution of a trait on an ultimate level in comparison with other species and over long evolutionary time, while the fourth addresses issues of adaptation in asking about how the trait might affect reproduction and survival.

An example of proximate (clinical) and ultimate (evolutionary) approaches to medicine can illustrate the difference between the two. If presented with a patient suffering from asthma, a medical doctor would presumably take a case history of the patient and would ask about family susceptibility to the condition, the length of time that the patient had experienced symptoms, the degree of severity of the symptoms, and the potential exposures that might trigger the condition. These exposures might relate to the immediate environment at home and elsewhere where the patient was spending their time. The doctor might consider allergens such as dust mites, household cleaners, pollen, and pets. The patient might be referred to a clinic for allergy testing for reactions to specific substances that could then be ruled out as irritants. In contrast, evolutionary medicine approaches illnesses such as allergies and asthma from a more long-term (the "ultimate") perspective. There is an extensive literature arguing that autoimmune disorders such as allergies (including asthma) have become prevalent within contemporary societies since we became removed from ancestral conditions where we coexisted with several pathogens, including intestinal worms called helminths [23–25]. There is evidence to suggest

that immunoglobulin E (IgE)—which becomes elevated with allergic conditions—in fact coevolved to respond to our earlier and common coexistence with helminths, which were down-regulating the system for their own benefit. In our cleaner and more hygienic environment, in the absence of helminths, the IgE system is dysregulated and responds instead to other foreign bodies resulting in autoimmune disorders such as allergies.

Much of this autoimmune topic is related to the "hygiene hypothesis" (see also Chaps. 15 and 17) where recent conditions of extreme cleanliness are thought to have detrimental consequences for the human immune system [25–27]. There is again growing evidence that humans need to "educate" their immune system during development by exposure to a wide variety of bacteria and other organisms (including helminths). The lack of such exposures might trigger a range of autoimmune disorders. Of course, it could be argued that knowledge of human evolutionary history, and the kinds of environments in which we lived in our past, is irrelevant to the way in which doctors treat their patients. But it is precisely this kind of knowledge that is leading to novel treatments of various autoimmune disorders by re-exposing individuals to what are sometimes called "old friends", meaning precisely helminths [28]. Preclinical trials are now evaluating the safety of infecting patients with more benign forms of helminths, such as pinworms, in an effort to achieve remission for a variety of autoimmune disorders including allergies, irritable bowel disease, and multiple sclerosis [29, 30] (see Chap. 17 for the most recent research in this area). It is doubtful whether these kinds of treatments could have arisen without an understanding of human coevolution with other organisms and how this has shaped the human phenotype. Similar understandings are now leading to an appreciation of how contemporary environments are dramatically changing our gut (and other) microbiomes, leading to novel and sometimes serious disorders [31, 32].

1.1.2 Research Areas, Concepts, and Assumptions

The example of allergies falls into one of the themes ("abnormal environments") that George Williams and Randy Nesse originally conceived in their landmark paper in the *Quarterly Review of Biology*, in 1991 [33], in order to create a structure with which to view a variety of human illnesses. They outlined 5 major areas where they felt that an evolutionary approach could make a contribution towards understanding health and illness. These were (1) infectious diseases (see Chaps. 14–17 and 19), (2) host–parasite coevolution (see Chap. 16), (3) injuries, breakdown, and toxins (Chaps. 10, 18 and 23), (4) genetic effects on diseases, and (5) abnormal environments (Chaps. 4–9 and 11). Randy Nesse was to develop this set of themes further in his later articles. "Abnormal environments" became subsumed under the now more common name of "mismatch", popularised in a book by Gluckman and Hanson in 2006 [9] although this was also to develop a more specific meaning in relation to early life development.

Authors supporting evolutionary approaches to medicine have gone on to argue that medical doctors need to understand the concept of adaptation and natural selection [2–4, 34, 35]. First, they have frequently pointed out that natural selection (which is just one of the forces shaping human evolution) works extremely slowly—on the order of thousands of years [36]. This fact has frequently led to the misunderstanding, even among people trained in evolutionary thinking, that humans have stopped evolving. An exemplar of this kind of misunderstanding is the concept of the "environment of evolutionary adaptedness" (EEA) introduced by the psychologist, John Bowlby, in 1969 [37] and later adopted, par excellence, by the Santa Barbara School of evolutionary psychology [38–39]. The EEA theory posits that humans are essentially adapted to the environment in which we spent thousands of years as Palaeolithic foragers, although the precise date and provenience of this environment, as well as the entire concept, have been heavily debated [40–41]. However, the concept of an EEA has tied in nicely with the theme of "mismatch" or "abnormal environments", where humans are frequently seen to be maladapted to contemporary environments where diets rich in fats and sugars and a highly sedentary lifestyle can lead to chronic conditions such as obesity and metabolic disorders [9, 10]. More recent molecular research that is rapidly expanding has, however, pointed out that humans are undergoing constant microevolution in response to both mutations and changing environments [42–45]. The growing field of epigenetics that examines how molecular markers turn genes on and off, or up and down, is also contributing to our growing understanding of how developmental and life course plasticity can alter the human phenotype [e.g. 46–48].

1.2 Contributions to Biomedicine

1.2.1 Rethinking the Optimal Body

Humans and other organisms have evolved features that are basically "jury-rigged compensations for a fundamentally defective architecture" [36: 63]. As our natural environments altered, causing various features to evolve, including bipedalism, evolution had to work on an existing structural design and to modify this where possible to develop features that would be adapted to a changing environment. Nesse's quote above is in relation to the vertebrate eye which is subject to several potential malfunctions as a result of the legacy of "jury-rigged" design compromises, or what has been called elsewhere "historical legacies" [15]. Nesse refers to myopia, detached retinas, and glaucoma as some examples of how the human eye can fail due to its intrinsic and evolved design [36]. Similarly, humans are vulnerable to a series of back problems not experienced by our quadrupedal relatives as a result of our evolution towards an upright posture [15]. Understanding these kinds of evolutionary constraints and the inevitable "trade-offs" in our physiology can be helpful in considering human vulnerabilities and potential treatments for many ubiquitous problems.

Myopia, or short-sightedness, which seems to be a problem caused through gene–culture (design and environment) interaction or coevolution is another excellent example of human developmental vulnerability and "mismatch". Human cultural evolution has led to children spending many hours indoors, away from sunlight, and in close-up work such as reading or electronic screens [49–51]. These practices during childhood, when the eye is still growing, have created a situation where approximately half of the individuals in Europe and the USA are now myopic, while the global development of Southeast Asian countries is leading there to what is described as an epidemic of myopia [50, 51].

The sum of such vulnerabilities forces us to re-evaluate the human body, not as an optimally designed machine, but rather as a series of compromises that indeed has left us vulnerable to a variety of conditions, particularly as we age. In fact, ageing represents the ultimate "trade-off" in evolutionary terms. As so elegantly expressed by George Williams [52, 53], ageing itself has evolved as a by-product of reproductive effort earlier in life. The basic point here is that life itself has not evolved to promote personal happiness and longevity and cannot continue without successful reproduction and surviving offspring which are favoured despite the trade-offs. The starkness of this biological statement makes for uncomfortable reading for highly cultural organisms that have developed traits that do indeed (in particularly favourable environments) promote health, longevity, and happiness, sometimes at the expense of individual reproduction [52, 53]. This should not, however, lead us to dispute the clinical significance of vulnerabilities in evolutionary design, and could help in understanding the source of individual disease and decline.

As an exemplar of the ultimate outcome of evolutionary success, as we age, humans and other sexually reproducing organisms suffer from what is termed the "declining force of natural selection" [54]. Our genes are passed on through reproduction, which generally occurs earlier in life. Traits that are maladaptive early in life would tend to be "selected out" because they would be passed on to our offspring who might not survive with these characteristics. However, deleterious traits that are expressed later in life, when successful reproduction is less likely, will not be selected against and can contribute to the ageing process. Furthermore, traits that are beneficial earlier in life and that promote reproductive effort might be associated with negative effects later on in life, a trade-off known as "antagonistic pleiotropy" [52, 53]. Again, this situation promotes the ageing process. Understanding these concepts can be helpful in researching and treating senescent conditions (see Chaps. 18, 19, 21).

Some design features of the human body are more vulnerable than others due to chance or stochastic events in our evolutionary history, as well as the possible action of other evolutionary forces aside from natural selection. These other forces are mutation, genetic drift, and gene flow (otherwise known as migration). Genetic mutations are, in fact, relatively rare and, by their nature, stochastic and are likely to have had a fairly minimum impact on the evolution of specific traits. Exceptions can occur where gene mutations affect control regions. An example of this is where humans evolved the capacity to digest lactose in adulthood—one of the better documented cases of recent human gene–culture coevolution [55–57]. Genetic drift

occurs in situations where small populations representing a sample of a wider gene pool remain in relative isolation and develop unique or specific genetic profiles as a result of their small sizes and isolation (this could be through geography or cultural practices). Where such isolated populations expand, a phenomenon known as the "founder effect" can occur, where specific and deleterious traits that occur by chance in the sample population increase in representation. Examples of such founder effects exist among the Amish in the USA where polydactyly is relatively common [58]. Population bottlenecks can also occur at various points in time where populations suffer serious demographic decline. The remaining small populations are likely to experience genetic drift.

1.2.2 Rethinking Medical Practice

Perhaps the least misunderstood and most accepted evolutionary concept in biology and medicine is that of competition with a variety of pathogens with whom we coexist. This has led to one of the most urgent crises in modern medical care, namely the emergence of antibiotic resistance ([59–61], Box 1.1). We are in fact in an "arms race" against rapidly evolving micro-organisms such as bacteria. The crisis is so urgent that the UK Chief Medical Officer, Professor Sally Davies, stated in March 2013 that "the danger posed by growing resistance to antibiotics should be ranked along with terrorism on a list of threats to the nation" [62]. Similarly, in May 2015, Germany's Health Minister, Hermann Gröhe, opined that: "If antibiotics are no longer effective, treatment options could return to those of a pre-Penicillin age" [63]. Understanding the concept of evolved, antibiotic resistance has led to a growing recognition to limit the use of antibiotics in everyday medical settings to cases where it is clear that a patient is in fact suffering from a bacterial (as opposed to a viral) infection, or in cases of less severe infections to allow patients to recover by themselves [64]. A similar adaptive approach is being discussed in relation to cancer chemotherapy in order to limit the potentially damaging effect of emerging resistant tumour cells [65, 66].

Box 1.1 Adaptation and Natural Selection: The Example of Antibiotic Resistance

Antibiotic Use Antibiotics were first developed for medical use in the early twentieth century, primarily to treat bacterial infections, although they also work against fungi and protozoa. As the pharmaceutical industry grew, and antibiotics were readily available, they were increasingly prescribed and have also been used prophylactically and extensively in farming to prevent loss of livestock from common infections. Unwittingly, the overuse—and sometimes inappropriate use—of antibiotics has led to the evolution of antibiotic resistance among communities of bacteria. What does this mean?

Fundamentals of Natural Selection As pointed out by Darwin in the "The Origin of Species" [90], natural selection requires three key features: first, organisms within a species must vary in their characteristics or traits. Secondly, traits must be heritable which means they can be passed on to future generations. Thirdly, the variants must have differing reproductive value in a given environment, some being fitter (i.e. reproducing more) than other. Several bacterial traits meet these preconditions, allowing the evolution of different frequencies of variants in their population.

Antibiotic Resistance Resistant bacteria are "selected for" if they have acquired—through inheritance or random mutation—characteristics which enable them to survive being targeted with antibiotics. Individual bacteria from a population might just by chance have properties that confer resistance, meaning they are better adapted. Surviving bacteria can then reproduce and pass the resistant traits to their descendants. The resulting population has evolved to resist the previously effective antibiotic (Fig. 1.1). The chances of resistance arising are related both to variability present in a population and to the rate at which it reproduces, as each round of reproduction will produce new variants into a population pool (either through replication errors or through recombination events). The reason why antibiotic resistance can evolve so quickly is because of the rapid reproductive rate of these tiny organisms, which can take from minutes to about 24 h.

Resistance to Evolutionary Terminology Despite these evolutionary fundamentals, Antonovics et al. [91] have pointed out a discrepancy in evolutionary terminology in academic papers that discuss antibiotic resistance, and they urge its increasing use. Specifically, medical papers only used evolutionary terms 3 % of time (preferring words like "emergence") compared to 68 % for evolutionary biologists. Antonovics and colleagues speculate that resistance to evolutionary terms may have been encouraged to avoid "controversy". In some countries, resistance to evolutionary thinking is linked to strong religious sentiment and may be influencing how antibiotic resistance is described. Note that the website for the Center for Disease Control, the US watchdog for infectious and other diseases around the world, also omits the term evolutionary in describing antibiotic resistance [92]. Garry Trudeau, the well-known creator of the satirical comic strip, Doonesbury, played on this theme by suggesting a creationist patient diagnosed with tuberculosis should be treated with older antibiotics that no longer work against current strains of the disease [93].

A more controversial area is that of "defence", i.e. viewing certain physiological, pathogenic reactions as adaptive in nature rather than as negative consequences to illness that must be treated [15]. An obvious area where this is discussed is how to treat fevers in patients. It is increasingly recognised that a raised temperature is a physiological, adaptive response to an invading pathogen [67]. Raising the body's

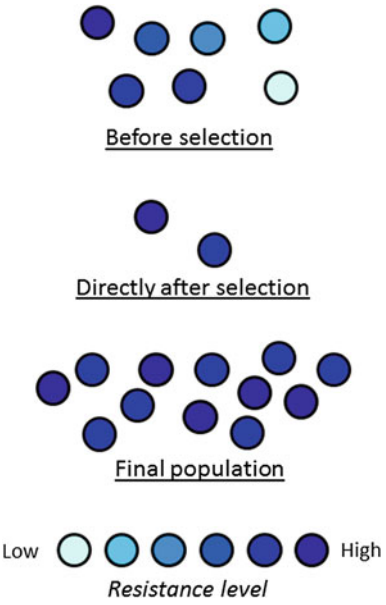

Fig. 1.1 Resistance acquired through selection by antibiotics

temperature inhibits the ability of a pathogen to overcome the body's defences. Fevers, particularly in children, were also treated in the past in order to prevent febrile convulsions, but it is now recognised that fevers alone do not cause this reaction, as outlined in UK National Institute for Clinical Excellence (NICE) guidelines [68].

A less recognised area of "defence" is developing in emergency medicine. Mervyn Singer, a consultant in intensive care medicine at University College London Hospitals, examined medical records for survivors from major historical battles such as Trafalgar and Waterloo [69–71]. Surprisingly, only a relatively small proportion of men died from trauma directly sustained on the battlefields (as opposed to those that suffered from infections as a sequela to injuries). These statistics led to a re-evaluation of the impact of many medical interventions that have come into use in emergency medicine, such as blood transfusions and ventilation designed to mitigate against blood loss and low oxygen levels in critical care patients. Singer [71: 1] has argued that emergency medicine has, in fact, "overventilated, overfluidised, overfed, overtransfused, and oversedated, and that these all contributed significantly to harm". Instead, emergency medicine needs fewer interventions that interfere with natural physiological adaptations designed to try and keep the individual alive in the face of massive trauma.

Singer [71] has also argued that: "We deferentially followed the seemingly unassailable logic that normal healthy values would provide the optimal milieu for either maintaining organ function or hastening recovery". This leads to a consideration of how doctors approach the concept of "norms". It is reasonable that doctors use concepts of "normal" values for a number of diagnostics to evaluate the

health of patients. In contrast, evolutionary biology embraces the concept of variation, since this is essential for the process of natural selection and adaptation. Appreciation of the importance of individual variation is filtering into medicine and forms the foundation for advocates of a more personalised medicine. It is also increasingly applicable to our understanding of tumour evolution and the unique properties of cancer in treating individual patients [72–74].

1.3 Doctors and Evolution: An Evolving Relationship

Given the applicability of evolutionary perspectives to medicine, why are evolutionary concepts unfamiliar to so many doctors? The two areas have not always been divorced. As pointed out by Fabio Zampieri [75], from approximately 1880 to 1940, Darwinian approaches to medicine enjoyed an early heyday referred to by him as "medical Darwinism", where many of the themes which today find resonance and relevance among practitioners were also prominent, including how infectious diseases might evolve, how "civilisation" might create predispositions to specific illnesses, and a concern with cancer. However, a preoccupation with heredity and inherited susceptibility to disease (known as diathesis and later constitutionalism) played into a growing interest in eugenics, a term coined in 1883 by Darwin's cousin, Francis Galton, who was impressed by the principles of selective breeding shown so clearly in relation primarily to plants and pigeons in Darwin's famous book "The Origin of Species", and who sought to bring these principles to bear on "improving" the human condition. The misuse of eugenic principles during the 1940s onwards led to a socially mandated suppression for several decades of any kind of Darwinian approaches to health [75].

Since the publication of the 1991 landmark article "Dawn of Darwinian Medicine" by Williams and Nesse [33], the problem in the revival of evolutionary medicine has been in attempting to demonstrate to clinicians the relevance and utility of evolutionary approaches. Fortunately, the field has developed far enough that considerable progress is already being made, and evolutionary medicine can perhaps be said to be entering into a new maturity. There are a number of relevant, edited books that have been published that are also suitable as teaching texts [13, 17, 18], two primary textbooks [14, 76], and other single-authored volumes [15, 16, 77]. The field has a new International Society, *the International Society for Evolutionary Medicine and Public Health* (ISEMPH) that met for the first time in March 2015 in Tempe, Arizona. There are two new journals that began in 2012 and 2013, respectively, the *Journal of Evolutionary Medicine* published by Ashdin and *Evolutionary Medicine and Public Health* published by Oxford University Press, and there are now a myriad of articles that have been published in other places that are far too numerous to mention.

Earlier, however, it would be fair to say that a few different strands of interests were developing within evolutionary medicine without a great deal of overlap between contributors. Aside from the growing number of articles by Randy Nesse,

two volumes edited by Stephen Stearns and Jacob Koella were published in 1999 [78] and 2008 [17] that focused primarily on topics related to host–pathogen coevolution, and genetics and vaccine development, although a couple of articles related to ageing and reproductive health overlapped with some social science topics. Edited volumes also appeared from scholars primarily in anthropology, concentrating on topics derived more from a social science perspective including maternal and infant health, and environmental mismatch [18, 79]. A fourth approach emerged somewhat later and has been spearheaded by physicians whose research represents the field of early life development or foetal programming, stimulated by work in the 1980's by David Barker at the University of Southampton. This parallel group, with strong overlap with some topics relevant to evolutionary medicine, developed into the *International Society for Developmental Origins of Health and Disease* (DOHaD) in 2003. The Society also established the *Journal of Developmental Origins of Health and Disease* in 2009. As stated on its website, its main aim is for "the scientific exploration of early human development in relation to chronic disease in later life" [80] (see Chap. 6), and it clearly has overlapping interests with the theme of "mismatch" from evolutionary medicine. The DOHaD group meets every two years in different locations around the globe, with the 2017 meeting in Cape Town, South Africa. In relation to evolutionary medicine, this developmentally-focused subfield has been represented primarily by Peter Gluckman, Alan Beedle, and Mark Hanson, who also wrote the first textbook in evolutionary medicine [14], albeit with a strong focus representing their particular interests. Finally, Paul Ewald (see Chap. 14) wrote several early influential articles and a book in 1994 (*The Emergence of Infectious Diseases*, [5]) that was to have a profound effect on the field of evolutionary medicine. In fact, Williams' and Nesse's section on infectious diseases in their 1991 article was heavily influenced by an earlier [81] article by Ewald. The latter is also editor in chief of the *Journal of Evolutionary Medicine*. Happily, the original disaggregation of the field into distinct sub-areas is disappearing, as evidenced by the converging of many authors into more recent edited volumes, or as coauthors of papers, perhaps representing a maturation of the field as it develops. A unifying figure across most of these separate strands has been Randy Nesse, who now heads the new *Center for Evolution and Medicine* at the Arizona State University [82], and spearheaded the inaugural international meeting in 2015 of the ISEMPH that was also hosted at Tempe, Arizona [83].

Nesse has also been one of the most vociferous advocates that evolutionary theory should form the foundation for medical education in the future [3, 4, 35, 84]. In the last few years, a number of US and European individuals from biological and medical backgrounds, funded by the National Science Foundation and the National Evolutionary Synthesis Center (NESCent) in Durham, North Carolina, USA, have been developing new questions for the US Medical Exams—the Medical College Admissions Tests (MCATs)—that will require premedical students to have much more knowledge of evolutionary biology than was previously necessary [34]. A number of student societies for evolutionary medicine are springing up on medical school campuses, as exemplified by Michelle Blyth at the Louisiana State

University [85], and a couple of innovative medical schools are conducting Grand Rounds with evolutionary biologists (not just medical doctors) in tow [86]. Durham University began a new MSc programme in evolutionary medicine in 2011 that attracts UK intercalating medical students who have completed their fourth or fifth year of medical school and who will hopefully carry on their careers equipped with the additional tools of evolutionary thinking [87]. Training in evolutionary medicine in mainland Europe can also be obtained at the Zurich Institute for Evolutionary Medicine [88].

All of these developments should give us hope that evolutionary medicine is finally coming into its own and has reached a stage where it can gradually infiltrate into many areas of traditional medical practice. Although the field was criticised in 2012 as having more "breadth than depth" [89: 246], the growing and intimate involvement of medical doctors with the field, as exemplified by the Grand Rounds at UCLA, belies this criticism. It has been almost 25 years since the publication of Williams' and Nesse's [33] article on the "Dawn of Darwinian Medicine" and 65 years since the demise of "Medical Darwinism" [75]. Perhaps this is the dawn of a new era that we might call "embedded evolutionary medicine" when clinicians and their trainers actually embrace evolutionary concepts and join forces with evolutionary scholars interested in health issues.

Glossary

Autoimmune disorders	Occur where the body produces antibodies against its own components (called autoantibodies) and attacks specific cells in the body. The causes of such autoimmune diseases are often unknown. They include conditions such as multiple sclerosis, rheumatoid arthritis, inflammatory bowel disease, and type 1 diabetes
Foetal programming	Refers to the potential for programming for alternative phenotypes during foetal life based on the environment experienced in utero and particularly where nutritional deficits constrain optimal foetal development during gestation
Genotype	Refers to the genetic make-up of an individual
Host–pathogen coevolution	Refers to the arms race that exists between an individual organism (the host) and a variety of other organisms that can cause diseases in that host (pathogens). Both hosts and pathogens will adapt over time to their coexistence, as they are under constant selective pressure for reproduction and survival

Immunoglobulin E	(IgE) is an antibody found in mammals and thought to have evolved as mammals became infested with parasitic worms (helminths) and protozoa (including malarial parasites). This antibody is produced and becomes elevated in allergic conditions such as asthma
Phenotype	Refers to the sum of the genetic make-up of an individual (its genotype) modified by environmental influences experienced during growth, development, and maintenance across the life course
Phylogeny	The evolutionary relationships between species across long time spans
Polydactyly	A genetic condition characterised by an excess number of digits or fingers
Population bottleneck	Occurs where populations of individuals reach sufficiently low numbers and variability in the gene pool that genetic drift is likely to occur

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