CHAPTER 1

Some Basic Information About Ageing

Defining old age

A person's age is typically measured in terms of how many years old they are; or in other words, how many times the Earth has been round the sun since they were born. This is known as *chronological age*. We tend to treat age as if it is a precise and accurate marker. In part this is because we use it as a legally binding guide to the age at which certain things become permissible. But these are essentially arbitrary measures, devised by countries according to their particular value systems. Thus, there are considerable national differences in the age at which it is legal to have sex or to drink alcohol. In the case of retirement, pensionable age varies widely between countries, from the mid-50s to the late 60s, and in many countries there is no compulsory retirement age.

Thus, retirement ages, although because they are official have an appearance of objectivity about them, in reality are fairly arbitrary (if they were not, then every country would have the same retirement age). Exactly the same argument applies to deciding the chronological age at which old age begins. It is possible to devote *very* lengthy discussions to this topic (and academics have been prolix on this matter), but to cut to the chase, we generally argue that old age begins when a person reaches the chronological age of 60 years. There is

nothing particularly important or significant about the age of 60.[1] Over the centuries, there had been an informal consensus that it was a reasonable rule-of-thumb measure of the onset of old age, but it was not until the 19th century that opinion became a more formal belief. In part this was because of the work of a Belgian statistician called Quetelet, who wrote a highly influential book on how humans could be statistically analysed (he also, incidentally, invented the body mass index). In one of his key pieces of writing, Quetelet asserted that from 'sixty to sixty-five years of age viability loses much of its energy, that is to say, the probability of life then becomes very small' (Quetelet, 1836, p.178). We now know this argument is utterly wrong, but because the rest of the book was well received, by the 1840s the 'fact' that old age begins around the ages of 60 to 65 was widely believed (Mullan, 2002), and current researchers also tend to use these same ages as indicating the onset of old age.

It must be stressed that the age of 60 is these days only intended as a useful guide. Because we have to have some sort of figure for the purposes of classification, etc., 60 is generally used. But it must also be stressed that nobody actually believes that on a person's 60th birthday they 'become' old so that the day before they were a hale and hearty person and the day after they are devoid of teeth, hair, senses and mobility. Nor does anybody seriously think that everybody who is the same chronological age is also identical in other respects as well. It is perfectly obvious that people change and become 'old' at different rates, to the extent that we can say that some people look 'young for their age' whilst others of the same chronological age look 'right for their age' or 'old for their age'. Researchers know perfectly well that this figure is arbitrary, but it is better than saying 'old age sort of begins in the 60s or later or sooner in some people'.

Changes in life expectancy

Presented with the words 'life expectancy' an intelligent person might reasonably interpret it as meaning how long a person can expect to

1 Humans tend to give greater significance to ages or anniversaries ending in zero. Conversely, they tend to find numbers ending in zero contrived in real-life data – hence the old adage never to hand in an expenses claim that ends in zero (e.g. a claim for £103.34 looks more plausible than one for £100.00).

live. This is accurate in one sense, but in another far more important way, it is totally misleading. Consider the following example, taken from Stuart-Hamilton (2011). In 1400, it is estimated that the life expectancy in Europe was 35 years; by 1841, this had risen to 40 years, but by 1981, it had skyrocketed to 71 years (i.e. over double the 1400 figure). To the unwary, these figures seem to indicate that in 1400 it was almost impossible to grow old since most people died before they reached 35. Indeed, the irresistible mental image for some is that in 1400 'old age' must have been in the 40s. This is wrong. The reason for the lower life expectancy in 1400 is because a lot more people died in infancy and childhood than they do today. If a lot of people die at a very young age in a sample, then this drags down the group's mean or median score very dramatically; but this does not mean that if you survived childhood, your *remaining* life expectancy would be as bad. To illustrate this point, consider the life expectancy figures for people who have already reached the age of 60. In 1400 their life expectancy was 69, in 1841 it was 73, and in 1981 it was 76. The difference between the 1400 and the 1981 figures has shrunk from 36 years at birth to seven years at age 60. This clearly illustrates the prime reason for differences in life expectancy figures over historical time – namely, fewer people die in childhood, not that people who reach adulthood live vastly longer lives.

However, it can be argued that nonetheless, even allowing for infant mortality, people still live longer these days than in the past. For example, even if one of our ancestors reached the age of 60, they were still likely to be in the grave years before someone of the same age today. Can this trend continue? At this point the argument gets quite technical. Some researchers (e.g. Oeppen and Vaupel, 2002) argue that the only way is up, and that life expectancy is going to continue to rise year upon year. Others (e.g. Post and Binstock, 2004) claim that the rise is attributable to infant mortality statistics exerting an influence, and that once infant mortality is reduced to as close to zero as possible, the life expectancy figures will stop rising. Others have argued that future health trends are uncertain and that whilst health care has lowered death rates, increased obesity coupled with lower levels of exercise might increase death rates again, leading to an uncertain future in which numerous mortality rates and hence life expectancy

figures could be possible (Continuous Mortality Investigation, 2006a, 2006b). Thus, whether the trend will continue is uncertain.

The above is about the future – what about the present? In Britain today, about 11 million (c. 16%) of the population is aged 65 or older. There are similar figures for other industrialised nations (OECD, 2004).[2] The study of ageing is the study of a significant proportion of the population; and moreover, it is the study of a time of life that the majority of us can expect to experience.

Differences in life expectancy

Where people live can have a big effect on how long they will live. For example, North America has a life expectancy of 78, Latin America 74, Europe 76, Asia 70 – and Africa 55 (Population Reference Bureau, 2010). This largely reflects the level of technological development in the countries concerned (e.g. in Europe, if the EU countries are considered separately, their life expectancy is 79). A better infrastructure means not only better care, but better housing and public sanitation create an environment in which infectious disease is harder to spread. Death by communicable disease is ten times higher in developing countries, whereas in industrialised nations, death is far more likely to be due to chronic (i.e. long-lasting) conditions such as heart disease and cancer (Leader and Corfield, 2007).

However, even within the same country, some places confer better life expectancy than others. For example, within the UK, people in the north east of England have a life expectancy two and a half years less than people living in the south west of England. Many reasons can be suggested for this – differences in climate, local health care, level of stress in daily life, etc., can all be proposed with some justification. Ultimately, however, wealth seems to be the single most dominant factor. If we consider smaller areas of the country, the differences become more startling, with Kensington and Chelsea (a very prosperous area of London) recording the highest male at-birth figure of 84.4 years, compared with, at the other end of the scale, Glasgow City (71.1 years) (Office for National Statistics, 2010a, 2010b). This accords with reports from other countries that found

2 Throughout I will tend to use UK figures. However, all the arguments raised from them are generally applicable to other industrialised countries.

similar regional differences, where they also are largely associated with socio-economic status (see Griffiths and Fitzpatrick, 2001). It is worth noting that the differences between groups need not be very pronounced for there to be a difference in life expectancy. Marmot and Feeney (1997) studied UK civil servants and found that even within this occupational group (where nearly all could be described as at least financially comfortable), life expectancy significantly rose the higher the income of the sub-group being considered.

It would be misleading to attribute all differences in life expectancy to wealth. Other lifestyle factors that are not particularly wealth-dependent also play a role. A striking illustration of this is the *Roseto effect* (see Egolf *et al.*, 1992). This is named after an Italian-American community in Roseto, Pennsylvania, whose members' susceptibility to heart disease increased as it became more 'Americanised'. The instinctive reaction is to assume this was a dietary issue as Italian food is often seen as adhering to the 'Mediterranean diet' ideal of few saturated fats. Nothing could be further from the truth – the regional variant favoured by the Roseto community included seemingly artery-clogging quantities of saturated fats (the people also drank and smoked). The reason for the greater resistance to heart disease was ascribed to the Roseto community itself: wealth was not flaunted, people supported each other and social life was family-centred. However, as time progressed, members of the community moved away from this communal view to a more individualistic one. A study of Roseto at the point when this transition was becoming apparent predicted that there would be a concomitant rise in heart disease (Lynn *et al.*, 1967) – alas, this prediction proved all too accurate. However, it should be noted that the effect on the inhabitants of Roseto was not a uniform one – younger men and older women were disproportionately more badly affected (Egolf *et al.*, 1992).

Roseto is not quite unique – several other immigrant communities have been found that also have longer life expectancies (see Leader and Corfield, 2007). There is no doubt that cohesive supportive societies can in some instances improve life expectancy. However, it is improbable that this is the total explanation. For every supportive community resembling the town in *It's a Wonderful Life* there are arguably many more close-knit places that are snooping, judgemental and, accordingly, stressful. Presumably, sometimes the right balance

of support and interference is attained, but this seems to be a hard thing to achieve, given the rarity of places like Roseto (the town was originally selected for study precisely because it was so unusual). And whether a longer life spent in a town where individuality is repressed and everyone knows their place is a blessing or a curse is a debatable point. This is not to deny the principal findings from Roseto *et al.* – undoubtedly community support can prolong life expectancy under certain circumstances (similarly, Giles *et al.*, 2005 found that having five or more friends at the start of a ten-year study gave people a quarter less chance of dying by the end of the research). But nor can the findings be accepted uncritically.

How to increase life expectancy

It is understandable that throughout recorded history, people have been searching for ways to prolong life, and these are woven deeply into our ideas and attitudes. In past centuries, the *antediluvian ageing myth* (the belief that in ancient times, people lived far longer because they led more virtuous lives) was widely promulgated. A variant on this is the *hyperborean ageing myth* (the belief that there is a far-distant land where people live to incredible ages). An example of this is the belief in Prester John, which preoccupied many people in medieval Europe. Prester John was (supposedly) a Christian king who ruled over a land of fabulous wealth and great piety somewhere far away (reports generally place the kingdom as either in central Africa or China). Expeditions were even sent to find him (part of Marco Polo's brief was to find the kingdom if possible). We might smile at the naivety of the idea now, but science fiction writers often play on it – think how many science fiction films and TV series present races of wise and serene space aliens as being very old in human terms.

Yet another myth is that of the *fountain of youth* – the idea that there is an elixir or magic food that will confer immortality. This is the stuff of many a legend and popular work of fiction. It is also a keystone of the cosmetics and health and fitness industries. However, making someone *appear* younger is not the same as actually slowing down or stopping ageing. So, assuming that you cannot find the land of Prester John or a fountain of youth, what options are open to you to increase life expectancy?

Choosing the correct lifestyle is a good start. For example, it is estimated that the root causes of 35 per cent of all deaths in the USA in this century are attributable to indolence, poor diet and smoking (National Center for Injury Prevention and Control, 2006). Increasing the level of exercise, eating sensibly, cutting out smoking and excessive drinking are all good things to do and supported by ample research evidence (Ostwald and Dyer, 2011). For those who reject the idea of exercise and a limited-calorie diet of lettuce leaves and tofu, and hope instead that medical science will provide anti-ageing treatments, there might be a long wait. Ageing involves changes in multiple biochemical processes, and treating one without the others is unlikely to be particularly beneficial (an analogy might be replacing the window frames of an old house whilst the unusable floor boards, wiring and utility supplies are left untouched). Thus, even if there is a breakthrough tomorrow in one area of anti-ageing research, researchers are still likely to have to wait for other areas to catch up before an effective treatment can be created (see Rose, 1999, for an excellent review of the issues involved). And if a 'cure' for ageing is found, this may create more problems than it solves, not least of which is that if everyone can avoid ageing, overpopulation to an unprecedented degree seems inevitable. In which case, anti-ageing treatments will be either rationed or banned, both of which in turn create serious ethical and political dilemmas.

In addition to their lifestyle and where they live, a person seeking a long life also needs the right sort of genetic inheritance. This is the most probable explanation of the *Winston Churchill argument* – namely, that there are some people who can do all the wrong things (over-eat, over-drink, smoke like a chimney, have a highly stressful life) and yet live to an extreme old age. Put simply, regardless of other factors, long-lived people tend to produce long-lived offspring (Murphy, 1978). Which genes promote longer life is not fully known, and a lot of research has concentrated on fruit flies (which although they have about 70 per cent of genes in common with humans, clearly are not identical). So far, over 500 genes have been found that are associated with longevity (de Magalhaes, 2011). It is very unlikely that a simple single gene will be found that guarantees long life.

Another aspect of genetic inheritance is gender. Put simply, women live longer than men. In peaceful societies (i.e. where heavy war casualties do not distort the figures), the balance of men and women is

roughly equal until about 45 years. Thereafter, men die at a faster rate, so that by 70, there are approximately six women for every five men, and by 80, this ratio has moved to 4:1. Many reasons for the earlier deaths of men have been suggested. A popular conception is that it is because men have traditionally led physically more strenuous lives. However, this seems at best a marginal explanation, since comparisons of men and women matched for levels of physical industriousness still show a strong sex difference in mortality rates. It is also unlikely that there is a hormonal or other biological explanation (Austad, 2006). The most feasible explanation is that men are slightly more prone to risk-taking behaviours than women. Thus, they are more likely to drive a bit faster, be less fussy about healthy eating, and so forth. These differences might seem to be minor, and at any one point in time they probably are. However, over the years, the cumulative effects of these behaviours begin to mount up, leading to men dying earlier (see Zhang, Sasaki and Kesteloot, 1995).

The cost of living longer

The Ancient Greeks had a tale of Tithonus, a mortal man who asked for immortality, but forgot to add the important caveat that he wanted to remain eternally youthful. The Greek gods, with their typical twisted sense of humour, allowed Tithonus to become immortal, and for all eternity he grew older and older and older. The point of this cheerful tale is that people want to live long, but not at any price. One of the abiding worries of many individuals is the (false) belief that the medical profession wants to keep older people alive, no matter what the pain or suffering or indignity, simply because they have the means to do. Not surprisingly, this mistaken belief is called the *Tithonus myth*. The reality is that the medical profession is not trying to make people suffer, but they are bound by a professional and ethical code to prolong life. But nonetheless, old age is not necessarily a time of unbridled joy.

One of the prices of living longer is that a person is more likely to contract painful but not necessarily fatal conditions such as arthritis, as well as other conditions that might ultimately be fatal but can be kept under control albeit with some discomfort (e.g. heart disease causing angina, pulmonary disease causing breathlessness). From birth, the average citizen of an industrialised country can expect to spend at least

the final 10 per cent of their life suffering an appreciable disability (World Health Organization, 2004). And if this is not depressing enough, serious chronic illness (i.e. enough to impinge significantly on quality of life, if not immediately to disable) will probably appear several years prior to this (World Health Organization, 2004). What this means is that although life expectancy has increased over the past couple of centuries, *active life expectancy* (the predicted remaining years of mobility and freedom from pain and/or disability) has not increased at the same impressive rate.

This looks like a version of the Tithonus myth is correct – namely, that medical science has increased our lifespans but we pay for it with pain. However, this is arguably too gloomy a picture. Many conditions, although in themselves painful, can be successfully kept under control through drug therapy and palliative treatment (e.g. painkillers). And furthermore, although this sounds likes a cliché, there are improvements in medical care all the time. Thus, many commentators have argued that the future will be a time of *compression of morbidity* – in other words, in the future, the time spent in discomfort in later life will be diminished (see Fries, 2000). Indeed, there is already evidence that active life expectancy has increased over recent decades (Manton, Gu and Lowrimore, 2008), although the actual size of the improvement depends upon how exacting a scale of measurement is used (Unger, 2006).

There is another cost to growing older, and that is financial. The proportion of the population who are aged over 60 is growing. This currently stands at about one fifth for most industrialised nations, but is expected to rise to one third or more by 2040. In the case of the UK, the current figure is circa 27 per cent and is expected to rise to circa 50 per cent by 2050 (OECD, 2004). This is potentially economically disastrous news. Older people are by far the biggest consumers of health care, and also of course are the largest recipients of pensions, but in the main, older people do not pay for these. Pensions and health care are funded from taxation, and the bulk of that is paid by younger adults in employment. However, whilst the proportion of older adults is increasing, by definition the proportion of younger adults is *decreasing*. Thus, just when more people are needed in employment, the means for paying pensions and health care is diminishing. This change in the old-age dependency ratio leads to what has been termed the *demographic*

time bomb – the major and potentially catastrophic financial burden on the economies of the coming decades created by a greying population.

How this problem is to be dealt with is currently a key preoccupation of most governments. The full story is too lengthy to be dealt with here and in any case falls well outside the scope of this book, and at the time of writing it is changing so rapidly that a detailed account would be almost instantly out of date. However, in essence, governments need to deal with a huge increase in pensions and health and welfare provision. One way to do this is to increase the state pensionable age. This is being done in many countries (e.g. UK from 65 to 68 by 2044; Germany 65 to 67 by 2031, USA to 67 by 2027), though not always without protest. For example, in France in 2010, it was proposed that the pensionable age for state workers should be raised from 60 to 62, and the age for receiving a full state pension from 65 to 67, resulting in nationwide protests and strikes.

Another method of dealing with the problem is to make current pension schemes less generous (e.g. by no longer linking them to final salary levels) – financially sound, but morally reprehensible for those workers who have already invested in a pension scheme in good faith and are now nearing retirement. A further method is to make new pension schemes less generous. This is potentially a good solution. Those who want a spendthrift early life can do so knowing they will only have a subsistence pension later on, whilst the more prudent have time through additional pension contributions to bolster their savings. However, this scheme will only have its full effect decades from now when these workers retire. A further method is to increase current pension contributions; but then this is asking employees to pay more at a time when wage rises are being kept low and prices are nonetheless rising. And the final and possibly worst problem of all is the at times perilous state of the stock market. Pension funds rely very heavily on investments in stocks and bonds. Poor returns on these mean in turn there is less money for pensions. The financial crisis resulting from the collapse of several important banks from 2008 onwards resulted in appallingly poor investment conditions at the very time when a financial boom was needed.

However, it is also possible to over-egg the pudding. There has never yet been an economic downturn that has not been met with expert predictions that catastrophe and universal poverty lay round

the corner, and yet, here we all are, and in the main enjoying a far better standard of living. There are also good grounds for arguing that the costs of health care of older adults may be exaggerated by current accounting methods, and the real cost may turn out to be significantly lower (see Sanderson and Scherbov, 2010). At the time of writing, there is an over-used slogan resurrected from a World War II poster, which simply says 'Keep calm and carry on'.[3] In many respects this is the sanest advice for dealing with media doom-mongering about the future of pensions and pensioner care. To put things into perspective: in a pre-industrial society, a person has barely an even chance of reaching their fifth birthday.

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So far in this chapter we have examined how we measure ageing and life expectancy, and some of the pitfalls that these measurements can create. In addition, we have examined how increasing life expectancy is not without potential problems in terms of both personal and financial cost. However, these issues beg the question of *why* we age at all. What are the processes that determine this, why do they exist, and what are their physical effects? The remainder of this chapter will be spent addressing these issues.

The ageing body

A full description of what happens to the body in later life would take a sizeable text, and in any case would be over-detailed for the purposes of this book. However, the fundamental message is this – as people grow older, they lose cells. This means that many parts of the body lose bulk and their functionality is lowered. For example, there is less muscle and skin, which get less elastic. If a fold of skin on the back of a person's hand is (gently!) pinched, the skin will go back to its original shape when the fingers let go. In younger adults, this happens almost instantly, because the skin is still very elastic. However, in an

3 On a note of historical accuracy, the poster was never actually publically used in World War II, merely prepared for distribution. It was a chance find of one of the few surviving copies that led to its reprinting a few years ago as a piece of decorative art.

older adult, the skin will revert to its previous shape more slowly. In a very old person with thinning wrinkling skin, the fold of skin might not totally revert to its previous shape, because the skin has lost a high proportion of its elasticity. Given that this pattern of decline is taking place throughout the body, it can be readily appreciated that this will have a profound effect upon older people's physical state and capabilities. In a word, the body becomes less *efficient*.

Loss of physical efficiency can have far-reaching psychological consequences. The changes might alter people's images of themselves and others. That this is seen overwhelmingly in negative terms is witnessed by the billions of pounds spent on cosmetic treatments each year in an attempt to hide or remove the signs of ageing. But even for those not concerned with their appearance, a less efficient body has direct effects on how well the brain functions. Less efficient respiratory and cardiovascular systems mean that the brain is less well supplied with oxygen and nutrients carried in the blood. This is akin to a car engine being expected to run on a low-octane fuel. The brain thus begins to run slower and tires more easily. One of the main reasons older adults tend to fall asleep after a meal is that the body requires so much oxygen and energy to digest the food that there is not enough in reserve to keep the brain fully active and alert.

The above applies to healthy, disease-free bodies. The situation of course only becomes worse if the body parts are damaged further. As we grow older, all the parts of our body get worn through use. This would be bad enough in itself, but on top of that, the longer we live, the greater the chances that we will develop chronic conditions such as arthritis, rheumatism, or one of a host of cardiovascular or respiratory diseases. Therefore, it is small wonder that the ageing body is an increasingly inefficient support mechanism for the brain and thus the mind. Furthermore, all the senses, responsible for directly providing the brain with information about the world, show deterioration (see Jackson and Owsley, 2003).

What of the brain itself? It has long been known that the brain decreases in volume in later life, even in healthy, well-functioning individuals. Recent brain-scan studies have found that in old age, we lose between 0.5 and 1 per cent of our brain tissue each year (Fjell and Walhovd, 2010). This loss is probably not uniform across the brain. For example, there is greater loss in the frontal and temporal regions of

the cortex. For the uninitiated, the cortex is the wrinkly outer surface of the brain, and is responsible for a very high proportion of what is termed higher-order functioning – in other words, what might be termed intellectual processes (as opposed to more basic mechanisms such as controlling hunger and body movements, which although of course important are not 'intellectual'). The frontal cortex (roughly speaking, the bit behind the forehead) and the temporal cortex (the bit behind the temples) are heavily involved in planning and memory. In general (though note there are exceptions), the more brain tissue that is lost, the greater the intellectual decline in old age (Fjell and Walhovd, 2010).

In addition to loss of brain tissue, the surviving brain cells also change in function. Generally, older nerve cells send signals more slowly and with greater *neural noise*. The concept of noise in the system is an important one in some psychological theories and is best explained by analogy. In the brain, millions of nerve cells are carrying messages all the time. In an ideal world, each neural signal would be transmitted without any leakage of that signal (rather like lots of people talking in the same room at once, but each whispering into the recipient's ear, so nobody else is distracted). In reality, these signals often 'leak' so that signals form background noise to each other (extending our analogy, this is like everyone whispering a bit too loudly, so that unintended listeners get to hear other people's whispers as background distraction). The end result of this is that neural noise interferes with brain processing and makes mental activity less efficient.

A further ageing effect on the brain is that many activities in the brain become less localised. We will return to this in a later chapter, but in essence what happens is this: in younger adults, many mental tasks seem to be performed by very specifically defined and relatively small areas of the brain, whereas in older adults, more of the brain (and often in two or more different areas) are required to do the same task. The reason for this is still being debated, but it seems likely that older people have to compensate for the decline in brain cell numbers and efficiency by using more of the brain to do the same mental tasks.

Thus, both mind and body are physically less efficient in later life, both in terms of the lowered number of cells and the poorer abilities of the cells that are left. However, this begs the question of why this should occur at all, and that is addressed below in the final section of this chapter.

Causes of biological ageing

'Biological ageing' is generally used loosely to refer to the physical state of the body and how it changes over time. This typically involves cataloguing what the state of the body is at different chronological ages, and thus can be used to measure whether an individual is physically ageing faster or slower than the norm. There is no escaping the fact that no matter how many skin creams are bought and trips to the gym are made, the body declines with advanced chronological age. In the hands of many biologists and medics, this can be expressed in unnecessarily depressing terms. For example, there is a famous description of later life as being *post-developmental*: 'all the latent capacities for development have been actualized, leaving only late-acting potentialities for harm' (Bromley, 1988, p.30). Physical ageing does indeed seem to be one of nature's cruellest jokes. Understandably, people have questioned why it should happen, as the numerous works of art and literature on the subject amply demonstrate. The scientific explanation is not yet fully known, but research over the past few decades has given us insights into at least the broad set of reasons, even if all the details have yet to be worked out.

The first point to establish is that the body's cells are not immortal – over a period of about seven years most of them die and are replaced by new cells or are lost. Cell loss becomes a notable feature from early adulthood onwards, with most bodily systems showing a decline of 0.8–1 per cent per annum after the age of 30 (Hayflick, 1997). The course of this loss is very slow and, as most bodily systems have over-capacity built into them, it is only in about the sixth decade of life that the change is first apparent to the casual observer. This explains why a lot of bodily ageing changes occur (quite simply, cells are lost and not replaced) and why they occur in later life (because over-capacity makes the loss unnoticeable for decades). This explains why we get thinning hair, weaker muscles, thinner and less elastic skin, and so forth. However, it immediately begs the question – why don't cells carry on replacing themselves? Why do they eventually die off after so many duplications?

There are two explanations as to why cells do not replicate themselves ad infinitum. The first is that the cells seem to have a limited 'shelf life' built in. The *Hayflick phenomenon* (named after its discoverer: see Hayflick, 1997) states that living cells taken from the body and

raised *in vitro* will only reduplicate themselves a limited number of times before dying (the *Hayflick limit*). In other words, cells seem to be pre-programmed to die. Why this happens is open to debate. One plausible explanation currently in great favour concerns the *telomere*, a sequence of DNA located at either end of all chromosomes. Each time the cell duplicates, the telomere shortens a bit, until eventually it disappears, and the genetic information disintegrates, preventing further duplication. The analogy frequently made is to imagine the chromosome as a shoelace, with the telomere as the plastic bit at either end that prevents the shoelace fraying. The analogy is an appropriate one, because it is felt that telomeres are a key component in maintaining the structural integrity of chromosomes (see Cong, Wright and Shay, 2002; de Megalhaes, 2011).

But this begs the question – why does the telomere do this? This leads to the second explanation of why cells don't replicate themselves ad infinitum. If the telomere did not shorten after each duplication, then the cell could in effect become immortal.[4] The problem is that although this sounds desirable, it is the last thing that one would want to happen. The reason for this is because each time the cell copies itself, errors occur. Up to a point, the cell copies are sufficiently similar to the original to be workable, but after a few copying processes, the cumulative errors make the cell in effect unusable for purpose.[5] The Hayflick limit is therefore a useful device – it kills off cells before they can be more of a hindrance than a use. Indeed, if damaged cells are accidentally revived and given longer and unbreakable telomeres, then they can and do result in cancerous growths (see Holliday, 2007).

This still begs the question as to why nature has given us such an appalling method of replicating cells. Why have we not got a better way of replicating cells that avoids the problems of poor duplication, the Hayflick limit, etc., so that we have no cellular decline in later life?

4 That is, only physically destroying it would kill it, but otherwise it could replicate itself time after time.

5 If you have the time, money, and inclination, try photocopying a black and white photograph on a photocopier. Then copying the copy. Then copying the copy of the copy, and so forth, ad infinitum. You will find that after a few copies, the original photograph is nigh-on unrecognisable. Clearly all that copying of copies does not preserve the original. Would you want one of these very poor copies to be preserved as an example of the original photograph? Of course not – the copy is in effect useless. A similar problem besets cell copies.

One argument occasionally raised is that the debilitation caused by ageing is planned. This states that bodies have an inbuilt programme to decay and die in order to make way for younger members of the species, and thus prevent the problem of overcrowding. Other versions include the concept that individuals grow weaker so that they become easier targets for predators, thereby preventing younger species members (still capable of breeding) from being chosen as targets. Such arguments are still accepted uncritically by a few commentators, but they are undermined by one simple fact: very few animals in their natural habitat reach old age. Accordingly, because older animals are so rare 'in the wild', it is unlikely that evolutionary pressure has created a method of 'self-culling' a species – predators, disease and accident do a satisfactory job in themselves (see Medawar, 1952).

For the majority of the time life has existed on this planet, nearly all multi-cellular organisms died long before they could show signs of ageing. Therefore, there has never been an evolutionary drive for better cell replication in later life. Incredible as it seems, the only time a large number of older animals can be seen is when they are family pets or in zoos. In the wild, old animals are scarce. What this means is that inefficient cell replication and the ageing changes resulting from this are something that nature never planned for: the effects of old age are extremely unusual in the natural world because old age is so rarely reached.

Thus, when we consider an older person, it is difficult to argue that a particular change in behaviour or bodily function is 'designed' to happen. Wrinkled skin, more brittle bones, lowered memory span or lowered response speeds may never have been 'planned'[6] by evolutionary forces – they have arisen because of one of several reasons. First, there was no evolutionary pressure to prevent them so they just happened (this is the basis of the *mutation accumulation theory*). Another theory argues that some bodily processes have beneficial effects in early life and only have bad effects in later life. But because people usually died of other causes before they reached old age and

6 Note that 'planned' is a metaphor: evolutionary change is essentially random even if the end result appears to be the result of intentional planning. People with some interpretations of religious belief may take exception to this argument and they are welcome to place their own explanation on the phenomenon, based on providential design.

the bad things happened, the downside of these processes was never controlled for by evolutionary forces. For example, high levels of male hormones might confer more strength and virility in early life, but if males are going to be dead before they reach old age, there is no reason to worry about the late life downside of baldness and heart disease (this is the essence of the *antagonistic pleiotropy theory*). Another theory (the *disposable soma theory*) argues that we spend early adult life optimising our reproductive success and keeping our reproductive systems in top condition so we can breed and spread our genes. If this maintenance means that other bodily systems decline in relative terms because they do not get maintained to the same level, then once again, there is no reason for concern, because death will occur before there are noticeable effects of ageing. These arguments might sound fanciful, but it must be stressed once again that most individual members of species in 'natural' surroundings are dead before these characteristics can ever manifest themselves (see Zwaan, 1999). It is both the curse and the privilege of modern life to see evolution cheated in this way.

Hayflick (1994) makes the useful analogy of the life course being, in evolutionary terms, like a satellite sent on a mission to survey a distant planet. Once a satellite has done its mission and sent back pictures of its target, it carries on into space, continuing to send back useful and informative signals until eventually accident or simple decay terminates its activities. In a similar way, individuals, once they have accomplished their target of producing viable offspring, continue to live until accident or illness kills them. However, the life of the satellite after sending back the photos of the target, or of the individual after breeding, is coincidental. Enough 'over-engineering' has to be built into the system to ensure that the job can be accomplished with something to spare. We interpret this extra life afforded by the over-engineering as a 'natural' part of the lifespan, but in fact in evolutionary terms it is an accidental gift, not a right. However, in another sense the argument that old age 'defeats' evolution is inaccurate – what is being shown in effect is what evolutionary pressure thinks of old age. Namely, it is a time when in evolutionary terms the usefulness to species survival is low, and thus it can act as a repository for all types of decay that will strike those who have 'failed' to die having successfully reproduced their genes.

Suggested further reading

An excellent text on ageing populations and the ageing body is *Brocklehurst's Textbook of Geriatric Medicine and Gerontology* (Tallis and Fillit, 2003). It should be noted that at the time of writing, the current hardback edition retails at circa £130 (thus, for many this may be a book for borrowing from a library). If you are not already fed up with the author's writing, then *An Introduction to Gerontology* (Stuart-Hamilton, 2011) is quite readable and indeed is excellent if you skip the author's own chapters. Fjell and Walhovd (2010) provide a solid review of brain anatomy changes in later life. For those interested in the history of ageing, then Thane (2000) is warmly recommended. In addition to being scholarly and informative, it is also an engrossing read. Although the book concentrates on the history of ageing and later life in England, many of the findings will be applicable to other cultures. For discussions of population change and its effects on policy, it is difficult to recommend a particular source for fear of it being immediately out of date. Government websites such as National Statistics Online (www.statistics.gov.uk) and the Population Reference Bureau (www.prb.org) are useful for basic up-to-date figures and often produce (surprisingly readable) summary reports. Wait (2011) gives an excellent review of the basics of policies on ageing and the issues facing policy-makers.