

Chapter 13

Ethical Perspectives in Biogerontology

Sebastian Sethe and João Pedro de Magalhães

13.1 Introduction

From the perspective of scientists involved in biogerontology, the branch of science focused on the biology of aging, ethical themes can be classified as either belonging to an ‘inner sphere’ where the conduct of the aging research itself is under ethical scrutiny; or, secondly, an ‘outer sphere’ where questions are raised about the philosophical and social implications of ‘curing aging’. There is of course some overlap between these spheres (most evidently when communicating between them) but generally commentators tend to focus on the ‘outer sphere’. Here, we shall focus on the ‘inside’ perspective of moral agents involved in biogerontology. We make no claim to be representing ‘the biogerontology perspective’ nor do we aim to chart, let alone consider in depth all the ethical issues that might arise from this perspective. However, in a debate where commentators—including biogerontologists—tend to discuss abstract positions, we suggest that considering these issues from the particular vantage point of a research protagonist provides a useful further angle to enrich the discussions.

There are some ‘established’ ethical issues that arise in biogerontology as they do in other fields, albeit with some particular characteristics: Research relying on elderly people may be more difficult to conduct since they may be more prone to frailty and regenerate less quickly plus the capacity for giving informed consent may be a greater issue than in other demographic groups. Animal research may involve keeping animals a longer time in confinement than in other fields giving rise to different husbandry requirements. Where research focuses on genetic or medical data privacy may be an issue. This is not necessarily surmountable simply by resorting to anonymization of data—a data subject may resent that her information

J. P. de Magalhães (✉)

Integrative Genomics of Ageing Group, Institute of Integrative Biology, Biosciences Building,
University of Liverpool, Crown Street, Liverpool L69 7ZB, United Kingdom
e-mail: jp@senescence.info

S. Sethe

NorthEast England Stem Cell Institute, United Kingdom
e-mail: ssethe@sgul.ac.uk

is being used in research to ‘cure aging’ whether or not personal data protection is an issue. These examples illustrate that even ‘standard’ ethical challenges may be structured differently in biogerontology, but it is worth remembering that when one considers the most immediate ethical issues that biogerontologists face, these are not very different from those encountered by researchers in other fields. Other such ‘standard’ issues have to do with a researchers’ personal ethics: How one treats one’s students and staff, how one behaves ethically as a peer and author and so on. At least one of these ‘standard’ ethical challenges merits some further discussion.

Herein, we discuss a spectrum of ethical considerations as they present themselves to biogerontology internally, when considering how to communicate the scientific developments and potential technological advances; and externally, when thinking about long-term social consequences of anticipated technological progress in this area, in particular issues derived from the potential for ‘curing aging’ such as overpopulation and cultural stagnation.

13.2 Communicating the Potential of Biogerontology

A primary ethical challenge to researchers is to communicate truthfully with non-specialists in explaining their research, the development and potential of their field. Aging is a personal, emotive and complex topic and in the battle to secure funding researchers must be mindful of not creating unrealistic hopes. In the context of ‘curing aging’ especially, one looks at a long history of hope and desire driving science and pseudo-science. So what can ethically be said about the potential of curing aging? Herein, we first discuss the state and potential of the biogerontology field, before comparing the study of aging to that of other diseases and discussing the motivations of researchers in this field.

13.2.1 Present Understanding of Aging and Potential Interventions

The underlying cellular and molecular mechanisms of aging and the process(es) driving the aging process are still poorly understood. Conceptually, biogerontologists have proposed two broad explanations for aging: Damage-based theories of aging that posit that aging results from random or stochastic damage and programmed theories of aging that suggest that aging results from predetermined mechanisms, usually with an element of genetic regulation (de Magalhaes 2011). Damage-based theories include the free radical theory of aging, which suggests that a gradual build-up of oxidative damage with age drives the aging process, and the idea that DNA damage accumulation with age causes the physiological and functional decline we call aging.

To understand modern ‘programmed’ theories it is necessary to appreciate that evolutionary origins of aging differ from those of other traits: Contrary to other processes, such as development which is orchestrated by our genome (with environmental inputs playing a role) and is the product of millions of years of evolution, human aging is not thought to have evolved ‘for a purpose’. Instead, aging can be considered a by-product of evolution. Because in the wild only a small percentage of organisms survive long enough to reach ‘old age’ the force of natural selection declines with age and so evolutionarily there is little pressures to, for example, favour genes that are only beneficial late in life (Kirkwood 2005). Therefore, it is widely accepted among evolutionary biologists that human aging is a process that was not ‘selected for’ by evolution but rather escaped the force of natural selection.

Because of the evolutionary theory of aging, modern ‘programmed’ theories tend to focus on programmatic aspects of aging, such as gene expression changes or genetically regulated chains of events. The developmental theory of aging, for instance, argues that developmental mechanisms can regulate aging as an indirect consequence of developmental processes optimized to maximize physiological function for reproduction which then become detrimental in adulthood (de Magalhaes and Church 2005). A combination of theories of aging cannot also be excluded and more than one may turn out to be correct, but this distinction between whether aging is caused by stochastic processes or by the genetic program has implications in terms of developing interventions and in the philosophical interpretation of those interventions: Are we repairing damage or are we trying to change our genetic program?

In spite of our incomplete understanding of aging, progress in biogerontology in the past few decades has been impressive, in particular at the genetic level. Researchers have identified hundreds of genes that when manipulated change the aging process in model organisms, from simple yeast to invertebrates and even mammals like mice (Kenyon 2010; de Magalhaes et al. 2009). For example, it is possible to extend the lifespan of mice by up to 50 %, delay the appearance of age-related diseases and increase health by disrupting a single gene (Flurkey et al. 2002). A number of gene variants have also been associated with human longevity and we are starting to understand the gene difference behind why some people live longer than others (Browner et al. 2004). This knowledge of the genetics of aging and longevity gives scientists a blueprint for intervening in the aging process, as discussed below.

The strongest effect from environmental manipulations of aging in mammals is observed from caloric restriction, an intervention that consists of restricting caloric intake without malnutrition, and which in some mouse strains (but not in all) can extend lifespan by up to 50 % (Fontana et al. 2010). It is unlikely such marked effects are applicable to humans, but a delay of human aging by targeting caloric restriction pathways and genes is possible within the coming years (de Magalhaes et al. 2012). Indeed, pharmaceutical or nutraceutical targeting of aging and/or caloric restriction related genes or pathways could lead to the development of new drugs for age-related diseases and potentially retarding the human aging process: The so-called anti-aging pill (Stipp 2010). Numerous companies and research labs are working in this paradigm with potential drugs undergoing clinical trials. For example, the drug

rapamycin can extend lifespan by 9–14 % when fed to middle-aged mice (Harrison et al. 2009).

While testing anti-aging drugs and diets for long-term effects is problematic, it is plausible that some have short-term effects on health parameters and on age-related diseases that can be tested in a clinical setting to obtain approval from regulatory agencies. It will then be up to the consumers to decide, based on short-term human clinical trials and animal studies, whether to adopt the anti-aging pill, diet or lifestyle in the long-term. Therefore, to some degree the life extending applications of research on aging are on the horizon. It will be up to individuals whether to adopt them or not and thus providing accurate and intelligible information to the public is a crucial task of biogerontologists.

In summary, although the essence of the basic process of aging remains contentious, there are many technical possibilities for how aging might be slowed. As a basic distinction, we can differentiate between ‘genetic engineering’ of the yet unborn and dealing with aging in adults. In the long-term reality, there may be a combination of these two possibilities. (Germline interventions are of course ethically contentious for other reasons. Aging researchers working in genetics are challenged to develop a position on this topic. However, as we have pointed out above, genetic research in aging may well pave the way to interventions other than germline alteration, ranging from adult gene therapy, to pharmaco- and nutrigenomics. Therefore, the ethical issues in germline gene therapy are not necessarily implicated in all research on the genetics of aging and are not what we would focus on here.)

13.2.2 The Possibility of a Cure

Most biogerontologists agree that life extending applications of research on aging are plausible (Butler et al. 2004). Contested remains the factor by which life expectancy can be increased (Richel 2003). Assuming we ‘cure’ aging and thus eliminate (or at least prevent the age-related increase in incidence of) all age-related pathologies, including cancer, heart disease, and neurodegeneration, our average lifespan would increase dramatically. A life expectancy of over a thousand years seems theoretically feasible. (The average lifespan ($t_{0.5}$) of a non-aging population is given by the equation: $t_{0.5} = -\ln 0.5/\text{IMR}$ (Finch 1990). Assuming the initial mortality rate, the IMR, of a typical population in an industrialized nation (0.0005/year) we have $t_{0.5} = 1,200$ years. Of course this assumes a constant IMR, which may not be the case if there are wars or pests that increase the IMR or conversely progresses in other areas that decrease the IMR).

Human aging goes beyond the general changes that occur at a molecular or cellular level; it likely has multiple organ-specific determinants (Sames 2005). Consequently, the challenge in aging is to apply both very general and very specific interventions, and to maintain their effectiveness indefinitely—a rather daunting endeavour. Nonetheless, some commentators have boldly suggested have that it may be possible to cure aging within the next few decades (de Grey and Rae 2008; Kurzweil and

Grossman 2004). Consequently, many are very critical of the suggestion that aging can even be cured (Warner et al. 2005).

Strategies to cure aging rely on a complex interface of technological advances in regenerative medicine and gene therapy (de Grey and Rae 2008) or computing and nanotechnology (Kurzweil and Grossman 2004) that may take a long time to materialise. A combination of extraordinary breakthroughs in several areas is required for these predictions to become reality. However, based on what we know about the aging process, there is no scientific reason why a dramatic extension of the mean as well as the highest achieved lifespan should not be possible. Precisely because aging is such a multi-faceted issue, it seems entirely feasible to solve the problem in a piecemeal fashion using a portfolio of medical and technological alternatives. It seems unlikely that a single intervention will suddenly abolish aging. More realistic is a stepwise approach, where life-years are added in small instalments. A paradigm shift would arguably come if this progress were to occur at such a high rate that it outpaces the rate of aging (de Grey 2004a).

However, at this point the ethical issue of responsible communication comes into new focus: If one agrees that aging is, in principle, amenable to drastic alteration, then keeping silent about this possibility is in itself ethically problematic (de Grey 2004b). While there are several ethical problems with unwarranted hype, there is also an ethical obligation not to 'undersell' aging research (Farrelly 2010).

This challenge incidentally arises on 'both sides' of the wider moral debate: The ethical obligation to be clear about the potential of a research field arises regardless of whether one agrees that aging requires a 'cure' in the first place—to be discussed next.

13.2.3 *'Simply' Avoiding Frailty?*

What can rather be excluded as a possibility is that significant life extension will simply prolong the period of old age. Harking to stories like the classic Tithonus or Gulliver's Struldbrugs, some accounts imagine life extension as the extension of 'old age' and increasing frailty. Fukuyama, for example envisages a geriatric society occupied with the perpetuation of decrepitude (Fukuyama 2002). Many gerontologists would claim that 'compression of morbidity' (Fries 1983) rather than life extension is the practical aim of their studies. This is a very appealing strategy to generate research funding: It is politically palatable by promising to mitigate the pervasive health impact of the 'silver tsunami' while not encroaching on ethically contentious territory. Instinctively, many would agree that a 'quick departure' is desirable.

However, those therapies which will have any noticeable effect on lifespan are very unlikely to act at the stage where system failure is imminent (de Grey 2005). Although there has been some minor progress, there is no evidence that the 'compression of morbidity' approach is effective (Fries 2003; Crimmins and Beltran-Sanchez 2011); in fact, it may detract from effective strategy (de Grey 2006). By making progress against frailty, it has been argued that interventional biogerontology needs to adopt a much more rigorous evidence base (Nadon et al. 2008), but there are also concerns

that regimes akin to the current schemes for marketing authorisation may not be appropriate. “A broad spectrum improvement in health is not an outcome that would currently motivate a drug trial and nor is frailty a recognised medical problem” (Partridge et al. 2011, p. 8).

These and similar discussions illustrate yet again the ethical importance of communicating the potential scope and impact of aging research clearly and truthfully: Such research does not fit conveniently into established political and regulatory categories.

13.2.4 *Is Aging a Disease?*

If there is potential to delay or even stop aging, how does that position a biogerontologist ethically towards the subject? Can aging be regarded as a disease with a view towards ‘curing’ it? This has significant ethical implications. ‘Age’ as a cause of death will rarely be found in mortality statistics. Barring accidents, most people in developed nations currently die of vascular complications or cancer—conditions intricately linked with aging, but that fit the common definition of disease. Still, some would object to classifying aging as disease, viewing aging as ubiquitous and natural (cf. Chap. 16).

It could be argued that contrary to popular belief, aging is not universal. A number of complex species, such as lobsters, rockfishes, and tortoises, do not show signs of aging (Finch 1990). Whatever the importance of death in evolution and in ecosystems, aging itself is certainly not a prerequisite to life. As mentioned above, senescence is now thought of as an evolutionary by-product rather than an end in itself. If aging is understood as a stochastic phenomenon, then it has been argued that “this makes aging unnatural and in no way an intrinsic part of human nature. As such, there is no reason why it is intrinsically wrong to try to reverse or cure aging” (Caplan 2005, p. 73).

We would doubt that common occurrence or ‘naturalness’ can be taken as a serious consideration either for or against treatment. Many of civilisations greatest achievements are a departure from ‘nature’. It seems odd if for those people who drive cars, take medicines, wear glasses, receive e-mail, watch television, and do not have to kill their own dinner think life-extension is unnatural. Consequently, the attempts to muster logical arguments distinguishing aging from disease tend to flounder rather helplessly. (Consider: “The difference between a biological cause, such as the mutation in a gene or the malfunctioning of a protein, and a life-process such as aging, is manifest. The gene and the protein are parts of the biological organism, whereas aging is a part of life as a lived process: Life as we live it” (Rehmann-Sutter 2011)). In the process, attempts to divine ethical problems may themselves appear of dubious ethical distinction: A person who is dying of cancer might not care whether the malignancy was triggered, hastened, or caused by a genetic disposition, a mutation, infection, an aberrant stem cell, immune system failure, oxidative stress, pollutants, radiation, or any combination of age-related factors. The question of whether curing

cancer in the young is morally desirable whereas curing cancer in the aged is inappropriate shifts the issue of whether aging is a disease from science to politics or indeed to regulation. “The clinical redefinition of aging as a disease state would not only make sense, but it would also foster the development of therapies of benefit to older people” (Gems 2011, p. 109). Thus, in this arena “Should we consider aging a disease?” is essentially asking “Should aging be cured?”

13.2.5 Biogerontology vs. Medicine

For strategic reasons, gerontology is moving away from a juxtaposition of aging and disease towards a discussion of age-associated pathology. It is apparently easier to make the case for curing Alzheimer’s, heart disease or stroke, tangible suffering associated with aging than to make a case for tackling the underlying condition.

Underlying conditions are also notoriously more difficult to address than symptomatic treatment. The fact that aging is so complex and pervasive makes it difficult to ‘narrow down’ findings towards distinguishable treatments (although some insights from biogerontology are more immediately subject to translation (Lim et al. in press)). In fact, in developing ‘respectability’ biogerontology needed to cast off associations with charlatanism, and “strengthen the boundaries between themselves and anti-aging practitioners and entrepreneurs (Fishman et al. 2008)”. Ironically, where biogerontologists have sought this distance, other biological sub-disciplines are challenged and struggling to become more translational. In fact this sort of ‘boundary work’, vigorously distancing biogerontology from ‘anti-aging medicine’ (Olshansky et al. 2002), has not helped to establish biogerontology as part of the moral endeavour of easing suffering. While the field of geriatrics shares with gerontology the ‘aging research’ umbrella term (and consequently even some conferences, societies, journals and funding pots), the two fields do not always have effective interface—not facilitated by the observation that both fields are inherently interdisciplinary (Clark 1993).

For various historical and conceptual reasons, aging research and medical research remain distinct (de Grey 2007), and this has not helped to clarify the role of aging as a disease.

This has never stopped biogerontology to—rightly—advertise its potential to address epidemiological and medical issues. Lately, aging research has risen to particular prominence as an imperative to tackle otherwise insurmountable demographic challenges (Rae et al. 2010). For this to be effective though, it may be necessary for biogerontology to develop more effective ways of making a difference—and this means going further than offering data interpretation in the field of geriatrics. By aspiring to make a real difference, biogerontology assumes automatically an ethical position towards aging.

13.2.6 Motivation

The motivation for aging research is of course not necessarily linked to wanting to cure aging. Indeed motivations of aging researchers are very heterogeneous (Underwood 2009). ‘The project’ to cure aging has been linked to various contentious ideological roots, and variously been identified as a political ploy either “aiming to control individuals” or/also as the expression of ultimate narcissistic individualism, as placing a burden on the elderly to perform in a consumerist marketplace, in short as another detriment of shallow capitalist materialism (Jones and Higgs 2010; Lafontaine 2009). This is of course at odds with the ‘lived experience’ of those suffering from the detriments of aging. Whether we live in a more individualist society or not, the experience of aging is a deeply individual one, not least because the very plasticity of aging explained above manifests at the level of the individual—and it is undeniably the case that for many but not all, the experience of aging is one of personal frustration and suffering.

It is this suffering that presents an ethical motivation to some biogerontologists and justification for supporting research and development in this area. Commentators may be inclined to detect causes for ethical concern in the underlying normative aesthetics, but to cast scientists who support working on aging with a view towards its eventual mitigation and abolition as unwitting cogs in a nefarious capitalist machine offers no useful foundation for different ethical decision making by the accused. Instead it would not seem unreasonable for an ethically motivated biogerontologist to feel a desire that aging research be developed into effective ‘anti-aging’ treatments. However, we accept that moral agents are challenged to consider not just the ethics of their actions but also the far reaching implications. Consequently, we will turn briefly first to discuss the ethical practicalities of implementing research findings, and then move to discuss some of the potential social consequences in case such implementation should ever come to pass.

13.3 Social Consequences

Every researcher needs to take account of where the research may ultimately lead to. The potential social, economic, psychological and practical implications of drastically increased lifespan have been subject to extensive deliberation. Common to these scenarios is an inevitable element of speculative conjecture. In the following, we will briefly consider the two of the most prominent ethical concerns about the social consequences of successful biogerontology: The ‘fairness objection’ and the ‘overpopulation’ argument.

Subsequently, given the ‘biogerontology perspective’ which we adopt here, we will address two further topics which are arguably of particular concern to scientists active in this field: The suggestion that a long life would lead to stagnant, less fulfilled lives and to diminished social progress and the accusation that research to abolish aging stigmatises and marginalises the elderly.

13.3.1 *Cost and Equity*

While it is difficult to foresee the costs of a hypothetical cure for aging, it has been assumed that such treatments will be expensive.

The basic premise is not uncontested. Firstly, any economic cost calculation also needs to take account of the economic benefit. Curing aging and extending healthy lifespan would be profitable for nations. Historically, the economic value of increased longevity from 1970–2000 was estimated at \$3.2 trillion per year for the US alone through increased productivity and significantly decreased healthcare costs, with economic gains from future health improvements also estimated to be large (Murphy and Topel 2006). In fact, effective anti-aging interventions are likely required to avert significant economic burdens associated with the current demographic situation (Aaron and Schwartz 2004).

Moreover, it could be suggested that even if curing aging is initially expensive, its universal desirability will help to recoup costs of investment relatively quickly, allow for low profit margins, create political and competition pressure to drive prices down, facilitate e.g. mass production and service infrastructures etc. as has happened in similar cases (Lucke et al. 2009).¹

Others would not wish to wait for such ‘trickle down’ effect or doubt its efficacy. Nonetheless, some commentators have identified the potential social inequities between those who may be able to afford effective anti-aging treatments and those who cannot pursue human life extension at all (Glannon 2002). To those voices, contemplating further life extension in an unequal world is ethical anathema (cf. Chap. 17). While the argument is usually put more eloquently (Pijnenburg and Leget 2007), it seems subject to distillation into the statement: ‘Healthcare in rich countries should not advance until the poorer countries have caught up’. Not only does this argument neglect the considerable burden of age-associated diseases already threatening to crush poor country economies (Smith and Mensah 2003), the underlying ideology would call for the cessation of any number of activities other than those directly aimed at improving life expectancy in poor nations (which are, it has been argued, not predominantly scientific but political barriers).

However, even if we assume for the sake of argument that life extension therapies will always be costly, it still remains questionable whether the notion of inequality is such a moral evil as to require “collective suttee” (Davis 2004). “We do not normally think it an ethical requirement to prevent good being done to some unless and until it can be done to all” (Harris 2002, p. 290). In transplantation medicine, for example, the availability of organs is a very real factor of life extension: Those lucky enough to be allotted an organ, survive much longer. Generally, society has been able to agree on laws that regulate the allocation of these special resources. We have not chosen to destroy all organs as they become available in order to preserve equality in despair. Similarly, if life extension treatments would turn out to be irrevocably scarce, this must not mean that only the ability to pay will be decisive. Treatment could be allotted

¹ For a different view, see chapter 19, this volume.

randomly (Broome 1984), as a social reward (Vance 1956), according to imminent need (Harris 1987), within a general utilitarian framework (Miller and Sethe 2005), or even as “affirmative prolongevityism” (Overall 2003). There is no intrinsic reason why such arrangements must be morally flawed if instituted in a society that can agree on an equitable system of apportionment.

In summary, upon closer examination the spectre of unequal availability cannot present an ethical barrier to biogerontology.

13.3.2 *Overpopulation*

The other main issue most frequently considered a social implementation objection centres on consequences for population growth.

It is also not always clear why a larger population is considered morally problematic, but most concerns seem to be based on the assumption that an ever greater population leads to a world that is ‘not worth living in’. Since Malthus raised his concerns, indeed since the doomsday scenarios were projected in the 1970s, population has increased manifold without any decline in living standards (Trewavas 2002). The question of population pressure is obviously defined by other characteristics than just life expectancy: birth rates, infrastructure, availability of resources, capacity to recycle waste, space management, land use, and concepts of privacy all play a role.

In popular discourse, overpopulation is seen as the root of many environmental problems and societal ills (Ehrlich 1968), but in many respects, moral issues in environmentalism are independent from future developments in population pressure. If we are using finite resources in a non-sustainable manner, then this problem needs to be solved independently of how long people live. Relying on death is not a very creative way to tackle such problems (More 2004) especially considering that population-linked doomsday scenarios have generally been dispelled by human ingenuity (Boserup 1981).

In concentrating on the underlying moral issues at play, we are challenged to question the relevance that these differing visions might have in the first place. If one decided that the vision of a crowded planet is too terrible to permit, what type of intervention should be adopted? Would we decline to invest in medical innovation? Withhold its use? Encourage suicide or sanction killings? In population ethics, one is precariously balancing the real interest of existing people against the hypothetical interests of those projected to be born (Parfit 1983) and potentially also balancing a hypothetical quality of life against the imposition of an early death (Davis 2005; Cutas 2008).

The instinct and desire to procreate is strong in many. This may be due to evolutionary reasons, but also a conscious decision to defy death by trying to perpetuate something of oneself—which indicates that such desires might be less strong in ‘immortals’ (Perry 2000). Some suggest a scheme where those who have become ‘immortalized’ could agree not to reproduce (Harris 2000). Apart from enforceability, one could regard this arrangement as troubling where it might lead to social

stagnation. Yet, at this point we really feel one enters such a complex area of pure speculation (scientific, economic, political, social, psychological and individual) that we fail to see how one could draw any ethical conclusions that should seriously undermine the moral imperative as we have outlined it to treat aging today. The question of imminence is valid: The most immediately effective way to control population pressures is to stabilise birth rates. Population changes are surprisingly slow in their response even to a dramatic life extension (Gavrilov and Gavrilova 2010). Even if a ‘cure’ for aging were developed tomorrow, the pressure of population whatever they may be would not amount to a marked increase for many decades. Thus if overpopulation becomes an issue it would be well into the next century or beyond. By then, social, technological and economic parameters are likely to be so different to render all anticipatory ethical concerns moot. Such an array of uncertainties surrounds potential population pressures that it seems presumptuous to preclude today the moral decisions that are the concern, privilege, and responsibility only of future societies. A biogerontologist needs to balance these considerations against the immediacy of alleviating age-associated suffering.

13.3.3 Stagnation

At first, a general fear of boredom may seem like one of the most trivial objections to increasing lifespan. In essence, it assumes that (a) patterns of experience will inevitably repeat and (b) that the ability to derive satisfaction from experience always diminishes in repetition. The somewhat cynical question of boredom alludes to a more serious issue in the longevity of the mind. Part of this argument seems to draw on the (usually unspoken) hypothesis that the human brain is not equipped to deal with vastly extended lifespans. It is common experience that personalities are essentially formed during childhood, whereas adults are often very set in their ways. This could mean that ubiquitous long life leads to an altogether less flexible and dynamic society, where the majority are less willing to change their outlook and convictions, where new culture and technology is stifled, strife and inefficiency perpetuated. There are good reasons to believe that some—if not the majority—of our decreasing ability to learn and adapt as we age is an artefact of brain aging (Lockett 2010) and may thus be subject to remedy of anti-aging treatments (Lynch 2011). In the long term, the ‘Markopoulos challenge’ is not such a trivial one, but requires a more nuanced approach to the psychology of boredom (Bortolotti and Nagasawa 2009).

Still, those who have spent a ‘lifetime’ developing a theory, following a creed, or hating an enemy are presumably less likely to change their mind than those younger and less encumbered by their past. As Max Plank suggests “A new scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die, and a new generation grows up that is familiar with the idea from the beginning” (Planck 1950). Yet the problem of such ossification is not confined to future technology. Similar considerations have inspired legislators to limit the term any one individual can spend in a position of power. To

rely on death as a driver of change is to take a very resigned view about our moral responsibility and capacity. Instead, we would agree that rather than dwelling on the ethics of variable aging it is important to address the actual “lived experience of those who engage, refuse to engage, or are unable to engage with these contested domains” (Jones and Higgs 2010, p. 1518).

Ultimately, it is intriguing to speculate that far from having reached its endpoint due to medical technology, evolution by natural selection would come into its own in these futuristic scenarios: Not only will there be strong dispositional selection pressure against those who reject such treatments for ideological reasons, it might well be the case that humanity will undergo a selection where those who can experience the greatest fulfilment from ongoing discovery will choose to live substantially longer, compared to other character types.

13.3.4 Stigmatising the Elderly

Sometimes hidden in some of the more obscure postmodernist critique of efforts to cure aging a particularly worrying allegation emerges that applies directly to the biogerontologist. In efforts to mitigate, abolish, cure, eradicate, defeat aging, are scientists forging a paradigm in which old people must feel as unfortunate failures? Conflating the disease with the sufferer would seem a straightforward fallacy to avoid, but we know from other situations in bioethics and beyond that in fact such issues need a very sensitive and empathetic link between the researcher and the researched. Stigmatization, ghettoization and loneliness of the elderly are a real problem already, but it is true that these could be exacerbated where a narrative of ‘successful aging’ casts off those who in some way ‘fail’ to evade it (Vincent 2006). However, this is precisely where biogerontology can make an ethical contribution: What commentators lamenting the impending alienation and stigmatization of the elderly often overlook is that there is in fact no ‘golden age’ where old people were treated fairly as research subjects or patients. Instead, we know that there is a paucity of research on age-related conditions and that sometimes even the most basic interventions are not applied to the elderly as they are ‘likely to die anyway’. Biogerontology can make a real contribution by challenging these assumptions.

A biogerontologist wishing to cure aging will be encountering those who share this aspiration but for whom no feasible treatment exists as well as those who reject the notion and feel shaken by the mere suggestion that what is happening to them may not be ‘normal’. Either situation calls for tact, respect, and sympathy. Neither situation will be trivial to navigate ethically. It is this kind of interaction between moral agents that brings us full circle back to what was called the ‘inner circle’ ethics in the introduction: Away from the flights of fancy that ethicists sometimes posit, the ‘social consequences’ of biogerontology are being shaped not (only) by the sinister workings of materialist modernity, but by the daily contact between people trying to make sense of their lives and trying to relate to each other with respect and sympathy.

13.4 Conclusion

Here, we have aimed to discuss some ethical issues in biogerontology from the perspective of biogerontology research. We have shown that, by assuming this perspective, certain ethical ‘dilemmas’ may appear less immediately relevant whereas others come into sharper focus.

On the one hand, research shows that aging is flexible, subject to intervention, amelioration and modulation. No ethical case can be made for denying or suppressing this fact. Aging is associated with evils such as grief, suffering, loss of dignity and loss of freedom. Worries about long term social consequences not only lack evidence, they fail in the face of the immediate ethical challenge. Efforts to hide this fact or to justify its existence on abstract philosophical or ideological grounds are ultimately immoral if they stand in the way of effective action in research or treatment.

However, it is also true that aging and death will remain a ‘fact of life’. This opens up a gap, a space for ethical deliberation and concern. The real ethical challenge is to identify involuntary aging as ‘the enemy’ while not abandoning or belittling the fate of those who are nonetheless subject to aging.

In summary, protagonists in biogerontology are subject to three ethical imperatives: (1) to represent the potential of aging research without hype but also without unwarranted political constraint; (2) to face the fact that aging causes suffering whereas the putative social drawbacks of controlling aging are speculative and contested; (3) to tackle the challenge of ‘fighting aging’ without fostering ageism.

References

- Aaron, HJ and WB Schwartz, eds. 2004. *Coping with methuselah: The impact of molecular biology on medicine and society*. Washington: Brookings Institution Press.
- Bortolotti, L., and Y. Nagasawa. 2009. Immortality without boredom. *Ratio* 22(3): 261–277.
- Boserup, E. 1981. *Population and technological change: A study of long-term trends*. Chicago: University of Chicago Press.
- Broome, J. 1984. Selecting people randomly. *Ethics* 95: 38–55.
- Browner, W.S., A.J. Kahn, E. Ziv, A.P. Reiner, J. Oshima, R.M. Cawthon, et al. 2004. The genetics of human longevity. *American Journal of Medicine* 117(11): 851–860.
- Butler, R.N., H.R. Warner, T.F. Williams, S.N. Austad, J.A. Brody, J. Campisi, et al. 2004. The aging factor in health and disease: The promise of basic research on aging. *Aging Clinical and Experimental Research* 16(2): 104–11.
- Caplan, A.L. 2005. Death as an unnatural process. *EMBO Report* 6(S1): S72–S75.
- Clark, P.G. 1993. A typology of interdisciplinary education in gerontology and geriatrics: Are we really doing what we say we are?. *Journal of Interprofessional Care* 7: 217–227.
- Crimmins, E.M., H. Beltrán-Sánchez. 2011. Mortality and morbidity trends: Is there compression of morbidity?. *Journals of Gerontology* 66B(1): 75–86.
- Cutas, D.E. 2008. Life extension, overpopulation and the right to life: Against lethal ethics. *Journal of Medical Ethics* 34(9): e7.
- Davis, J.K. 2004. Collective suicide: Is it unjust to develop life extension if it will not be possible to provide it to everyone?. *Annals of the New York Academy Sciences* 1019: 535–541.

- Davis, J.K. 2005. Life-extension and the malthusian objection. *The Journal of Medicine and Philosophy* 30: 27–44.
- de Grey, A., and M. Rae. 2008. *Ending aging: The rejuvenation breakthroughs that could reverse human aging in our lifetime*. New York: St. Martin's Press.
- de Grey, A.D.N.J. 2004a. Escape velocity: Why the prospect of extreme human life extension matters now. *PLoS Biology* 2(6).
- de Grey, A.D.N.J. 2004b. Biogerontologists' duty to discuss timescales publicly. *Annals of the New York Academy of Sciences* 1019: 542–545.
- de Grey, A.D.N.J. 2007. Understanding and tackling aging: Two fields communicating (A little) at last. *Rejuvenation Research* 10(4): 637–640.
- de Grey, ADNJ. 2006. Compression of morbidity: The hype and the reality, part 2. *Rejuvenation Research* 9(2): 167–8.
- de Grey: ADNJ. 2005. Resistance to debate on how to postpone aging is delaying progress and costing lives. *EMBO Reports* 6(S1): S49–S53.
- de Magalhaes, J.P. 2011. The biology of aging: A primer. In *An introduction to gerontology*, ed. I. Stuart-Hamilton, 21–47. Cambridge: Cambridge University Press.
- de Magalhaes, J.P., and G.M. Church. 2005. Genomes optimize reproduction: aging as a consequence of the developmental program. *Physiology (Bethesda)* 20: 252–259.
- de Magalhaes, J.P., A. Budovsky, G. Lehmann, J. Costa, Y. Li, V. Fraifeld, et al. 2009. The human ageing genomic resources: online databases and tools for biogerontologists. *Aging Cell* 8(1): 65–72.
- de Magalhaes, J.P., D. Wuttke, S.H. Wood, M. Plank, and C. Vora. 2012. Genome-environment interactions that modulate aging: powerful targets for drug discovery. *Pharmacological Reviews* 64: 88–101.
- Ehrlich, P.R. 1968. *The population bomb*. New York: Ballantine.
- Farelly C. 2010. Why aging research? The moral imperative to retard human aging. *Annals of the New York Academy of Science* 1197: 1–8.
- Finch, C.E. 1990. *Longevity, senescence, and the genome*. Chicago: The University of Chicago Press.
- Fishman, J.R., R.H. Binstock, M.A. Lambrix. 2008. Anti-aging science: The emergence, maintenance, and enhancement of a discipline. *Journal of Aging Studies* 22(4): 295–303.
- Flurkey, K., J. Papaconstantinou, and D.E. Harrison. 2002. The snell dwarf mutation Pit1(dw) can increase lifespan in mice. *Mechanisms Ageing and Development* 123(2-3): 121–130.
- Fontana, L., L. Partridge, and V.D. Longo. 2010. Extending healthy lifespan—from yeast to humans. *Science* 328(5976): 321–326.
- Fries, J.F. 2003. Measuring and monitoring success in compressing morbidity. *Annals of Internal Medicine* 139: 455–459.
- Fries, James F. 1983. The compression of morbidity. *Milbank Memorial Fund Quarterly* 61: 397–419.
- Fukuyama, F. 2002. Our posthuman future. *Farrar, Straus and Giroux*.
- Gavrilov, L.A., N.S. Gavrilova. 2010. Demographic consequences of defeating aging. *Rejuvenation Research* 13(2–3): 329–334.
- Gems D. 2011. Tragedy and delight: the ethics of decelerated aging. *Philosophical Transactions Rhode Social* B366: 108–112.
- Glannon, W. 2002. Reply to harris. *Bioethics* 16: 292–297.
- Harris, J. 1987. QALYfying the value of life. *Journal of Medical Ethics* 13: 117–123.
- Harris, J. 2000. Intimations of immortality. *Science* 288: 59.
- Harris, J. 2002. Response to glannon. *Bioethics* 16(3): 284–291.
- Harrison, D.E., R. Strong, Z.D. Sharp, J.F. Nelson, C.M. Astle, K. Flurkey, et al. 2009. Rapamycin fed late in life extends lifespan in genetically heterogeneous mice. *Nature* 460(7253): 392–395.
- Jones IR, Higgs PF. 2010. The natural, the normal and the normative: Contested terrains in aging and old age. *Social Science and Medicine* 71(8): 1513–9.

- Jones, I.R., and P.F. Higgs. 2010. The natural, the normal and the normative: Contested terrains in aging and old age. *Social Science and Medicine* 71(8): 1513–1519.
- Kenyon, C.J. 2010. The genetics of aging. *Nature* 464(7288): 504–512.
- Kirkwood, T.B. 2005. Understanding the odd science of aging. *Cell* 120(4): 437–447.
- Kurzweil, R., and T. Grossman. 2004. *Fantastic voyage: Live long enough to live forever*. Emmaus, PA: Rodale.
- Lafontaine, C. 2009. Regenerative medicine's immortal body: From the fight against aging to the extension of longevity. *Body and Society* 15(4): 53–71.
- Lim, E.L., K.G. Hollingsworth, B.S. Aribisala, M.J. Chen, J.C. Mathers, and R. Taylor. (in press). Reversal of type 2 diabetes: normalisation of beta cell function in association with decreased pancreas and liver triacylglycerol. *Diabetologia*.
- Lockett, G.A., F. Wilkes, R. Maleszka. 2010. Brain plasticity, memory and neurological disorders: An epigenetic perspective. *NeuroReport* 21(14): 909–913.
- Lucke, J.C., P.C. Diedrichs, B. Partridge, W.D. Hall. 2009. Anticipating the anti-aging pill. Lessons from the history of the oral contraceptive pill and hormone replacement therapy. *EMBO Reports* 10(2): 108–113.
- Lynch, G., L.C. Palmer, C.M. Gall. 2011. The likelihood of cognitive enhancement. *Pharmacology Biochemistry and Behavior* 99(2): 116–129.
- Miller, J., S. Sethe. 2005. Gods with a limited budget: putting the utility back into utilitarian health politics. *Interdisciplinary. Science Reviews* 30(3): 273–278.
- More, M. 2004. Superlongevity without overpopulation. In *The scientific conquest of death—essays on infinite lifespans; librosEnRed*, ed. B.J. Klein, S. Sethe, et al., 169–185.
- Murphy, K.M., and R. H. Topel. 2006. The value of health and longevity. *Journal of Political Economy* 114(5): 871–904.
- Nadon, N.L., R. Strong, R.A. Miller, J. Nelson, M. Javors, Z.D. Sharp, J.M. Peralba, D.E. Harrison. 2008. Design of aging intervention studies: The NIA interventions testing program 2008. *Age* 30(4): 187–199.
- Olshansky, S.J., L. Hayflick, and B.A. Carnes. 2002. No truth to the fountain of youth. *Scientific American* 286(6): 92–95.
- Overall, C. 2003. *Aging, death, and human longevity: A philosophical inquiry*. Berkeley: University of California Press.
- Parfit, D. 1983. *Reasons and persons*. Arlington: Clarendon.
- Partridge, L., J. Thornton, and G. Bates. 2011. The new science of aging. *Philosophical Transactions Rhode Social* B366: 6–8.
- Perry, M. 2000. *Forever for all: Moral philosophy, Cryonics, and the Scientific Prospects for Immortality*. Boca Raton, FL: Universal Publishers.
- Pijnenburg MA, C. Leget. 2007. Who wants to live forever? Three arguments against extending the human lifespan. *Journal of Medical Ethics* 33(10): 585–7.
- Planck M. 1950. *Scientific autobiography and other papers*. London: Williams and Norgate.
- Rae MJ, RN Butler, J Campisi, AD de Grey, CE Finch, M Gough, GM Martin, J Vijg, KM Perrott, BJ Logan. 2010. The demographic and biomedical case for late-life interventions in aging. *Science Translation Medicine* 2(40): 40cm21.
- Rehmann-Sutter, C. 2011. Aging as disease?. *Swiss Medicine Weekly* 141.
- Richel, T. 2003. Will human life expectancy quadruple in the next hundred years? sixty gerontologists say public debate on life extension is necessary. *Journal of Anti-Aging Medicine* 6(4): 309–314.
- Sames, K. 2005. Aging as a consequence of organ differentiation. In *Extending the lifespan: Biotechnical, gerontological, and social problems; LIT*, ed. K Sames, S Sethe, A Stolzing.
- Smith, S.M., G.A. Mensah. 2003. Population aging and implications for epidemic cardiovascular disease in sub-Saharan Africa. *Ethnicity and Disease*, 13(2 SUPPL. 2): S2–77–S2-80.
- Stipp, D. 2010. *The youth pill: Scientists at the brink of an anti-aging revolution*. London: Penguin Books.
- Trewavas, A. 2002. Malthus foiled again and again. *Nature* 418: 668–670.

- Underwood, M., H.P. Bartlett, W.D. Hall. 2009. Professional and personal attitudes of researchers in aging towards life extension. *Biogerontology* 10(1): 73–81.
- Vance, J. 1956. *To live forever*. New York: Ballantine.
- Vincent, J.A. 2006. Aging contested: Anti-aging science and the cultural construction of old age. *Sociology* 40(4): 681–98.
- Warner, H., J. Anderson, S. Austad, E. Bergamini, D. Bredesen, R. Butler, et al. 2005. Science fact and the SENS agenda. What can we reasonably expect from aging research?. *EMBO Report* 6(11): 1006–1008.