

The foreseeability of real anti-aging medicine: focusing the debate

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Abstract

There has recently been a sharp and very welcome increase in the rate of appearance of articles discussing the concept of medical interventions that would greatly increase the maximum healthy human lifespan. Much of this literature has emphasised the current non-existence of any such therapies, and has done so with laudable accuracy and authority. Regrettably, however, such articles have frequently extended their ambit to include the issues of how soon such interventions could be developed and of how advisable such an effort would be anyway, and have addressed these much more weakly, thereby diminishing the force of their main message. Here a survey is made of the more conspicuously flawed arguments suggesting tremendous difficulties or dangers in developing such interventions, with the aim thereby to tow those arguments firmly out into the ocean and give them the decent but unambiguous public burial that they so richly deserve. It is hoped that, by clearing the debate on future anti-aging advances of these obfuscations, the many aspects of this topic that have hitherto received much less attention than they warrant will be brought to the fore.

Introduction

Biogerontologists naturally find it expedient to identify their topic as a clearly distinct subfield within biology, and also within medicine. This led, long ago, to a widespread adoption of the mantra that “aging is not a disease” (Hayflick, 1994). It remains controversial—though perhaps not productively so—whether this statement is actually true. What is of greater concern, however, is the truth or otherwise of an assertion easily inferred from it: that “aging cannot be cured”. Since aging cannot *currently* be cured, despite this having been a widespread aspiration of humanity for at least the whole of recorded history, it is natural for non-specialists to make this extrapolation. Unfortunately, it has also often been made by gerontologists themselves, on the basis of extraordinarily ill-founded arguments. The persistence of such arguments, historically perhaps just an irritation, is now becoming highly problematic as technologies emerge that allow the rational design of such interventions—i.e., of what we can reasonably term “real anti-aging medicine”—because they divert many scientists from the detailed, goal-directed efforts that are critical to the translation of that potential into reality.

Accordingly, here I dissect several such arguments, spelling out exactly why the time when they had any cogency, if it ever existed, is long gone. Additionally I address, with a similar goal, several sociological arguments which powerfully reinforce the scientific arguments by undermining criticism of them.

“There’s no evidence that aging can be appreciably altered”

“Appreciably” in this assertion can only mean “much more than we can do yet”. The assertion is thus, in an important sense, vacuous: all it says is that progress occurs at a finite rate, such that it will be some time before we can do much more than we can now. But that is not the assertion’s main flaw. In claiming the lack of evidence for the possibility of a technological advance, one is mixing apples and oranges: using the term “evidence” as would be appropriate in science, but applying it to technology (Le Bourg, 2003; de Grey, 2003c). In science, we explore the truth or falsehood of hypotheses. What, then, is the hypothesis “aging cannot be appreciably altered” embodied in the above assertion? All it can be is:

“No non-aging complex animal can exist, even with arbitrarily advanced technology to keep it so”. This is plainly false.

The term “evidence” is perfectly applicable to technology, however: it merely means something subtly different than in basic science. Evidence can certainly exist for or against the feasibility of a *particular approach* to solving a technological problem—whether the approach can be implemented, and whether it will work. With this usage in mind, we can certainly ask useful questions about the foreseeability of real anti-aging medicine; but those are not the questions that those who proffer the “evidence” argument have in mind, since they rarely discuss particular approaches.

“Evolution is cleverer than you are”

Or, to put it less succinctly than Crick (Dennett, 1984): “How dare you suggest we could cure aging when there are no non-aging mammals? You obviously have no conception of how indescribably ingenious evolution is, and hence of how hard something must be if evolution has failed to do it.”

This assertion gives a whole new meaning to the title of Tom Kirkwood’s talk at the 2002 European Congress of Biogerontology, “25 years of being disposable”: it demonstrates that Kirkwood’s “disposable soma theory” (Kirkwood, 1977) remains widely unappreciated. Kirkwood [and, in a more limited form, Edney and Gill a decade earlier (Edney and Gill, 1967)] noted, of course, that the rate of aging is determined above all by the rate of extrinsic mortality from predation, starvation, etc., and furthermore that even a complete absence of extrinsic mortality would not lead, in organisms with body plans anything like our own, to an absence of aging. It is simply not economical to maintain the species indefinitely by maintaining the organism indefinitely, when there exists the (less attractive from our point of view, but not from evolution’s) alternative of periodic reproduction. [The qualification regarding body plans is necessary because Kirkwood’s logic assumes a cost, in increased sophistication of maintenance and repair, associated with bearing down on the rate of aging. In very simple organisms such as *Hydra*, however, which possess no indefinitely long-lived cells, that relation is asymptotic, so that maintenance sufficient to preserve the organism for a few weeks will also preserve it indefinitely (Martinez, 1998). Indeed, this is also why the germ line itself escapes Kirkwood’s logic.]

As in the previous section, the real problem with the assertion under discussion is not that it is incorrect in essence but that it is inappropriately applied. Of course evolution is cleverer than we are, and will be for much longer than the foreseeable future. In seeking a cure for aging, therefore, we are wise to exploit evolution’s ingenuity. But it is quite unnecessary to restrict ourselves to its efforts to solve the same *overall* problem that we seek to solve [as some, including those whose timescales for doing exactly that I consider wildly *over-optimistic* (Miller, 2002), have implicitly assumed]. First, evolution has not tried at all hard to solve it; second, it can be broken down—by us—into sub-problems that evolution has solved for different reasons entirely, just as bacteria do not possess restriction enzymes to perform microscopic Southern blots, and just as the Wright brothers did not exploit the physics of flapping. See Table 1.

Reversal of life-limiting change	Feasible way to achieve it
Digestion of lysosomal aggregates	Bacterial/fungal hydrolases
Apoptosis of senescent cells	Senescence marker-targeted toxins
Killing of age-related tumours	Total telomerase deletion plus cell therapy
Nuclear rescue of mtDNA mutations	Allotopic mt-coded proteins (copying algae)
Immune system restoration	IL-7-mediated thymopoiesis
Cleavage of AGE cross-links	Phenacyldimethylthiazolium chloride

Clearance of extracellular aggregates	Immune-mediated phagocytosis
Cell replacement and regrowth	Stem cell therapy, growth factors
Hormone restoration	Genetically engineered muscle

Table 1. SENS (strategies for engineered negligible senescence). Interventions to reverse aging in rodents and subsequently humans. For details, see (de Grey et al., 2002a, 2002b; de Grey, 2003a, 2003b).

“There’s so much in biology that we don’t remotely understand”

This assertion differs from the previous one in emphasising our lack of knowledge rather than of ingenuity. It is somewhat rebutted by the arguments already proffered, but a more direct response is warranted. For millennia we have been able to augment the endogenous repair and defence processes on which other animals entirely rely, by actions determined by intelligent analysis and experimentation—to wit, medicine. In the past 200 years, the scope of our ability to cheat nature in this way has massively accelerated; the germ theory (Schwarz, 2001), with its consequences both in the development of drugs and in the appreciation of the indispensable benefits of hygiene, is the most conspicuous of innumerable examples. Yet, our endogenous defences against disease are awesomely complex; though the immune system is the focus of research by huge numbers of scientists, our understanding of it shows no sign of nearing what could remotely be described as completion. Clearly aging is harder to combat than most early-onset diseases—otherwise we would have cured it already—but the suggestion that it is *qualitatively* harder lacks the slightest basis.

“Organisms have biological warranty periods”

This is the most recent (Carnes et al., 2003) wording of the inference that, because (like that of machines) aging of organisms is a product of neglect rather than “deliberate” design, we can never ward it off indefinitely. Hayflick, in an earlier statement of the same idea (Hayflick, 1994), compared nature’s construction of organisms with a certain “longevity determination” to NASA’s construction of satellites.

Here the error is in forgetting that machines’ effective warranty periods depend not only on how well they were built but on how well they are maintained. The reason we can keep *Hydra* alive forever is that they need only a modest amount of help to get to the (finite) level of maintenance that stops them from accumulating any deleterious changes at all. This level is finite for other species too—merely a lot greater. It takes a lot of work to keep a car on the road indefinitely, but vintage car races are frequent reminders that this is not beyond us. The error in comparison with satellites is that no maintenance of them is possible.

It is also incorrect to suggest that the “warranty period” idea applies more ineluctably to humans in the industrialised world because their mortality is dominated by senescence, rather than age-independent causes. That is simply because the things that kill us these days happen mainly to be processes with a progressive course, which do not harm us until they have accumulated to a certain level. This says nothing about whether or not the periodic reversal of such damage would keep us alive and healthy.

“Your proposed therapies are far too crude to be effective”

As an energetic designer and advocate of interventions to reverse key candidate components of mammalian aging (de Grey, 2000a, 2002), I frequently encounter this criticism. However, it overlooks a direct corollary of the “evolution is clever” point that the same critic typically makes: namely, that organisms (especially long-lived ones) possess homeostatic machinery of a sophistication whose surface we have hardly scratched. I do not remotely dispute that the therapies I advocate are crude—very crude. They would have side-effects, without doubt. But would those side-effects be pathogenic? The answer,

by definition, is: “Only if our endogenous homeostatic mechanisms could not eliminate them”. But in this reply is embodied the fact that our endogenous sophistication is working *with* us, not against us, to preserve tissue and organismal integrity indefinitely. If we do something that restores the body to a broadly more youthful state, but also inflicts damage that is not characteristic of aging, we are essentially turning a healthy old person into a sick young person. Which of those people typically lives longer? The sick young one does, because s/he is well equipped to recover from the sickness.

“Your proposed therapies are far too ambitious to be developed”

This challenge is directed at me particularly often because all the types of intervention that have occupied me in print are indeed highly ambitious. However, this is no accident. I have periodically enumerated the likely components of a truly effective panel of interventions into aging (de Grey et al., 2002a, 2002b; de Grey, 2003a, 2003b), and noted that several of them are already being expertly and energetically pursued by others, along lines of research that I consider likely to be effective. I have therefore busied myself with those areas that are being addressed less well at present. Since biologists tend not to neglect easy problems, “my” areas are naturally the most challenging and requiring of outrageous thinking.

I use the word “outrageous” advisedly. Since I lack experimental training, a natural reaction from experienced bench scientists to my more radical proposals is that nobody who knew how hard experiments are would give them a second’s thought. I am highly sympathetic to this reaction; far from underestimating the difficulty of bench work, I am in unalloyed awe of anyone who can reproduce a result. However, the intuition of experimentalists concerning how hard a particular proposal would be to implement is often unreliable—and I say this with the defence of an objective measure. All the technologies on which I have focused have several parts, each with its own difficulties. Some of those components have already seen substantial progress; others have not. When I present the overall concept to a gerontologist colleague for the first time, it is positively the norm that the aspects they assert to be the hardest are ones that have in fact already been implemented, but reported in literature too distant from gerontology to have come to my critic’s attention (e.g. Gershon, 1997; de Grey, 1997). In summary, and confrontational though it may at first seem coming from a theoretician, this challenge to the foreseeability of real anti-aging medicine is in my experience ultimately founded on neglect of the relevant experimental work.

“Don’t discuss reversing mouse aging: first let’s halve its rate, which must be far easier”

This is another case where terminology seduces the critic into neglecting the relevant biology. If one defines “retarding aging” in the abstract, in terms purely of the trajectories of remaining healthy life expectancy in the presence and absence of the intervention in question, then retarding aging must indeed be easier than reversing it, since any aging-reversal technology could be applied less aggressively (and hence, we can accept, less effectively) against a continual onslaught of pro-aging phenomena (to wit, aging itself) and thereby retard aging by reversing it more slowly than it is occurring. But that misses the point.

Suppose you are in a small dinghy, in the middle of the ocean, and a leak appears. You will sink before help arrives if you do nothing, and you have few tools. You could halve the rate of entry of water by rudimentary plugging of the leak or by bailing water out of the boat, but this is not good enough to save you. But suppose you have a companion, who can double the quality of either the plugging or the bailing: the former type of assistance will double your survival time, but the latter will keep you afloat forever. In other words, there are two sorts of retardation of a process: ones that can be converted to reversal of the process merely by a quantitative enhancement, and ones that can never cross that threshold however much faster they are performed. The former type is bound to be easier than reversal—but only marginally so, since quantitative enhancements tend to be easy. The latter may be easier, or it may not.

“Well”, the critic retorts, “OK, in principle retardation techniques of the ‘plugging’ sort might be no easier than those of the ‘bailing’ sort, but in practice I bet they will be far easier.” But this is easy to answer: we need only examine what technologies are prerequisites of each approach. There are, to be sure, approaches to retarding aging that are technically quite easy—small molecule drugs, for example—but it is not disputed (not, at least, by those who would make the assertion under discussion in this section) that these are only minimally effective and are likely to remain so. Any approach to seriously retarding aging (of already-alive mammals, which are our main short-term concern, as will be discussed below) will need somatic gene therapy. And once we acknowledge the necessity of that, we concede the major technical obstacle to aging-*reversal* technologies (de Grey et al., 2002a). Hence, once the details are examined, the inescapable conclusion emerges that reversing aging should be almost exactly as hard as retarding it.

“Replacing brain cells would endanger continuance of identity”

Wholesale replacement of the brain, in one go, would clearly constitute a discontinuity in the cognitive state of the recipient too drastic to be contemplated as an attractive medical intervention. Whether the person is the same person after such an operation is, in practice, moot: even if we persuade ourselves intellectually that they are, the extent to which they have become a different *version* of the same person is so severe as to be beyond what we are interested in bringing about—not least given the minimal degree to which we can (in the foreseeable future, anyway) determine the structural basis of someone’s individual cognitive state, let alone recreate it in a new brain.

However, this is but one end of a spectrum of procedures, the other end of which is equally imaginable—and indeed occurs naturally. The brain, like the rest of us, continually destroys and regenerates cellular components, from the smallest organic molecule to enormously complex organelles such as mitochondria. The same is true of supracellular aspects of neural structure, such as synaptic connections between neurons and even loss of neurons. Processes of this sort are—by elimination, for what other candidate is there?—the underlying cause of our progression through life in terms of our attitudes, behaviour and so on, and also of (for example) our inability to recall facts that we know we could recall perfectly 20 years ago. None of these changes in our cognitive state gives us the faintest cause for hesitation when asked whether we are the same person as back then.

This brings us back to the assertion at the head of this section. Since one-step brain replacement does indeed compromise identity, but replacement one molecule or organelle at a time [even at a rate that means, for example, every mitochondrion in the brain being replaced once a month or so (Menzies and Gold, 1971)] does not, there must be a threshold granularity of replacement below which identity is entirely preserved. Is that granularity exceeded by the cell therapy presently being pursued to treat Parkinson’s and other diseases associated with neuron loss (Bjorklund, 2000)? I have scoured the literature for reports that beneficiaries of such therapy are protesting that it is interfering with their identity, but without success.

“It’s more important to compress morbidity”

We move now to arguments that seriously extending lifespan, feasible or not, is an improper focus. I omit here those arguments based on principle—religious and other standpoints to the effect that we should not develop such technology even if we will never know exactly why we shouldn’t have done so. I confine the remainder of this article to challenges founded upon sociological concerns.

The problem with a focus on compressing morbidity is not, of course, that there is anything to be said in favour of morbidity. Interventions that increased healthy lifespan without similarly raising total lifespan would be unconditionally welcome. The only real controversy here concerns the idea that one’s loved ones need time to get used to one’s impending demise, and I contend that this “need” really only exists

even now when the death in question is unusually young. (I thank Judith Campisi for her suitably severe reaction to my originally contrary position.) No: the problem is that compressing morbidity is impossible. “Impossible?!”, the critic cries. “How dare you say that something so modest is impossible, while also saying that a hugely more ambitious advance, to wit actually reversing (curing) aging, is foreseeable?!”

This objection, solid though it may seem, collapses immediately we consider exactly how “compressing morbidity” is defined. It means reducing the interval between the onset of morbidity and its conclusion (i.e., death). Thus, it can be achieved only by delaying the onset of morbidity *and not* similarly delaying death. The latter criterion is the problem. It is not that any particular technical advance is impossible, but that the requirement is to *avoid* making certain advances despite making other ones. And unfortunately, society has long demonstrated its obstinate determination to extend total lifespan as far as it can, and to put at least as much effort into doing so as into postponing morbidity. Add to this the simple biological truism that being healthy is not risky—i.e., that interventions to extend healthspan will tend to extend total lifespan by a similar amount even *without* medical advances to keep frail people alive—and it is inescapable that a focus on compressing morbidity is, and will forever remain, quixotic. Stark confirmations of this abound: see, for example, analysis of statistics of hospitalisation (Himsworth and Goldacre, 1999) and activities of daily living (Manton et al., 1997; Hodes, 2001) in (de Grey et al., 2002b), and also (Olshansky et al., 1991).

This sombre conclusion is no basis for despair, however. A potential solution exists: a way to diminish, sharply, the proportion of individuals who are suffering age-related disability. Once we develop true aging-reversal therapies, ones that can restore people from a frail state to a robust one, all the above logic collapses. Morbidity is implicitly defined as a *permanent* loss of health; thus, in a sense it is instantly eliminated with the development of such therapies. Perhaps this does not really occur until these therapies are universally available (something addressed below), but the difference is just a matter of time.

“The population crisis would be unmanageable”

The first point to stress is that it would be many decades, if ever, before even a complete cure for aging exerted an effect on global population that compared with acknowledged uncertainties inherent in our ignorance of future birth rates. The United Nations maintains a range of projections of global population out to 50 years in the future, and even though none of these projections assumes any dramatic progress in extending lifespan they vary by more than three billion depending on quite modest differences in projected birth rate (United Nations, 2001, Figure V.4). Yet, by 2050 the effect of a total cessation of death of those over 60—even starting today!—would be under two billion (United Nations, 2001, Table II.25). This figure rises rapidly, of course, for more distant projections—but so does the uncertainty in birth rate.

A variant of this argument is as follows: though society would have a long time to prepare for the inexorable population increase that would eventually result from totally curing aging, we would have to cope with it then, so it is not too soon to think about it and perhaps modify present actions in consequence. There are two errors here. Firstly, the only modification that could possibly result from such rumination is a delay in developing life-extension therapies. But it is abundantly clear that society lacks interest in drawbacks of its actions that would not materialise for several decades: one need only consider the entrenched apathy that we exhibit concerning climate change and other environmental matters. Hence such rumination seems quite worthless. Secondly, one reason underlying this apathy contains a germ of truth: our ability to meet challenges that we face in the distant future as a result of present actions is unknown, but is sure to be much more advanced than we can accurately imagine. Public apathy about the distant future is in this sense no different than not bothering to make advance arrangements for ground transport at the far end of a long-haul flight: the view that “something will turn up” is somewhat justified.

Finally we must remember that we will face this crisis only when we have been living for some decades with all the other societal consequences of the defeat of aging, which will surely be profound. We simply have no idea whether, for example, we will still think it remotely onerous to forego children indefinitely. The astounding decline in birth rate associated with every single example of a country achieving what we now call “developed” status (Keyfitz, 1996) points clearly in this direction: the supposed biological urge to procreate seems much more ephemeral than any 19th-century biologist or psychologist would have credited.

“Tyrants will reign forever; treatments will only be for the rich”

These are two of the more dystopic predicted societal consequences to which I alluded above. There is a very good reason to point out these dangers, but it is not the one stressed by those who typically do so.

The warning implicit in such forebodings (Hayflick, 1994, 2000; Holliday, 1995) is that, therefore, we must avoid such disasters by not curing aging in the first place. The myopia of such a view is considerable, since it overlooks the possibility that those with asocial inclinations might develop such technology themselves—and that, if they did so, they would inevitably have greater control over its availability than if others developed it first. The best defence against such dangers is, therefore, for benevolent sections of society (i.e., most of it) to get on and cure aging as soon as possible. No, the real reason to consider those dangers is this: given the certain eventual arrival of such technology, the undesirable social consequences that we may anticipate will be best avoided if we identify them and do all we can to pre-empt them. It may reasonably be argued that the time to begin this forward planning is now, and hence that the irresponsible gerontologist is the one who delays that planning by downplaying the plausibility of its premise.

“We must say nothing to cause optimism until we succeed in mice”

This brings us to possibly the most serious absentee from the current debate on real anti-aging medicine. Forward planning for something that we can be confident is at least 20-30 years away is of doubtful value, but when the timescale is quite probably only 10-15 years and could be as few as 7-8, the imperative to anticipate the event is clear. The longer timeframe applies to serious human life-extension advances—even to their development, let alone their widespread availability. But the shorter one applies to advances in extending mouse lifespan by an order of magnitude more than we can so far (de Grey et al., 2002a). I do not mean that we will by then be able to perform genetic interventions that extend mouse lifespan by ten years or more (contemporary manipulations achieve about a year), but what we probably will develop are late-onset interventions (say, imposed only at 75% of life expectancy) that increase mouse lifespan by ~2 years, whereas at present there is no way to add even two months with an intervention begun that late.

The reason this must concern us becomes obvious when we ask what the public reaction would be to such advances (de Grey et al., 2002b; Harris, 2000). We delude ourselves if we claim that laypeople currently think they—or even their children—will benefit personally from real anti-aging medicine. Documentaries on the biology of aging are popular in the same way that Star Trek is popular: fascinating, entertaining, but with exactly zero impact on people’s perception of their own lives. If you doubt this, just consider how many viewers respond to such programmes by altering their life insurance or pension arrangements.

But would this detachment survive the creation of mice such as those described above? It seems abundantly clear to me that there is a threshold of impressiveness of laboratory—not medical—advance that will tip public opinion over the edge into acknowledgement that they stand a real chance of personal benefit from the future translation of such work to the clinic. That advance will be more impressive than it should logically need to be, because the fear of being just a little too old to “make the cut” generates a tremendous reluctance to raise one’s hopes for fear of having them dashed. But that very inertia in public anticipation means that, when the wall of contemporary denial is finally breached, it will crumble to

nothing all the more precipitously. And when it does, the consequences will quite clearly not be restricted to life insurance decisions: they will encompass all manner of life *prolongation* decisions, as people seek to maximise their chance of surviving to benefit from such technology. The results will include difficulties in hiring people into vital but risky professions, and in adjusting the economy to support greater expenditure on health care at the expense of other areas. The political fallout will be even more destabilising: it will include, above all, an imperative to maximise national resources so as to expedite the availability of such therapies, even if this entails internationally unpopular (if I may employ classic British understatement) actions. Forward planning for this transition seems advisable (de Grey, 2000b; de Grey et al., 2002b).

When, then, will that breach occur? I claim no expertise in group psychology, but my intuition is that there is a big difference in attention-grabbing capacity between doubling a nematode's life expectancy and doubling a mouse's. Further, this difference is greatly amplified when the "late-onset" component is added to the argument. Unless and until there is a really dramatic breakthrough in somatic gene therapy technology, germ-line interventions in mice will still be a far cry from somatic interventions in humans, so germ-line-engineered mice might be insufficiently impressive; and the scale of progress in mice specified above will certainly need genetic manipulations. But researchers can happily implement them by somatic gene therapy of (middle-aged or older) mice—a technique that can, of course, be pursued with decidedly less caution concerning safety than it can in humans, and may thus arrive much sooner than in humans.

“Failure would rebound as loss of funding”

Some gerontologists feel that the difficulty of developing a compelling forebear of real anti-aging medicine (such as the mice discussed above) is so great that even the most cautious public statement concerning timescales is worse for public attitudes, and hence for the field in the longer term, than no such statement at all. My complaint is not at this reasoning: I challenge only the initial assessment of likely timeframes, not the pronouncements discouraged in consequence of that assessment. But the funding-fear argument is based on a wholly different logic: that even if one *does* feel that major advances in curing mouse aging are only a decade or so away, one should *still* not say so, because if those timescales slip then the disparity between prediction and actuality will induce funders to divert their benevolence to higher-achieving areas.

Leaving aside the question whether this is a risk worth taking, in view of the phenomenal benefits to humanity of success, we can demolish this view purely by consideration of appropriate precedent. The fields generally cited as portending this future funding crisis are ones not connected with health, such as artificial intelligence [which did indeed suffer severely in this way some decades ago (Lighthill, 1973)]. But medical precedent is plainly a more reliable indicator of whether this would happen with work on curing aging. Is there such a precedent? Of course there is.

In December 1971, President Nixon announced a sea-change in the US administration's expectations regarding the future of cancer. The administration had been persuaded that a major financial infusion into cancer research might largely eliminate it as a cause of death within five years. Hence Nixon declared a “war on cancer”—a massive increase in public funding for cancer research (Epstein et al., 2002).

And where are we now? Over thirty years after Nixon's speech, cancer remains the #2 killer in the US, just as it was then. Certainly *some* progress has been made, but only a vastly less significant advance than that predicted in 1971 to occur within just five years.

Yet, where is cancer funding now? Far from being penalised, the National Cancer Institute budget has risen almost every year since 1971 (National Institutes of Health, 2001). So little is the funding-fear logic felt to apply to the NCI that its chief recently made an announcement echoing Nixon's (Kaiser, 2003).

“Politicians fear life extension, so advocating it will harm funding”

Whatever we may sometimes think of the decisions of our elected representatives, it is both unwise and derogatory to suppose that their non-receptiveness to a view that we consider obvious is necessarily due to inability to grasp simple logic. When one is not compelled to justify one’s views to someone who disagrees, it is often a poor use of one’s time to volunteer such a justification; and this is just the position which politicians generally occupy, especially when the issue enjoys bipartisan consensus within legislatures. We must therefore divine their true reasons for any stated resistance to developing a cure for aging without relying on their enthusiastic help.

A very probable reason is that they know how hard it is to communicate even quite simple ideas to their constituency—especially when the course they are being asked to advocate clearly brings with it serious upheavals of people’s way of life, as is surely true of a cure for aging. People rarely welcome the idea of continuing working into their second century; the argument that they will be so youthful that this is what they will *want* to do tends to fall upon deaf ears. Similarly, a steep rise in the proportion of society who cannot support themselves is, like it or not, the first thing that comes into many people’s heads when they consider life-extension—and, being such a grim prospect, often the last thing too. Politicians know this.

But this is not remotely an argument for avoiding such topics: for, what is the alternative? The only one, if biogerontologists’ actual statements are indicative, is talking up compression of morbidity (Hodes, 2001). Some argue that this has succeeded, but I contend that funding for biogerontology remains vastly short of what is justified by the rate of progress of relevant biology and biotechnology. Is this a surprise? Not if we consider that politicians might perhaps see through the spectacularly porous logic underlying the idea that compression of morbidity is a plausible product of aging research, as rehearsed above. Since any who do so are forced to conclude that we are deluded, duplicitous or both, the prognosis for our suit is hardly rosy.

Thus, we must tell politicians the truth about what our work can and cannot deliver, and ease their path to public advocacy of this effort by telling the public the same via the (abundantly receptive) media. The supposed drawbacks of this course in terms of “engendering unwarranted optimism” are dissected above.

“We’ll be seen as charlatans”

There is, as we are constantly reminded, a large industry based on the public’s desire to alleviate the physiological decline of old age. Purchasers of such products may or may not really believe they will live longer as a result. What is clear, however, is that the purveyors of such products—in line, I think it only fair to acknowledge, with the purveyors of almost anything—agonise very little about the reasons for their customers’ purchasing decisions, except insofar as such understanding may help them to swell those purchasers’ ranks. This is manifest, of course, in the language used to advertise such products, which is not the style of communication with which scientists are familiar or comfortable, and which is roundly criticised by many scientists (myself included) as a result (Olshansky et al., 2002).

So far, so good. Unfortunately, many eminent gerontologists, sociologists and ethicists sincerely dispute the various arguments I have presented here and subscribe to the clichés with which each section has been introduced. Hence, the reader of a statement by a gerontologist expressing distaste for terminology that those with commercial motives have adopted (the prime current example being the term “anti-aging medicine”) is irresistibly drawn to the conclusion that the speaker is in two minds, at best, concerning the merits of curing aging (Le Bourg, 2003; de Grey, 2003c). For those (such as myself) whose lives are unswervingly dedicated to that goal, this is unacceptable—even if the alternative is to allow a misguided interpretation by some that we are as ignorant or blasé about the efficacy of contemporary interventions as those in the anti-aging “industry” may be. No: as usual, honesty is the best policy. We must communicate our distaste for aging (Table 2) just as clearly as our appreciation of its current near-immutability.

	Fox hunting	Human aging
Traditional	✓	✓
Keeps the numbers down	✓	✓
Fundamentally barbaric	✓	✓

Table 2. A peculiarly British characterisation of aging.

Conclusion

The cure of aging must now be taken seriously by responsible gerontologists, because it is no longer science fiction. It is patently not yet science fact either, but it has crossed the boundary into science foreseeable. Its elevation to science fact is a foregone conclusion, but the timeframe of that elevation is not: it depends critically on analysis and creative manipulation of the available technology, to mould it into interventions that actually work. This will not happen until those with the expertise to engage in it are also imbued with the fervour that comes with a reasonable expectation of eventual success [and is utterly impossible without that expectation]. Such individuals must communicate, collaborate and cooperate in addressing the detailed technical obstacles that will inevitably be encountered continually during that effort. This dialogue should be in full swing today. In my view, the main reason it is not is that spurious arguments have, jointly, monopolised public debate and swamped any emerging discussion of substantive matters. I have attempted here to alleviate that problem by a moderately comprehensive onslaught on several downright incoherent (in my view) rejections of the foreseeability and/or desirability of real anti-aging medicine. Time will tell whether this and similar outbursts will make a difference to the rate of progress of the relevant science and technology.

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