Aging, Evolvability, and the Individual Benefit Requirement; Medical Implications of Aging Theory Controversies

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ABSTRACT

There is a class of theories of aging (variously termed adaptive aging, aging by design, aging selected for its own sake, or programmed death theories) that hold that an organism design that limits life span conveys benefits and was selected specifically because it limits life span. These theories have enjoyed a resurgence of popularity because of the discovery of genes that promote aging in various organisms.

However, traditional evolution theory has a core tenet that excludes the possibility of evolving and retaining an individually adverse organism design, i.e. a design characteristic that reduces the ability of individual organisms to survive or reproduce without any compensating individual benefit. Various theories of aging dating from the 1950s and based on traditional evolution theory enjoy substantial popularity. Therefore, any theorist proposing an adaptive theory of aging must necessarily also propose some adjustment to traditional evolution theory that specifically addresses the individual benefit issue. This paper describes an adaptive theory of aging and describes how one of the proposed adjustments (evolvability theory) supports adaptive aging.

This issue is important because adaptive theories are generally more optimistic regarding prospects for medical intervention in the aging process and also suggest different approaches in achieving such intervention.

Keywords: evolution; ageing; gerontology

Theories of aging fall into three main classes. One class, still popular with those primarily familiar with human aging, holds that human aging is the result of generic fundamental deteriorative processes similar to those that cause aging in machinery or chemical systems. A second class that appeals to those familiar with the extreme variability of life span characteristics in non-human species holds that organisms are designed to have a species-specific life span. Traditional evolution theory postulated that it is impossible for an organism to acquire a design that produces a net reduction in individual benefit (life span or reproductive capacity) and led to development of a third class of theories that have been popular with biologists since the 1950s. These theories are based on Medawar's idea[7] (1952) that the evolutionary impact of organisms declines with age beyond puberty. Therefore the major individually adverse manifestations of aging (e.g. weakness, sensory deterioration, even death of old age) had only minor or even negligible effect on evolution. Relatively longer life spans thus provided negligible additional competitive advantage to wild mammals, which therefore did not evolve or retain maintenance mechanisms necessary for longer life spans. Alternately, aging was an unavoidable side-effect of some useful function. These theories explained the observed wide differences in mammal life span without violating the individual benefit requirement.

The relatively recent discovery of aging genes[1,2] and other recent discoveries have resulted in a renewal of interest in theories that consider that complex organisms are designed to have a limited life span and that such a design results from an evolutionary process. Evolvability is one of the adjustments to traditional evolution theory that support adaptive aging.

Evolution of Evolvability

It is generally accepted that organisms possess design features that enable the process of evolution. For example, all organisms possess the ability to pass information describing their designs to descendents, to store that information during the life of the organism, and to copy the information for distribution to multiple descendents, in addition to mechanisms that support accumulative adaptive modification of that information. The question here is whether it is possible for design properties that support or enhance the evolution process to *vary* between different organisms. If such was possible, then could not organisms evolve improvements in their ability to evolve? Would not such enhancements represent an obvious benefit in that organisms possessing them would be able to adapt more rapidly or comprehensively to changes in their environments? Would not any theory of evolution need to deal with variation in the capacity of organisms to evolve?

Traditional evolution theory ignores the evolvability issue. Either of two assumptions supports such a position. The first is that the capacity for evolution is a fundamental property of life that does not and can not vary between populations or species, and that therefore evolvability is a constant that does not need to be considered in devising theories of evolution. The second is that evolvability is enclosed in traditional concepts of

fitness and is therefore covered by traditional theory. Arguments are presented below to the effect that neither of these assumptions is correct.

Unnatural Variation

Darwin described a property of organisms that is essential to the evolution process. He proposed that evolution was dependent on "natural variation" in inheritable design characteristics between individuals. Evolution depends on this variation because natural selection selects between the differences. If, at some point in time all the members of a population were genetically identical, evolution in that population would not be possible, a zero-evolvability situation.

Some might say that variation is the result of mutations to the genetic data that defines organism designs, that surely all species undergo mutations, that the design of the organism does not affect this, and that therefore mutation and resulting variation is a fundamental and invariant property of life. For Darwin this was certainly a reasonable assumption. However, we now know that the variation in fitness parameters that we see in complex organisms actually results from a long list of obviously evolved mechanisms. Complex organisms maintain a pool of mutational differences (i.e. single nucleotide polymorphisms or SNPs) each of which is possessed by some (by definition at least 1 percent) but not all the members of the population. (The human population is thought to possess several million SNPs.) The variation we see is the result of assembling the differences in *combinations* that, through cascading the effects of multiple individual differences, produce the observed effects. The magnitude of variation produced by cascading is generally very much larger than the effect contributed by any one SNP. The mutations in the pool are in effect pre-screened by natural selection to eliminate those that result in major adverse effect and therefore consist only of those that are (considered individually) beneficial, neutral, or mildly adverse and are therefore a potentially useful part of such an assembly.

Further, we now know that nature uses a digital method for handling the organism design information and is therefore limited by the fundamental characteristics that are common to any digital data construct. One such fundamental property is that while it is relatively easy to produce a verbatim copy of digital data it is very difficult to produce meaningful, structured variation. These "digital genetics" limitations[3] have required development of many complex organism design features that process the digital data in producing the observed variation. Mutations are therefore only the feedstock to a very complex system. It is clear that this "variation producing system" has evolved very dramatically between single cell prokaryotes and complex sexually reproducing species. Producing and maintaining variation even involves behaviors: An organism could have an inherited behavior pattern that caused it to seek mates that were not close relatives thus increasing variation.

The inheritance system itself represents a conflict with the idea that natural selection, selecting between phenotypic differences in individual organisms, completely explains evolution. To illustrate, a text document (also digital data) could be written completely

defining the design of some complex structure. The document could then be copied and distributed to multiple builders for execution. The methods and systems used to copy and transmit the data do not affect the design of the structure; (the architect could even have written in a different language). In the same way the design of the inheritance system, which also involves a language, decoding, interpreting, merging, copying, and other complex processes does not affect the phenotypic design of organisms defined by the data it carries. Therefore, traditional evolution theory can not explain the development and evolution of complex (sexually reproducing) inheritance systems while evolvability advantage (structured variation) does present an explanation.

Considered as the result of an evolved design characteristic, variation is individually adverse. Imagine a population of well adapted animals. We could presume that the average height of individuals (we could have picked nearly any characteristic) is nearly optimum from a fitness viewpoint. Therefore all the animals that are shorter or taller than average are less fit. Individuals in a population consisting of clones of an average animal would therefore be more fit than most of those in a population having more variation. Variation therefore benefits evolvability but detracts from fitness. Evolvability is *not* handled by traditional theory. It appears that most (possibly all) design characteristics that benefit evolvability are individually adverse or at best neutral. In the last decade, evolvability issues have been extensively discussed in scientific literature[4, 5, 6].

So let us accept for the moment that many organism design characteristics can affect evolvability, that evolvability varies in the sort of continuous way that fitness varies, that evolvability is generally individually adverse, and that therefore the design of complex organisms must represent a compromise between evolvability and individual benefit. What other design characteristics might benefit evolvability?

Adult Death Rate

We discussed the many complex evolved design characteristics that result in each member of a sexually reproducing species possessing a different combination of all those SNP alleles. Since each has a different combination, each individual could be considered a trial or test of that specific combination. Will the individual possessing this combination live longer and breed more? This sort of logic suggests that the number of lives lived per unit time would be a factor in evolvability. A species that could live more lives could perform more tests. Therefore *death rate*, equivalent to lives lived per unit time would be an evolvability factor.

This concept needs some additional refinement. Natural selection theory says that a characteristic must be *expressed* in such a way as to affect survival or reproduction in order to be selected and that latent characteristics cannot be selected. Therefore, an organism that died prior to becoming an adult generally cannot contribute to the evolution of adult characteristics because adult characteristics are not fully expressed in juveniles. We could therefore suggest that *adult death rate* was a factor in evolvability. Design characteristics that increase adult death rate (adult lives lived per unit time) would increase evolvability. This idea suggests that any organism with an unnecessarily long

life relative to the time required for maturation and reproductive capability would be at an evolvability disadvantage and generally fits the observed loose relationship between organism maturation and life span.

Some theorists point out[7,8] that under wild conditions, an *average* senescing animal in a typical mammal population would not have a shorter life span than that of an non-aging version of the same mammal because of predation, food supply, and other external conditions and suggest that therefore there would not be any expressed difference between aging and non-aging to drive evolution in either direction. Population models typically used in such a formulation assume that an *average* animal will produce descendents at a fixed rate following puberty. For such a model there will necessarily be a consequent fixed *average* life span in order to maintain a stable population. Since deteriorative forces and processes clearly exist, gradual aging would be a plausible "default" result in a situation in which there was no evolutionary force to drive development and retention of maintenance and repair functions.

However, *some* individuals in a non-aging population could be expected to live very long lives and produce very many descendents. (Note that this is an individual benefit. All those descendents would carry the genes of their individual parent. The idea that aging under wild conditions *does* create an individual disadvantage caused development of competing tradition-based theories.) Assuming a stable population and the same model as above, *many* other individuals would consequently necessarily have to die without descendents, presumably as juveniles. A non-aging population thus represents a lower adult death rate, less diversity, and in effect fewer combinations tested, an evolvability disadvantage, and suggests an evolvability purpose for organisms to evolve a system to limit life span as a compromise with individual benefit. Characteristics of complex organisms such as intelligence, immunity, and societal behaviors such as pecking order tend to worsen this situation by increasing the advantage of older individuals and further reducing diversity.

Intelligence and immunity represent cases in which an evolved inherited characteristic depends for its expressed selectable beneficial effect on acquisition of something (experience, exposure to pathogens) that accumulatively increases with age. "Intelligence quotient" embodies this concept. In the absence of a life span limitation, older individuals with their superior acquired characteristics would have an advantage over younger individuals with superior evolved characteristics, which would work against the evolution process. Evolution of intelligence and immunity would therefore appear to specially require a design-limited life span.

A related observation is that age of male puberty in many species appears to be delayed relative to the age that seems to be plausibly required merely for physical (growth) development of reproductive systems. In species in which the male protects or otherwise supports its young, a case can be made that such delay has individual benefit. In reptiles where no such male function exists, delayed male puberty is individually adverse. However, delayed male puberty would have an evolvability benefit by delaying breeding until the individual was mature, therefore expressed adult characteristics, and presumably

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had at least partially passed the life-test. Also, again referring to the model above, delayed puberty has an effect that is similar to and complimentary to life span restriction in improving evolvability by mediating adult death rate. Age-at-puberty and life span seem to be related in this regard. If a population had a lower age-at-puberty, *some* individuals would begin reproducing earlier, and, in a stable population, adult death rate would be reduced.

It also appears, because adult death rate is affected by external factors, that an organism that could locally or temporarily adjust life span to compensate would have an advantage. An organism that could compensate for scarcity by reducing reproduction while increasing life span would have an advantage. This is a possible explanation for the caloric restriction[31] effect.

Some mating rituals (e.g. Bighorn sheep[3]) seem to have a similar effect in generally delaying reproduction beyond puberty age (an individually adverse effect with evolvability benefit).

Group selection[9] (Waynne-Edwards, 1962) proposes a situation in which a future group benefit of some design characteristic trades off against a more immediate individual disadvantage to allow the propagation and evolution of an individually adverse design characteristic such as altruism. Using the traditional model (e.g. Price's equation[10] 1970), propagation becomes progressively more difficult as the size of the group increases and the group benefit is therefore increasingly delayed relative to the individual disadvantage. How does an individually disadvantageous design characteristic propagate into a sufficiently large group for the benefit to be expressed? Subsequently, the feasibility of group selection was heavily criticized (e.g. Williams[11] 1972). Many theorists therefore dismiss group benefits as too "weak" and too "late" to be a feasible trade with individual disadvantage.

Some also think of evolvability as equivalent to species-level group selection, that is, evolvability benefits the species, or benefits future species, or otherwise has benefit that is felt only in the distant future relative to the individual disadvantage. They suggest that a long-term, deferred, large-group benefit can not outweigh an immediate individual disadvantage such as a shorter life span and that therefore evolvability explanations for adaptive aging or any other individually adverse design characteristic are implausible. However, it is clear that logically, evolvability is significantly different from group selection. Evolvability benefits the *evolution process*. Evolvability operates in a very different manner from group selection in that it acts to create conditions that must **preexist** (e.g. variation) in order to enable or enhance the natural selection process. The evolvability concept thus does not require a group larger than or a term longer than the fitness concept. Individual benefit, evolvability, and group selection therefore represent three different evolutionary modalities and a valid propagation model must therefore treat evolvability differently from group selection as well as differently from individual benefit. Prior analyses purporting to show that group selection is infeasible cannot be legitimately applied to evolvability without modifying them to account for the logical differences.

A conceptual hurdle involves trying to visualize how an organism that had an individual advantage over another organism could fail to have an advantage in propagating its design. I find it helpful to remember that evolvability was necessary in order to create a selectable difference in the first place, and that the magnitude of that difference depended on the magnitude of evolvability present. The traditional way of thinking about or analyzing the evolution process is to **assume the existence** of a **selectable** phenotypic difference (e.g. aging vs. non-aging) and then track how natural selection operates upon that difference. This approach does not work for an evolvability characteristic because such characteristics act to create phenotypic difference (variation) or to enhance selection (see prior discussion of adult death rate, intelligence, mating rituals, delayed male puberty, etc.) Evolvability characteristics therefore act to create or set up the *initial conditions* for the scenario being analyzed. The traditional approach therefore captures the disadvantage of an evolvability characteristic without accounting for its compensating benefit, which occurred *prior* to the beginning of the analyzed scenario. To be valid for an evolvability characteristic, the analysis must encompass the entire process, not just the part extending forward from the point at which a selectable phenotypic difference exists.

Propagation Issues Associated with Inheritance Processes

As details of the inheritance processes in sexually reproducing species have gradually emerged it has become apparent that these complex processes could differentially affect the propagation of mutational changes and thus affect evolution. Brief examples: *Genetic linkage*[12] and *unequal crossover*[13] create a situation in which a set of mutational differences that had similar loci on a single chromosome would propagate very differently from an identical set (with phenotypically identical effect) that was more widely distributed in the genome. Further complexity is introduced by other features of inheritance systems such as *transposition*[14], duplication of genetic data, *introns*[15], and creation of *modules* or objects in genetic data[16]. These features result in moving genetic data around in a genome thus affecting genetic linkage or otherwise affect propagation. These features interact with each other and with natural selection in very complex ways and also involve processes that are "long-term" even by evolutionary standards (e.g. movement of functionally similar genes to different loci or different chromosomes in descendent species, increases in introns in more complex species, etc.) suggesting that propagation concepts that require a longer term such as group selection may eventually prove to be much more feasible than supposed by traditional theory. Many aspects of non-phenotypically-functional "junk" DNA appear to have propagation implications. Ultimately, propagation models will need to deal with these issues.

A theory of gradual aging based on individual benefit has never completely gelled despite nearly sixty years of effort. Nagging inconsistencies persist and experimental confirmation has proved elusive. Multiple competing theories (i.e. mutation accumulation[7], antagonistic pleiotropy[17], and disposable soma[18]) with their variants and proponents still exist. Other observational discrepancies with the individual benefit concept (sexual reproduction, elaborate evolved inheritance mechanisms, "acute aging" (biological suicide), some mating rituals, altruism, excessive male puberty age; all of

which have evolvability explanations) are generally more severe but have received less attention and are typically ignored by those producing or defending aging theories based on traditional propagation concepts. Adjustments to traditional evolution theory including group selection theories[19] and the selfish gene theory[20] have been proposed in addition to evolvability concepts. *All* of these adjustments propose to reduce the impact of the individual benefit requirement in efforts to make evolution theory better fit observational evidence. It is increasingly clear that the propagation concepts used to initially develop and now used to defend the traditional aging theories are in need of extensive revision in order to fully incorporate the evolvability and other propagation issues summarized here.

Weismann (1882) proposed what is probably the first evolvability-based aging theory[21]. Skulachev[22] and I[23] have proposed evolvability theories that suggest ways in which gradual aging is superior to acute aging seen in less developed species in producing evolvability benefit. Mittledorf[24,25], Bowles[26], Promislow[27], and Libertini[28] have proposed adaptive aging theories based on group selection or kin selection, and/or have written extensively criticizing the logic behind the tradition-based aging theories.

A currently promoted tradition-based theory[29] explains the approximately 100:1 difference in life span among mammal species as being the result of corresponding differences in the number and effectiveness of a large number (perhaps thousands) of maintenance functions. These maintenance (or "anti-aging") functions each evolved *separately and independently* in order to counteract specific deteriorative conditions. We could imagine maintenance functions directed at preventing cancer, others that act to prevent heart disease, and so forth being more effective in humans than mice thus explaining why both species display the same aging symptoms but at grossly different rates.

Proponents[8] point out that phenotypically an organism with relatively less effective maintenance mechanisms would be identical to one with a relatively more aggressive pro-active suicide mechanism that caused the same symptoms in the same diverse tissues and systems. Therefore they suggest that even if one believes in an adaptive theory based on evolvability or group selection, that theory could be satisfied by the non-adaptive maintenance scenario. Existence of pro-active suicide mechanisms that also produce diverse deteriorative symptoms in lower species (e.g. salmon) is dismissed as irrelevant to mammal aging.

A major difficulty with this idea is that substantial empirical evidence shows that those diverse symptoms of aging are *highly related* in ways that are incompatible with the idea that each results from absence of mechanisms that evolved separately and independently. Hutchinson-Gilford progeria and Werner's syndrome are human conditions in which many (even most) symptoms of aging result from genetic defects that impact a single gene product[30]. Caloric restriction[31] affects many or most aging symptoms and inactivation of even one "aging" gene can substantially affect aging.

Another difficulty is that short-lived and long-lived mammals display very similar manifestations of aging (e.g. cancer, heart disease, cataracts, general mobility and sensory

degradation, etc.) This suggests that the deteriorating processes involved all operate over a time frame that is shorter than then the life span of a short-lived animal. If this were not so, short lived animals would lack at least some manifestations. This in turn suggests that longer lived animals do not need more or more powerful maintenance mechanisms but merely need the same mechanisms to operate for a longer period, a conclusion that supports adaptive aging over traditional theories.

This very issue, the degree of coordination or interrelation between the diverse manifestations of aging, is extremely central to prospects for medical intervention in the aging process. If aging is the result of the absence or ineffectiveness of thousands of independent maintenance functions, medical intervention, as traditional theorist Williams predicted,[17] may well be "impossible." If all or many of those diverse deteriorative processes are coordinated by a pro-active biological suicide mechanism, then interfering with the operation of that mechanism is a reasonable expectation. Further, approaches to intervention are very theory dependent. Those investigating suicide mechanisms will be looking for signaling, coordination, and regulation mechanisms, even nervous system and sensory connections, as well as commonalities between mechanisms that control aging and those that control puberty and other reproductive functions.

For a century aging theory has been seen as having little practical value. Most people still think that aging is a fundamental and unavoidable process and theories based on traditional evolution mechanics tend to reinforce this belief because they typically predict that aging is the result of a large number of very intractable or even unalterable factors. Those that believe in generic deterioration theories have an even dimmer view. Adaptive theories tend to be much more optimistic regarding the possibility of medical intervention in the aging process. If aging is substantially or even significantly the pro-active result of a design, then it is reasonable to believe that ways will eventually be found to alter or compensate for that design aspect, a familiar problem in medicine. Observational data including caloric restriction[31], aging genes, progeria/ Werner's syndrome, and species with negligible senescence[32] tend to support the idea that changing a small number of plausibly treatable factors could dramatically alter aging.

Multiple theories of adaptive aging exist based on two different evolutionary modalities. These theories provide a better fit with empirical evidence than the traditional theories and the main objection is based entirely on propagation issues. Aging theory is therefore essentially a choice between a particular evolutionary propagation model and observational evidence. Given the medical implications and the emerging propagation issues is it not time for a serious and comprehensive reevaluation of this choice?

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