Potential of longevity: hidden in structural complexity

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Abstract

To understand the phenomenon of longevity in biological world, the relationship between the potential of longevity of an organism and its structural complexity is analyzed. I. Potential of longevity is the maximum longevity of a species if the individuals live in an ideal environment. Longevity of an organism includes two parts: the time for development (mature time) and the time for structure-maintenance (maintaining time). II. The mature time for an organism depends on its structural complexity. The maintaining time for an organism is related to two factors, the degree of damage-exposure and the potential of functionality for structure-maintenance. Potential of functionality include two parts: the capacity of basic functionality and the potential of functional compensation. The capacity of basic functionality is built in the structural complexity of the organism. However, the structural complexity and the functionality of an organism will be reduced gradually with age by the accumulation of Misrepairs. Functional compensation can slow down the decline of functionality during aging by two mechanisms: functional substitution and regeneration of cells, and theses two functions are also built in the structural complexity. The potential of longevity of an organism is therefore hidden in its structural complexity, which is determined by the gene configuration. III. An animal has limited longevity because it has limited structural complexity. Limited structural complexity and limited longevity are essential for the survival of a species. IV. In spite having the same potential of longevity, individuals of a species normally have different lifespans. The lifespan of an individual is related to the degree of damage-exposure, which is determined by the living environments and the living habits of the individual.

Keywords

Longevity, lifespan, potential of longevity, structural complexity, potential of functionality, accumulation of Misrepairs, aging, functional compensation, functional substitution, regeneration, repeated developments, redirected development, individual lifespans, living environment, and living habits
To have a longer lifespan is a dream of everybody. For this, we need to know what factors
determine the potential of longevity and why we all have a limited longevity. Some research
groups are searching for “lifespan-related genes” for uncovering the secret of longevity;
however “lifespan-related” does not mean “lifespan-determined”. Aging is related to
longevity; however a concrete link between aging and longevity is missing. To understand
aging, we have proposed a generalized concept of Misrepair in our novel aging theory, the
Misrepair-accumulation theory (Wang, 2009). The new concept of “Misrepair” is defined as
incorrect reconstruction of an injured living structure, such as a molecule (DNA), a cell and a
tissue. Main idea of this theory is that: aging of an organism is a result of accumulation of
Misrepairs on tissue level. This theory can help us not only understand aging and diseases but
also understand longevity. In the present paper, we will analyze the phenomenon of longevity
with a concept of structural complexity. Our discussion tackles the following issues:

I. Concept of longevity

II. The potential of longevity of an organism: hidden in structural complexity

2.1 Concept of structural complexity
   2.1.1 Determining factors for the structural complexity of an organism
   2.1.2 The time for development: determined by structural complexity
2.2 Gradual Reduction of structural complexity and functionality by Misrepairs
2.3 Basic functionality and functional compensation: built in structural complexity
2.4 Limited potential of longevity: a result of limited structural complexity
2.5 A long potential of longevity obtained by redirected development in a queen ant
2.6 A long potential of longevity obtained by repeated developments in trees

III. Individual lifespans: related to the living environments and the living habits

IV. Conclusions

I. Concept of longevity

The term of longevity is referred to as the life expectancy of an organism, namely, the length
of time that an organism exists as a whole structure. It includes the time for development and
the time for maintenance till the breakdown (death) of the structure. Before discussing
longevity, several concepts that are related to longevity should be clearly defined and
distinguished; they are: lifespan, average longevity, and potential of longevity. The term of
longevity is a statistical prediction of life-length, and it is strictly speaking different from the
actual life-length of an organism, which is called “lifespan”. Longevity of a group of
individuals of a species is often predicted on the basis of average lifespan of the individuals in
this group, called average longevity. The organisms that are of the same species but living in
different geographic areas and climates can have big difference on average longevity. When
we compare the longevities of different species’, we are actually studying their maximum
longevity, which can be called “potential of longevity” of a species. Potential of longevity is

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the life expectancy of an organism when it lives in an “ideal” environment, where it can obtain sufficient food and be exposed to the least damage. In summary, the following three notions characterize longevity:

- **Lifespan**: the actual length of life of an organism

- **Average longevity**: the average life expectancy of a group of individuals of a species, who live in similar environments.

- **Potential of longevity**: the maximum life expectancy of an individual of a species if it lives in an ideal environment

II. The potential of longevity of an organism: hidden in structural complexity

On predicting the potential of longevity of an organism, two elements are fundamental: one is the complexity of its structure, which determines the time for development, and the other is the potential of its functionality, which determines the time for structure-maintenance till the death of the organism. Since the functionality of a system is determined by the structure, the potential of functionality of an organism should lie in the complexity of its structure. In the following, we will discuss how the complexity of a structure determines its functionality and determines its longevity.

2.1 Concept of structural complexity

In physics, complexity is an approach to describe the relationship between a system and its sub-systems in a complex system such as a living being. Complexity of a system manifests on several aspects, including emergence, feedback effect, self-organization, and adaptation (Haken, 1990; Wunderlin, 1992). **Emergence** is the phenomenon that the behavior of a system cannot be tracked back to the behaviors of its sub-systems. For example, a heart has the ability of pumping of blood, but a singular cordial muscle cell, as a part of the heart, has not this ability. **Feedback effect** is the phenomenon that sub-systems can make responses to the changes on a system, and wise verse. For example, cordial muscular cells are the functional basis for a heart; however failure of the heart caused by arterial hypertension can result in death of cordial cells. **Self-organization** is the phenomenon that a system can develop its structure automatically by organizing its sub-systems given sufficient energy and substance. Development of embryo is an example of self-organization. **Adaptation** is the phenomenon that sub-systems can make suitable responses to the changes in environment for increasing the surviving chance of the whole system. For example, overloading of the heart by hypertension will induce the enlargement of cordial cells, which can make functional compensation for the heart. All of these phenomena in complex systems manifest the complex functional relationships between a system and its sub-systems. Important is that all of these functional relationships are built in the organization, namely the special spatial relationship, of sub-systems. For a system and an organism, the functional complexity is based on structural complexity. Therefore, to understand the potential of the functionality of a system, it is essential to study its structural complexity. In this paper, structural complexity is defined
as the complexity of the organization of sub-systems (or sub-structures) of a system (or an organism).

2.1.1 Determining factors for the structural complexity of an organism

Firstly, the degree of the structural complexity of an organism is related to the number of hierarchic levels of sub-structures, the number of types of sub-structures, and the amount of sub-structures of each type. For example, molecules, cells and tissues are the three levels of sub-structures of a multi-cellular organism. In a tissue, there are different types of cells and extracellular matrix molecules (ECMs). Structural complexity of a tissue will increase with the increase of the number of types and amounts of cells/ECMs. Secondly, the manner of organization (distribution) of sub-structures also contributes to the structural complexity of a structure. An organization will have a higher complexity if it enables each of the sub-structures to communicate with more other sub-structures. As shown in Figure 1, organization C has higher complexity than organization A and organization B: in organization C each sub-structure (such as the sub-structure z) can communicate with six other sub-structures, whereas in A (or B) each sub-structure can communicate only with three (or four) other sub-structures.

![Figure 1. Different complexities in different manners of organization of sub-structures of a structure](image)

An organization will have a higher complexity when it enables each sub-structure to communicate with more other sub-structures. For example, organization C has higher complexity than organization A and organization B. Each sub-structure (such as the sub-structure z) in organization C can communicate with six other sub-structures, whereas in A (or B) each sub-structure can communicate only with three (or four) other sub-structures.

Multiple communicating pathways between sub-structures compose a communicating network like that in the organization of lobules in a liver (Figure 2). In the liver, every lobule has six branches of portal veins, and three neighbor lobules share a branch of portal vein. By such an organization, each lobule functions as a common pathway for six branches of portal veins to a central vein; and each portal vein has three pathways to “communicate” with central veins via lobules. Such organization of lobules reduces the risk of failure of the liver when some lobules or portal veins fail. Network-like organization of sub-structures is an effective strategy to provide functional compensation for an organ. Like that between neuron cells in the brain, the complexity of a communicating network is determined by the number of
dendrites (contacts) of each sub-structure (neuron cell) to other sub-structures. In summary, structural complexity of an organism is not only determined by the number of levels of sub-structures, the diversity and the amount of sub-structures, but also by the number of communicating pathways of each sub-structure with other sub-structures, namely the complexity of the communicating network of the sub-structures.

Figure 2. Network–like organization of lobules in liver

In a liver, every lobule has six branches of portal veins, and three neighbor lobules share a branch of portal vein. By such an organization, each lobule functions as a common pathway of six branches of portal veins to a central vein, and each portal vein has three pathways to “communicate” with central veins via lobules. Network-like organization of lobules is an effective strategy to reduce the risk of failure of the liver when some lobules or veins fail, and it gives the liver a great potential of functional compensation.

2.1.2 The time for development: determined by structural complexity

Development of an organism is a process of constructing of a structure and its complexity. A more complex organism needs longer time for development. Animals from different species’ have different sizes of organs; therefore they have different degrees of structural complexity and need different time for development. However, the period of development cannot be too long; otherwise the structure has a high probability to be destroyed before being fully developed. In nature, full development of an organism is only possible when the rate of construction is higher than that of destruction by damage. Development of an embryo needs to be in a protected environment, since such a high complexity cannot be built up successfully in damaging environment. Nature pressure restricts the increase of structural complexity of creatures. An animal has limited structural complexity because it has limited development. Different species’ of animals have different limits on structural complexity, and the limits are determined by their gene configurations. Some plants including most species’ of trees undergo repeated developments after reproduction age, and the repeated developments increase their structural complexities continuously. Some trees seem to have no limit on structural
complexity genetically; however their developments will be stopped by a catastrophe in nature.

2.2 Gradual reduction of structural complexity and functionality by Misrepairs

An organism is able to maintain its structure by exerting functionality on different aspects. The potential of functionality of an organism on each aspect is built in its structure, which is normally fully developed. An organism goes to failure on functionality in two ways: one is rapid, directly from a severe injury, which destroys the structural integrity of the organism; and the other is gradual, through a process of aging. In our view, aging of an organism is a result of accumulation of Misrepairs of its structure (Wang, 2009). In situation of a severe injury, when a full repair is impossible to achieve, Misrepair, a repair with altered material and in altered remodeling, is a strategy for maintaining the structural integrity for increasing the surviving chance of an organism. Misrepairs are essential for an individual to survive till the age of reproduction; therefore Misrepair mechanism is essential for the survival of a species. However, a Misrepair results in structure-alteration and function-reduction like that in scar formation. Misrepairs are irreversible and irremovable; therefore they accumulate with time, appearing as aging of the organism. Accumulation of Misrepairs results in a gradual reduction of structural complexity and gradual reduction of functionality of an organism.

2.3 Basic functionality and functional compensation: built in structural complexity

For an organism, the potential of functionality includes two parts: the capacity of basic functionality and the potential of functional compensation. Basic functionality is determined by the amount of “functional units” in a structure, which is determined by the number of sub-structures on each level. For example, the digesting capacity of a stomach is determined by the number of glands in the stomach mucosa. Basic functionality can be in most cases approximately evaluated by the size of a tissue/organ, which is in ratio to the number of functional units. Functional compensation is the phenomenon that the lost functionality of a tissue/organ caused by injuries can be completely or partially compensated by a renewed sub-structure or by a substitutive sub-structure. Through functional compensation, the rate of decline of the functionality of a tissue/organ during aging is slowed down. There are mainly two strategies for functional compensation: one is functional substitution through network-like organization of sub-structures, and the other is regeneration of sub-structures. The potentials of functional compensation by these two strategies are also built in structural complexity.

A network-like organization of sub-structures is the structural basis for functional substitution. In this network, each sub-structure functions as a crossing point of several communicating pathways, and the communications between two sub-structures have more than one pathway. The multiple pathways between two sub-structures can substitute to each other in functionality. When one pathway fails, the functionality of the whole structure will not be severely affected. For example, in a structure like that in Figure 3A, sub-structure X2 can communicate with sub-structure Z2 via two pathways: Y1 and Y2. Y1 can communicate with Y2 via pathway X2 and via pathway Z2. The pathway between X2 and Z1 and the pathway between X1 and Z2 cross at Y1. In mediating the communication between X2 and Z2, Y1 can
function as a substitution for Y2. When Y1 fails, its functionality can be substituted by Y2; and the communicating efficiency between X2 and Z2 will not be severely affected. The distribution of small blood vessels in an organ is also in a network. Blood vessels cross each other, composing a network, which is similar to that of veins of a plant leaf (Figure 3B). For exam, in the leaf, vein R1 crosses with vein T1 at point S1, and the part of tissue at point S1 has two sources of water supply: vein R1 and vein T1. For S1, vein T1 can substitute vein R1. Similarly, the parts of tissue at points S2, S3, and S4 have all two sources of water supply: vein T and vein R. A network-like distribution of branches of blood vessels reduces the risk of death of an organ from the blockage of some branches of blood vessels. The potential of compensation through functional substitution is determined by the number of communicating pathways of each sub-structure with other sub-structures.

A

B

Figure 3. Functional compensation through functional substitution

A. Communicating network between sub-structures In the organization of sub-structures X1, X2..., Y1, Y2..., and Z1, Z2..., sub-structure X2 can communicate with sub-structure Z2 via two pathways: Y1 and Y2. Y1 can communicate with Y2 via pathway X2 and via pathway Z2. The pathway between X2 and Z1 and the pathway between X1 and Z2 cross at Y1. These pathways between different sub-structures cross with each other and compose into a network. In mediating the communication between X2 and Z2, Y1 can function

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as a substitution for Y2. When Y1 fails, its functionality can be substituted by Y2; and the communicating efficiency between X2 and Z2 will not be severely affected.

B. Network-like distribution of the veins in a plant leaf In the leaf, vein R1 crosses with vein T1 at point S1, and the part of leaf at point S1 has two sources of water supply: vein R1 and vein T1. For S1, vein T1 can substitute vein R1. Similarly, the parts of leaf at points S2, S3, and S4 have all two sources of water supply: vein T and vein R. The network-like distribution of veins reduces the risk of death of a leaf from the blockage of some branches of veins.

Regeneration of cells in a tissue is induced by death of cells, and new cells are used for replacing the dead ones. Regeneration has two aspects of importance for an organism: one is to maintain the structural integrity, and the other is to compensate the functionality. Regeneration of cells can compensate functionality by making up the lost structural complexity. Full repair of a tissue with death of cells can only be accomplished by regeneration of cells. Those tissues that are exposed constantly to external damage, including epithelium and endothelium, could not function long time if they have no potential of cell-regeneration. Misrepair can be also accomplished by regeneration of cells; however, in a Misrepair, the reproduced cells are in an altered reorganization, and they can only partially compensate the lost functionality of the tissue (Figure 4). Reduction of functionality during aging can be therefore slowed down by cell-regeneration. The potential of compensation through cell-regeneration is determined by the amount of stem cells. In summary, the potentials of functional compensations through substitution and through regeneration are both built in the structural complexity of an organism. The creatures that have short longevity such as worms have low structural complexity; therefore they have low capacity of basic functionality and low potential of functional compensation.

Figure 4. Functional compensation by regeneration of cells in full repair and in Misrepair

Regeneration of cells in a tissue is induced by death of cells, and new cells are used for replacing the dead ones ( ). Full repair is only accomplishable through regeneration of cells, in which the structural complexity and the functionality is completely restored (full repair). Misrepair can be also accomplished by regeneration of cells; however the reproduced cells are in an altered reorganization, which can only partially compensate the lost functions of a tissue (Misrepair).
2.4 Limited potential of longevity: a result of limited structural complexity

The time for development depends on the structural complexity of an organism, whereas the time for structure-maintenance is related to two factors: the potential of functionality and the degree of damage-exposure. The potential of functionality is determined by the structural complexity, and the degree of damage-exposure is determined by the living environment. Gradual reduction of functionality is driven by the accumulation of Misrepairs as a result of struggling of an organism with damaging environment. The more destructive the environment is, the more rapid an organism loses its functionality. Limited potential of longevity of an organism is a result of limited structural complexity. In the same living environment, those species’ that have higher structural complexity will have higher potential of longevity. However, the increase of longevity of organisms will be restricted by the destructive pressure in nature. If an organism is too complex on structure, its development will need too long time. When most individuals cannot survive till reproduction age, the species cannot survive. A limited complexity and a limited longevity are essential for the survival of a species. Nature pressure restricts the increase of structural complexity of creatures.

2.5 A long potential of longevity obtained by redirected development in a queen ant

The big difference on lifespan between a queen ant and a worker ant is amazing. As a matter of fact, a queen ant develops differently from that of other female ants, although they have the same gene configuration. A queen ant can continue developing and growing after reproduction age, whereas other female ants stop developing after mature age and become working ants or defending ants. The redirected development of the queen ant is induced by environment factors; and this altered pathway of development makes a queen ant have a distinct body structure. Continuous development, by increasing the structural complexity and the potential of functionality, makes a queen ant have a much bigger body and a much longer longevity than other ants (Figure 5). Apart from higher structural complexity, living in protected environment also contributes to the long lifespan of a queen ant.

![Diagram](image_url)
Figure 5. Different development and different potential of longevity of a queen ant from a worker ant

A queen ant develops differently from that of other female ants, although they have the same gene configuration. Difference on the pathway of development between a queen ant and a worker ant is the basis of their different potentials of longevity (PL). A queen ant can continue developing and growing after reproduction age (Q), whereas other female ants stop developing after mature age, and become working ants (W). Redirected development of a queen ant is induced by environment factors. Longer development, by increasing the structural complexity and the potential of functionality, makes a queen ant have a much bigger body and a much longer longevity than a worker ant.

Some genes are found to be “aging-related” or “lifespan-related”, and gene modification is thought to be a strategy for extending the longevity of animals. However, if genetic modification could extend longevity, it should be through altering the process of development rather than by retarding aging. Modification of a gene can possibly redirect the development and alter the final structure of an organism. With altered structural complexity and altered potential of functionality, an organism can have an altered longevity. The research group of Helfand has successfully made the lifespan of drosophila be extended by two times through introducing a mutation on gene Ingy (Mardon, 2003). Although the mutant individuals of drosophila have normal metabolism and normal flying ability; their reproducing ability was reduced significantly, especially in the condition of low calorie nutrition. In another study, the mice that have 25 % lower expression of protein mTOR, which is involved in cell metabolism, have 20% longer lifespan (Wu, 2013). The mutant mice are healthy; however they are slightly smaller than normal mice and they are more sensitive to infections. In these studies, although it is not known how a genetic modification of a gene affects the development of tissues and organs, alterations on development have been observed anyway on the changes in the mutants, including the changes on the body size, on the immunity, and on the ability of reproduction. In our view, a strategy for extending longevity through gene modification cannot be successful, while such a strategy can lead to defective development. With defect on functionality, with low chance of survival, and with low rate of reproduction, the modified species cannot survive long time.

2.6 A long potential of longevity obtained by repeated developments in trees

Compared with animals, many species’ of trees can survive much longer. A main reason is that trees can develop repeatedly after reproduction age, by which they obtain additional structural complexity and additional functionality (Figure 6). Differently from the redirected development in some animals, which is induced by environment factors, the repeated developments of a tree are genetically controlled. Some trees seem to have no limit genetically on structural complexity; however a catastrophe in nature will terminate the developments. The lifespan of a tree is a result of competition between construction and destruction of the structure in nature. The trees that have longer longevity often have higher functionality on defending and self-protecting, which are built in a higher structural complexity. However for a higher complexity, they need longer time for development, and more individuals can die before becoming mature. Those species’ of trees that have extreme long longevity are often the species’ that are on the edge of extinction. For example, the
Ginkgo biloba trees, which can survive 3000 years, need 20 years for development till mature on functionality; whereas willow trees, which have longevity of 150 years, need only 2-3 years for full development.

![Graph showing longevity comparison between trees and animals](image)

**Figure 6. Long longevity of a tree: obtained by repeated developments**

Some species’ of trees have much longer potential of longevity (PL) than animals (PL of a tree and PL of an animal). A main reason is that trees can develop repeatedly after reproduction age, by which they obtain additional structure complexity and additional functionality. Some trees seem to have no limit genetically on structure complexity; however a catastrophe in nature will terminate the process of developments (PL).

III. Individual lifespans: related to the living environments and the living habits

With the same potential of longevity, the individuals of a species can have quite different lifespans. Twin brothers often have different lifespans although they have a similar genetic background and a similar structural complexity. Here we mainly discuss the aging-associated lifespans of human being. With aging we are approaching to death with diseases, such as tumors, arterial hypertension, and atherosclerosis. Direct cause of death is often the failure of a key organ such as the heart and the brain. Failure of the key organs can be acute, progressive and chronic respectively. **Acute failure** of an organ is a result of breakdown of its structural integrity by a severe injury; which can be a consequence of aging of other organs. For example, cerebral bleeding is fatal by destroying the structural integrity of the brain; however it is often a consequence of arterial hypertension, which is a result of aging of arterial walls. **Progressive failure** of an organ is often a result of tumor invasion. **Chronic failure** of an organ is a result of gradual loss of functionality with aging of the tissues. Alzheimer’s syndrome is a disease caused by aging and the chronic failure of the brain.
Three factors can affect the “lifespan” of a key organ: rate of aging, inducing factors of failure of the organ, and random factors. Firstly, the rate of aging is determined by the degree and the frequency of damage-exposure of an organ, which is closely related to the living environments and the living habits of an individual. The rates of aging can be quite different on different organs in different individuals. For example, smokers can have much quicker aging on the lung than non-smokers. Individuals who are often exposed to strong sunlight can have accelerated aging on the skin. Individuals who live in a more polluted and colder environment can have higher probability to have chronic pulmonary inflammations and have accelerated aging on the airway walls and on the lung. Secondly, complete failure of an organ takes place often when there is an inducing factor. For example, arterial hypertension increases the risk of disruption of arterial walls; however cerebral bleeding is often induced by excitement of the nerve, which can increase further the blood pressure to local arteries. Patients with cancers often die from infections because their immune system is severely destroyed by the invasion of tumor cells. Inducing factors are the factors that can overload the aged and fragile organs. Thirdly, some random factors are also related to the lifespan of an individual. For example, the degree of the malignancy of a tumor is one of the determining factors on the surviving time of a patient with a tumor.

Some animals have special competence for self-protecting and for survival in nature. For example, tortoises obtain their long longevity, more than 100 years long, mainly by long-period of hibernation. During hibernation, they have reduced opportunity for damage-exposure by bad weather and by animal attacks. In fact, during half of their lives, tortoises are in a state of hibernation; thus they do not “live” long, but “sleep” long! An african elephant can have a lifespan of 70 years, because it has almost no natural enemy except human being! Being able to build a protective living environment is the secret of long life of some animals including human being. As human being, we have been able to build a society with agriculture, industry and medical system for a better living condition and for higher living security. It is the civilization that has help extend the average longevity of human being more than two times in the last five centuries.

IV. Conclusions

We have discussed in this paper the determining elements of the potential of longevity of a species and the influencing factors on individual lifespans. Development of an organism is the process of building of a structural complexity and functionality. However, the structural complexity and the functionality of an organism will be gradually reduced by the accumulation of Misrepairs. The potential of longevity is hidden in the structural complexity of an organism, since the structural complexity determines the time for mature and also determines the potential of functionality for structure-maintenance. For animals, limited longevity is a result of limited structural complexity. Some trees have long longevity because they can obtain additional structural complexity by repeated developments. A queen ant has much longer longevity than a worker ant because it undergoes a “redirected” development. Limited structural complexity and limited longevity is essential for the survival of a species. In spite having the same potential of longevity, individuals of a species can have much

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different lifespans; and the lifespan of an individual is related to his living environment and his living habits.

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