Processes of Aging: Ontogenetic Perspectives on Life, Nutrition, and Longevity

Alessandro Goncalves Campolina

ALESSANDRO GONÇALVES CAMPOLINA is a physician and scientific researcher at the Cancer Institute of Sao Paulo (Center for Translational Research in Oncology) at the University of Sao Paulo, Brazil. E-mail: <alessandro.campolina@hc.fm.usp.br>

ABSTRACT: Whiteheadian concepts of life, food, "empty" and "occupied space" provide a theoretical basis to unpack an ontogenetic perspective on aging. Focusing on the so-called "Selective Optimization with Compensation" (SOC) strategy, this work will explore this concept in relation to some scientific evidence in the fields of "epigenetics" and molecular nutrition. Further, the role of caloric restriction in health and longevity will be discussed as a SOC strategy, based on the metabolic theory of aging. SOC strategy applied to the processes of aging, when linked with Whitehead's philosophy of organism, makes it possible for us to think about life as a selective process provided by "empty space." A continuum within the physical field optimizes a "living society," which evolves in permanent social deficit, by means of compensation by nutritional metabolism.

Introduction

In *PR* Alfred North Whitehead makes an interesting statement about the connection between food and life:

> The highly complex inorganic societies required for the structure of a cell, or other living body, lose their stability amid the diversity of the environment. But, in the physical field of the empty space produced by the originality of living occasions, chemical dissociations and associations take place which would not otherwise occur. The structure is breaking down and being repaired. The food is that supply of highly complex societies from the outside which, under the influence of life, will enter into the necessary associations to repair the waste. Thus life acts as though it were a catalytic agent. (*PR* 106)

I will not be examining the physical or metaphysical distinction between life and food. Instead, my interest is more generally the relation between what Whitehead calls "empty space" and what he calls "occupied space."

But in choosing to treat these two space-related concepts, I will uncover some ontogenetic perspectives more attached to the notions of time and development. By doing so, I propose to examine some characteristics of the living society (or living organism) that could help in understanding the aging process, based on some of Whitehead's concepts.

In Whitehead's philosophy, the term "society" refers to more than a set of actual entities to which the same class-name applies. To constitute a society these entities must exhibit a kind of self-sustaining order, i.e., each member, by means of their common character, derivate genetically from the other members of that same society. In other words, we could say that a society is a set of actual entities with "social order."

But a "living society" is a kind of society in which some "living nexus" (or "living occasions") are also included. This means that another kind of order is necessary to make a living-type society. Whitehead calls it "personal order." Thus, a "living nexus," in virtue of its life, may support a thread of personal order along some historical route of its members. This is to say that, while "living nexus" lacks the genetic power which belongs to societies, it points toward the very characteristic of life that constitutes a "living person": the origination of conceptual novelty.

These concepts led Whitehead to think that the characteristics of a "living society" involve a structure of inorganic societies that is woven together for the production of a personal (and non-social) nexus, characterized by the physical experiences of its members. This intense experience, that is the condition for spontaneity and novelty, made him propose that life is a characteristic of "empty space" and not of the "occupied space" by any corpuscular society participating in a living organism.

The relation between "empty" and "occupied space" will unfold in the development of the present article, but in order to prevent some misunderstandings about the thesis I will try to problematize in this work, I will initially specify three different points:

1. A "living society" evolves in permanent social deficit and can be thought about through the relation between "empty" and "occupied space."
2. The characteristic of a "living society," i.e., its life or vitality, is produced by the "empty space" in a process of construction and deconstruction, while producing a physical field.
3. Food participates as a supply for these highly complex "living societies" to repair structural wastes and to keep the living functional.

Thus, the thesis I would like to defend in this article is the following one: Life, as a process of selection, constructs and deconstructs a "living society" in permanent social deficit, i.e., in the process of aging, by means of compensation by nutritional metabolism.

For this purpose, I will review some of the theoretical propositions characteristic of the Life-Span Developmental Approach, focusing in the so called "Selective Optimization with Compensation" (SOC) strategy (see the works by Baltes). At the same time, I will explore the relations of food and life, appraising some scientific evidence in the field of "epigenetics" and molecular nutritition (see Gillies). Finally, I will discuss the role of caloric restriction in health and longevity, as a SOC strategy, based on the metabolic theory of aging.

The objective, then, is to think with the Whiteheadian concepts of life, food, "empty" and "occupied space," some of the ontogenetic perspectives on the processes of aging provided by contemporary biology.

SOC Strategy and the Processes of Aging

The lifespan developmental approach is considered a family of perspectives involved in the study of constancy and change in behavior throughout the life course, from conception to death. In recognition of the contextual factors and the unique combination of influences that shape a given lifespan, researchers of this ontogenetic process focus on complex and pluralist explanations of development (as in the work of Baltes).

In short, the systemic whole of individual development is studied in terms of three general functions: (a) the function of growth; (b) the function of maintenance, including recovery (resilience); and (c) the function of regulation of loss. Some of the theoretical propositions characteristic of this developmental approach are: multidirectionality, development as gain/loss, plasticity, historical embeddedness, and contextualism.

The work of Paul Baltes and colleagues, for example, is a clear effort in the search for a general process of systemic functioning that would serve as an effective strategy for dealing with lifespan architecture in old age. According to the authors, for older adults to maintain autonomy in specific domains of functioning, the effective exercise and use of dependent behavior is a compensatory necessity. By invoking dependency and support, resources are freed up for use in such domains "selected" for personal efficacy and growth.

These understandings led to the proposition of the SOC strategy: an orchestration of selection, optimization, and compensation that is not unique to human aging, but inherent in any developmental process. The basic assumption in this strategy is the notion of development as selective "canalization," which would optimize positive change in adaptive capacity toward desirable outcomes, requiring the application of behavior enhancing factors such as cultural knowledge, physical status, goal commitment, practice, and effort. Compensation would be operative whenever a given set of means is no longer available, either because of direct losses of these means (e.g., hearing loss), negative transfer (e.g., incompatibility between goals), or new limiting constraints in time and energy. Thus, with Whitehead, we can say that although "life in its essence is the gain of intensity through freedom," it could also be submitted to "canalization and so gain the massiveness of order."

The case of the 80-year-old concert pianist Arthur Rubinstein is treated by Baltes as a good illustration of proficiency in SOC strategy. When asked in a television interview how it was possible for him to maintain such a high level of expert piano playing at an advanced age, the pianist hinted at the coordination of three strategies. First, he plays fewer pieces (selection); second, he practices these pieces more often (optimization); and third, he suggests that to counteract his loss in mechanical speed, he uses a kind of impression management, such as introducing slower play before fast segments, so to make the latter appear faster (compensation).

Regarding the theorization about the relations of life and food, it is possible to think about SOC strategy in a wider perspective, expanding lifespan developmental concepts to the cosmology of Whitehead's philosophy of organism. For this purpose it is important to consider the description of a living cell proposed by the author:

> (i) an extremely complex and delicately poised chemical structure; (ii) for the occasions in the interstitial "empty" space a complex objective datum derived from this complex structure; (iii) under normal "responsive" treatment, devoid of originality, the complex detail reduced to physical simplicity by negative prehensions; (iv) this detail preserved for positive feeling by the emotional and purposive readjustments produced by originality of conceptual feeling (appetition); (v) the physical distortion of the field, leading to instability of the structure; (vi) the structure accepting repair by food from the environment. (*PR* 106)

Up to now, it has been sufficient to notice how food acts as compensation (vi) in a vital process of continuing selective optimization. Selection is made by a vectoral crossing of positive and negative prehensions (iii and iv), that make the optimization of "a extremely complex and delicately poised chemical structure" possible in dynamic instability (i, ii, v). I will return to this point later, after discussing the relations of "empty" and "occupied space," to make clear the processuality of two seminal concepts in biology (metabolism and epigenetics), regarding the points specified in the introduction of this article.

Metabolism and Empty Space

As stated before, in Whitehead's philosophy, life is a characteristic of "empty space" in that "life lurks in the interstices of each living cell," life is a process from "the between," life flows through the gaps of the biological tissues and cellular structures.

We can take, for instance, the life of a cell as a historic route of "actual entities," which inherit from each other, constituting a "society." The members of this "society" are arranged in a serial order by their genetic relations, i.e., they possess "personal order." But it is important to consider that there is no "society" in isolation. Every "society" must be considered with its background of a wider environment of "actual entities." The given contributions of the environment must at least be permissive of the self-sustenance of the society. The background contributes general characters which the more special character of the "society" presupposes for its members. But this means that the environment, together with the "society" in question, must form a larger society in respect to some more general characters than those defining the society from which we started. Thus, we arrive at the principle that every society requires a social background.

For example, we speak of a molecule within a living cell, because its general molecular features are independent of the environment of the cell. Thus, a molecule is a subordinate society in the structured "society" which we call the living cell. But there may be another "nexus" included in a structured "society" which, excepting the general systematic characteristics of the external environment, presents no features capable of genetically sustaining themselves apart from the special environment provided by that structured "society." Recurring to the example of a living cell, it will be argued that the occasions composing the "empty space" within the cell

exhibit special features which analogous occasions outside the cell lack. Thus, the "nexus," which is the "empty space" within a living cell, is called a "subordinate nexus," but not a "subordinate society." In practice a "society" is called living only when an "entirely living nexus" is regnant.

So, these "entirely living nexũs" that constitute the "empty space," according to Whitehead, are where "chemical dissociations and associations take place." This is a place where "structure is breaking down and being repaired." This is a place where, in biological terms, metabolic processes (or metabolism) occurs.

Metabolism is a term derived from the Greek *metabole* (change or overthrow) and which is used to refer to all chemical reactions that occur in living organisms. It can also be used to express the set of life-sustaining chemical transformations. Since the environment is under inevitable change, the body and its parts are in a continuous state of dissolution and nourishment. Consequently, metabolism is necessary to allow organisms to grow and reproduce, maintain their structures, and respond to external stimulation (see Bing).

The chemical reactions of metabolism are organized into metabolic pathways, in which one chemical is transformed through a series of steps into another chemical. Enzymes are crucial to this process because they drive desirable reactions that would not occur without required energy at an adequate speed. So, enzymes act as catalysts that allow the reactions to proceed more rapidly, making possible the regulation of metabolic pathways in response to changes in the cell's environment or to signals from other cells.

Moreover, in terms of selective properties, the metabolic system of a particular organism determines which substances it will find nutritious and which ones it will find poisonous. In terms of optimization, it is important to state that the speed of metabolism, the metabolic rate, influences how much food an organism will require and it also affects how it will be able to obtain that food.

The notion of "metabolism" is, thus, a key concept in modern biology and a fundamental biochemical process in developmental theories, including some of the most remarkable theories of aging (see Barzilai, et al.). Some of these theories relating to the rate of living and the metabolic theory of aging, for example, suggest that longevity is closely related to metabolic rate. Some hormones, such as insulin, have an important role in regulating energy metabolism with aging. Moreover, insulin resistance (IR) represents

a major component of metabolic syndrome (MS) and is commonly observed in older adults. IR is a physiological condition in which cells fail to respond to the normal actions of the hormone insulin. The body produces insulin, but the cells in the body become resistant to insulin and are unable to use it as effectively, leading to hyperglycemia (the elevation of glucose in the blood). This is the main biologic process in metabolic syndrome (MS), which is a disorder of energy utilization and storage, diagnosed by a co-occurrence of three out of the following medical conditions: abdominal obesity, elevated blood pressure, elevated fasting, elevated fasting plasma glucose, high serum triglycerides, and low high-density cholesterol levels.

Thus, aging is arguably the most universal contributor to the etiologies of metabolic decline and related diseases, including type 2 diabetes mellitus (T2DM), cardiovascular disease (CVD), and stroke. Thus, key features of metabolic signaling pathways (MSP) can modulate age-related disease risk and longevity. MSP is part of a complex system of communication that governs basic cellular activities and coordinates cell actions. The ability of cells to perceive and correctly respond to their microenvironment is the basis of development, tissue repair, and immunity as well as normal tissue stability. Gene activations and metabolism alterations are examples of cellular responses to extracellular stimulation that require communication, based on signaling pathways.

In humans, IR accompanied by compensatory hyperinsulinemia (a condition in which there are excess levels of insulin circulating in the blood, resulting from a variety of metabolic diseases, not only diabetes) has clearly been implicated as a risk factor for multiple age-related diseases. In support of this observation, improved longevity and multiple features of delayed aging have been described in mice, rats, and other mammals in which insulin sensitivity is increased by genetic mutations (see Barzilai et al.).

Thus key features of metabolic signaling pathways could modulate age-related disease risk and longevity, calling attention for the importance of metabolic optimization all along the life course.

Epigenetics and Occupied Space

An understanding of selective optimization, as a developmental and also a metabolic process, can be attained by paying attention to the characteristic of a "living society," according to Whitehead's philosophy,

which is "a complex structure of inorganic societies is woven together for the production of a non-social nexus characterized by the intense physical experiences of its members." But it is fundamental to notice that in the initial phases of this continuing process, an "objective datum," derived from this complex structure, must be offered for the occasions in the cellular (or inside) "empty space." This means that some kind of reaction to the outside stimulus should be considered as a threshold that must be surpassed, or as a condition for the metabolic processuality of the non-social nexus of the "empty space," where the intense physical experiences will happen.

The contemporary notion of "epigenetics" is useful to understand how a complex corpuscular cellular structure (the genome, for example) reacts to a physical field created by the interstitial (or outside) "empty space." The term "epigenetics," which literally means outside conventional genetics, is now used to describe the study of stable alterations in gene expression potential (the process by which information from a gene is used in the synthesis of a functional gene product, like proteins, for example) that arise during development and cell proliferation (see Jaenisch and Bird). Although epigenetic processes do not involve mutations of the DNA itself, they are essential for development and differentiation because they are heritable in the short term.

Further, external influences on epigenetic processes are seen in the effects of diet on long-term diseases such as cancer (see Davis and Ross). Epigenetic mechanisms seem to allow an organism to respond to the environment through changes in gene expression. Subsequently, cells of a multicellular organism are genetically homogeneous, but structurally and functionally heterogeneous, by means of epigenetic alterations, owing to the differential expression of genes.

It was proposed in 1975 that DNA methylation plays a fundamental role in epigenetic processes. DNA methylation is a biochemical process where a methyl group is added to the DNA molecular structure. This modification of the DNA alters the genes expressed in cells when they divide and differentiate from embryonic cells into cells of a particular tissue. This might be responsible for the stable maintenance of a particular gene expression pattern through mitotic cell division, although biochemical evidence indicates that DNA methylation is just one component of a wider epigenetic program that includes other post-synthesis modifications of chromatin (a complex of macromolecules found in cells, consisting of

DNA, protein, and RNA). The primary functions of chromatin are: (1) to package DNA into a smaller volume to fit in the cell, (2) to reinforce the DNA macromolecule to allow cell division, (3) to prevent DNA damage, and (4) to control gene expression and DNA replication (see Jaenisch and Bird).

In general, the known developmental effects of DNA methylation on gene expression involve long-term silencing of gene expression. The attractive idea that genes are transcriptionally activated by removing DNA methylation has lacked strong experimental support until recently. DNA transcription is the first step of gene expression, in which a particular segment of DNA is copied into RNA by the enzyme RNA polymerase. Many correlations between expression and loss of DNA methylation have been reported, and the methylation of some reporters has been shown to inhibit expression (see Lee and Zhu; Johanning, Heimburger and Piyathilake; and Hauser and Jung).

Switching between active and inactive states of gene expression would also depend on transitions between different chromatin spatial conformations (or the instability of the "occupied space"). These transitions are dynamic and seem to depend on a balance between factors that sustain a silent state and those that promote a transcriptionally silent state.

Epigenetic reprogramming of the genome normally occurs during germ-cell development and ensures that the gamete genome provides an appropriate platform for the genetic program that drives embryonic development. This process refers to erasure and remodeling of epigenetic marks, such as DNA methylation, during mammalian development. Reprogramming resets the epigenome of the early embryo so that it can form every type of cell in the body. In mammals, the relation between epigenetic states and environmental signals is less defined, but both hypo- and hypermethylation have been associated with aging. It is becoming increasingly apparent that the multiple changes in cancer cells, including chromosomal instability, activation of oncogenes (genes that have the potential to cause cancer and in tumor cells they are often mutated or expressed at high levels), silencing of tumor suppressor genes, and inactivation of DNA repair systems, are caused not only by genetic, but also by epigenetic abnormalities.

Additionally, the maternal genotype also contributes to the early nutritional environment of progeny through its influence during pregnancy. It is possible that a maternal exposure in pregnancy could induce a

"metabolic cascade" to subsequent generation, whereby fetal epigenetic reprogramming could alter later adult metabolism (see Pembrey, Saffery and Bygren). But, in light of emerging evidence, it is also hypothesized that non-genetic, non-cultural effects to progeny must be extended to the traditional epidemiological approach so as to include not only early-life paternal and maternal exposures, but also ancestral exposure data (see Pembrey, Saffery and Bygren; Pembrey et al.; Kaati, Bygren and Edvinsson; Kaati et al.; Bygren, Kaati and Edvinsson; Bygren et al.).

In this sense, epidemiological findings draw attention to transgenerational effect down the male line of nutrition-related circumstances during a period of childhood with a bearing on risks for cardiovascular disease and diabetes (see Pembrey, Saffery and Bygren). An intergenerational "feedforward" control loop has been proposed that links grandparental nutrition with the grandchild's growth. This mechanism has been speculated to be a specific response, e.g., to their nutritional state, directly modifying the setting of the gametic imprint on one or more genes (see Bygren, Kaati and Edvinsson). So nutrition might induce, at some loci, epigenetic or other changes that could be transmitted to the next generation that impact health. The slow growth period (SGP) before the prepubertal peak in growth velocity has emerged as a sensitive period where different food availability is followed by different transgenerational responses (see Kaati et al.). The conclusion to be taken from these studies is that early social circumstances (food availability) might influence longevity by epigenetic mechanisms and that overfeeding of children might program their own metabolic systems for life in a manner that could be transmitted to the next generations.

The interaction between genes and regulatory mechanisms of gene expression at the epigenetic level is also important when the precautionary principle is applied in developing and consuming genetic-modified foods (GMOs). There is considerable controversy over the production and use of GMOs because of concerns over the health and the environmental, social, economic, ethical, and political effects of these foods (see Bawa and Anilakumar). These issues go beyond the scope of this work, but it is important to consider that with GMOs the natural regulatory biological processes and the "fine epigenetic adjustments" that lead to the development of phenotypes in nature are not respected, contrary to what happens with traditional improvement and the natural evolution of organisms (see Keller). How this could impact human longevity is still unknown. If we

consider that genetically manipulated organisms could represent a substantial risk to the ecological balance of nature, and also if we consider that transfers, not births, shape senescence in social species (transgenerational epigenetic inheritance), based on the evolutionary theory of aging, it would be reasonable to speculate that the future impact of GMOs on human longevity would not be negligible (see Lee).

Some scientific evidence is now suggesting that the main challenges for the future will be to assess whether environmental conditions, such as diet, influence methylation changes that occur with age in normal individuals and whether such epigenetic alterations predispose individuals to long-term diseases such as cance (see Lee and Zhu; Kaput and Rodriguez; Kaput; Johanning, Heimburger and Piyathilake; Fang et al.; Ferguson; Davis and Ross). When it comes to molecular nutrition, "nutrigenomics" refer to these challenges and can be defined as a prospective analysis of differences among nutrients with regard to the regulation of gene expression (see Gillies). The central idea is "to understand our personal uniqueness with regard to genetic variation is to open the door to managing and optimizing our health through tailored nutrition." One of the expectations of "nutrigenomics" is that a wide range of nutrient modifiable genes will be identified, validated, and incorporated into dietary strategies for the optimization of health and the prevention of disease (see Kaput).

In summary, epigenetic mechanisms constrain expression by adapting regions of the genome to maintain either gene silencing or gene activity. This is achieved through direct chemical modification of the DNA region itself and by modification of proteins that are closely associated with the locus.

This is to say that epigenetic process communicates two types of "empty space": one from inside and the other from outside a cell. This creates a continuum of metabolism that, by means of selection, provides chemical associations and dissociations and makes the delicate and complex molecular structure ("living society") become unstable. So compensation by food is a necessity for provision of energy, for the reiteration of metabolic process, and for repairing the structural instability established by the dynamic changing of the external environment.

Caloric Restriction, SOC Strategy, and Aging

One of the most robust observations in biology of aging is the ability of caloric restriction (CR) to delay or prevent a range of age-related

processes and significantly extend lifespan (see Anderson and Weindruch; Fontana, Partridge and Longo; Morley, Chahla and Alkaade; Speakman and Hambly). CR is a dietary regimen that is based on low calorie intake that, without malnutrition, has been shown to work in a variety of species to decelerate the biological aging process, resulting in longer maintenance of youthful health and an increase in both median and maximum lifespan. Essentially, every species of non-primate animals that has been studied has shown an increased lifespan when subjected to a CR diet. Yeast, protozoans, worms, flies, spiders, water fleas, chickens, and rodents all live longer when subjected to CR (see Green, Sawaya and Dollar).

The Wisconsin study of CR in primates initiated a restricted diet in adult monkeys aged 7–14 years. This would roughly correspond to humans aged 20–40 years. Based on the available evidence, initiation of CR during adulthood is likely to impart significant benefit. The population of rhesus macaques maintained at the Wisconsin National Primate Research Center has a lower incidence of age-related deaths. At the time point reported, 50% of control fed animals survived, compared with 80% survival of CR animals. Further, CR delayed the onset of age-associated pathologies. Specifically, CR reduced the incidence of diabetes, cancer, cardiovascular disease, and brain atrophy (see Colman et al.).

CR experiments in mammalian species as well as taxonomically distant organisms suggest the universality of its impact on lifespan. Given the obvious parallels between rhesus monkeys and humans, the beneficial effects of CR on rhesus monkeys may also occur in humans. This prediction is supported by studies of people on long-term CR, who show fewer signs of cardiovascular aging (see Green, Sawaya, and Dollar).

An important consideration regarding CR in humans, however, involves weighing the possible benefits of CR against known quality of life concerns. This would lead to a fundamental question: What would happen if one were to institute caloric restriction with high-quality nutrition within an environment of modern sanitation and health care?

Scientific evidence provides only partial answers to this intriguing question. In some studies, CR has been shown to promote weight loss and improve several health indices in humans while simultaneously preserving or improving bone mineral density, lean mass, strength, and aerobic capacity (see Cruzen and Colman; Dahlman et al.; Holloszy and Fontana; Fontana and Klein). Evidence also suggests that diet composition is an important factor in the CR-mediated effects on longevity (see Green,

Sawaya, and Dollar).

Despite the huge amount of evidence supporting the favorable impact of CR on longevity (see Fontana and Klein; Fontana, Partridge and Longo; Holloszy and Fontana; Anderson and Weindruch; Cruzen and Colman; Dahlman et al.; Green, Sawaya, and Dollar; Morley, Chahla and Alkaade; Colman et al.), the real biological mechanism underlying the CR effects is not completely understood. One popular idea is that CR's effects are mediated through nutrient-sensing pathways that detect availability of various nutrients as part of an evolutionary response to famine. CR-induced metabolic reprogramming may be a key event in the mechanism of lifespan extension. Improvements in metabolic function conferred by CR, specifically insulin sensitivity, have been consistent and striking. Biological characteristics of animals exposed to CR include numerous other changes in the transcriptome (the set of all RNA molecules, including mRNA, rRNA, tRNA, and other non-coding RNA transcribed in one cell or a population of cells), metabolome (the complete set of small-molecule chemicals found within a biological sample that can be a cell, a cellular organelle, an organ, a tissue, a tissue extract, a biofluid, or an entire organism), and proteome (the entire set of proteins expressed by a genome, cell, tissue, or organism at a certain time), as well as increases in stress hormones (corticosterone or cortisol depending on the species). Some of these effects represent preservation or restoration to levels typical of younger individuals (such as lower insulin and glucose concentrations), whereas, paradoxically, other changes resemble aging (such as low GH/IGF-1).

The notion of hormesis can also be invoked as one of the most relevant explanations of CR's mechanism: "The chronic presence of a low-intensity stressor improves the organism's ability to survive a more intense stressor by reducing inflammation and up-regulating genes involved in cellular protection." Up-regulation, in the biological sense, means an increase in the number of receptors on the surface of target cells, making the cells more sensitive to a hormone or another agent. This idea is consistent with the inflammatory hypothesis of human aging, but it is also reasonable in the face of the metabolic theory of aging, described above (see Green, Sawaya, and Dollar).

Hormesis can be considered a mechanism that explains the optimization of metabolic process provided by the selectivity involved in a caloric restricted diet. This selectivity can be unconscious, when the quantitative reduction of calories of a diet induces biochemical selectivity in an organism

because of the short disposal of nutrients; or conscious, when there is an active selection of healthy food, considering the influence of cultural aspects. This leads us back to Baltes, when he asserts that the demand for culture also increases in the course of life because, as individuals reach old age, their biological potentials decline. This view of "culture as compensation" is a major tenet of many evolutionary theories in cultural anthropology, but is also compatible with Whitehead's view of "food as compensation."

Finally, it is important to refer back to the introduction where it was stated that a "living society" evolves in permanent social deficit. This is compatible with the view of lifespan scholars, who proceed in their theoretical efforts from the basic assumption that human development is essentially incomplete. Whitehead proposed that completion is the perishing of immediacy, so it never really is. Incompleteness results first from the fact that biological and cultural co-evolution has not come to a standstill, but is an ongoing process. More important is the idea that incompleteness results from the fact that the biological and cultural architecture of human ontogeny is relatively undeveloped in more advanced ages (the second part of the lifespan).

Conclusions

The three points previously specified in the introduction of this work can now be recovered in a simple statement based on SOC strategy applied to ontogenetic perspectives of the biology of aging: life is a selective process provided by emptiness and which produces a continuum in a physical field that optimizes a living society evolving in permanent social deficit by means of compensation by nutritional metabolism.

Contemporary developmental and biological theories, when applied to aging, show a fundamental characteristic of a "living society": it requires food. Food involves societies to be destroyed by other societies; food involves compensation in initial and final phases of ontogenesis; food involves an interplay of societies and environment; food, according to Whitehead, turns life into a robbery process in need of justification.

In a sense, caloric restriction, taken as "caloric selective optimization," is a fundamental process in the course of aging, not only because of the amount of empirical evidence supporting its efficiency, but also because of its theoretical ontogenetic basis.

In accordance with Whitehead's doctrine of life, the primary meaning of life is the origination of novelty, what he calls "novelty of appetition." This means that a "living society" is one which includes some "living occasions" that flow through the emptiness. But at the same time, life is a bid for freedom, life is the name for originality and not for tradition. Mere response to stimulus is a characteristic of all societies, living or not. The main characteristic of life is its reaction adapted to the capture of intensity, under a large variety of circumstances. So, to match intensity with survival, life puts a paradoxical problem for Nature: the construction of highly complex structured societies, which need robbery and deconstruction.

This is why food could be taken as Nature's gift, a potential for deconstruction (and death) that provides a noble and fundamental service to life. This is the point where dietetics becomes ethics.

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