

## A review of anthropological approaches to ageing

*Maria Kaczmarek, Anita Szwed*

### *Abstract*

It is evident that the pattern of ageing among humans has a unique character. Therefore, when undertaking any research on human ageing one has to specify a proper methodology and methods which are available in the anthropological perspective. The paper is aimed at providing a review of anthropological approaches to the study of ageing. On the basis of the meaning and scope of the concept of ageing, its sources and causal factors are discussed. Further, functional, physiological and morphological indicators of ageing are briefly described. Much attention is focused on the concept of biological age which is the key notion for assessment of variation in the rates of human ageing.

Maria Kaczmarek, Anita Szwed, 1997; *Anthropological Review*, vol. 60, Poznań 1997, pp. 35–46, figs 2, tables 2. ISBN 83-86969-18-0, ISSN 0033-2003

*“Eos, a mythological goddess, asked Zeus to allow Tithonus, the mortal she loved, to live forever. Zeus granted Tithonus immortality, and the lovers lived happily – but not ever after. Tithonus began to grow old until he became so infirm that he could not move. Yet he was denied the gift of death, and to this day he lives on, long after Eos had finally left him, ‘a helpless, drivelling vegetable’. Eos had made a grievous error – she had forgotten to ask Zeus to grant her lover eternal youth along with eternal life...”*

(from Greek Mythology)

### **The meaning and scope of ageing**

An organism does not remain the same biologically over its life span. The structure and function of one’s biological processes undergo various changes. Some of these changes are termed growth, some development, others maturation or ageing. Although these processes occur simultaneously, they have slightly different biological meaning. Growth is defined as an irreversible,

quantitative increase in size or mass, involving the production of new protoplasm. Development is defined as a progression of changes, either quantitative or qualitative, that lead from an undifferentiated state to a well integrated, highly organized, matured form. Maturation in this approach means a functional capacity of the organs and the entire organism [BOGIN 1993:2]. When defining ageing, one should consider two quite distinct approaches to the definition. The first is through an individual level and describes a set of deleterious changes which occur primarily in the postreproductive period.

They are manifested as a gradual decline in a variety of body functions. The other way of defining ageing refers to the population level and means that the overall process of all deleterious changes affects the ability of the organism to survive, which is manifested in the increase of an age-specific death rate [COMFORT 1979, STREHLER 1978, KIRKWOOD, HOLLIDAY 1986]. This is strictly meaningful only for a species where the individual organism is clearly defined and where the individuals are capable of reproducing repeatedly during their lifespan. Moreover, it is noteworthy that ageing is not intrinsic to all living organisms. For instance, due to some indefinite regenerative mechanisms, Prokaryotic and many Eucaryotic microorganisms are able to escape senescent changes [COMFORT 1979].

Going further into our considerations, the population concept of ageing may be introduced in terms of a survivorship pattern, as shown in Fig. 1. The pattern is well illustrated with an age-specific mortality rate curve and a survival curve which are plotted against the lifespan of a typical human population. Both curves, presented in Fig. 1 are closely approximated by a simple exponential (Gompertzian curves) [GOMPERTZ 1825 quoted after KIRKWOOD, HOLLIDAY 1986, COMFORT 1979].

It is evident from Fig. 1 that the pattern of survivorship and mortality rate in humans is characterized by reasonably high survival and low mortality for the earlier part of the lifespan, followed by, respectively, a steady decline and increase thereafter, until the age when survivorship reaches zero and the mortality rate reaches its maximum. This age has a constant value for the species. In-

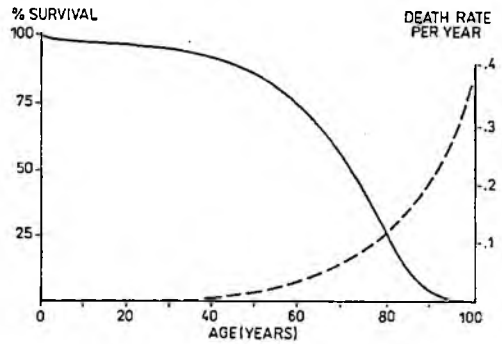


Fig. 1. Survivorship (continuous line) and mortality rate (dashed line) as function of age for a typical human society

tuitively, underlying the population concept of ageing is the idea that the process of ageing involves the entire life course. In this instance, growth may be seen as a part of ageing. According to Riley: "Ageing is a life-long process of growing up and growing old. It starts with birth (or conception) and ends with death... Ageing consists of three sets of processes – biological, psychological, and social; and these three processes are all systematically interactive with one another over the life course" [RILEY 1979:4].

The process of ageing is familiar in human societies and has attracted many investigators for a long time. The unique pattern of ageing in humans, viz. the extent to which ageing is manifested and the existence of a clearcut menopause, demands an appropriate methodology and techniques for the assessment of this process. The anthropological perspective of this survey reveals the nature and peculiarities of human ageing as far as the individual and the population are concerned. It also displays the associations of life-style, well-being and nutritional status with determinants of variation in rates of ageing. This article provides

readers with an overview on the methodology and methods of the ageing research in modern human populations.

### Why do we age? The origin of ageing

Considering the idea that changes associated with biological ageing are those associated with a lessened chance of survival, one may ask about the causes of this decreasing survival potential of the population. There are a number of biological theories which focus on the proper answer to this question. However, it seems that each of these theories of ageing is incomplete as it offers a successful explanation of only a portion of the ageing phenomenon, moreover, none of them has achieved general acceptance. Nevertheless, they are not mutually exclusive, and there is a great deal of overlap among them. Furthermore, they share the common characteristics which have a genetic basis [SHOCK 1977]. Support for the genetic basis of ageing comes from Hayflick's experiments which show that certain cells of the body, grown *in vitro*, are only able to divide a limited number of times. He observed that the older the individual from whom the cell samples were obtained, the fewer doublings the cells would undergo. These two facts corroborate the concept of the genetic basis of ageing [HAYFLICK 1965]. In most of the theories on biological ageing two basic lines of thought may be distinguished. The first represents the deterministic approach and postulates that ageing is an innate, genetically programmed process and as such is subject to only minor modifications. The other one follows the stochastic approach and postulates that ageing is the result of the

accumulation of harmful products of metabolism in tissues. These two methodologically different approaches have different implications. If ageing is considered to be a programmed phenomenon, nothing much can be done to modify this process. On the other hand, if ageing is taken to be a consequence of stresses, it may be possible to modify the life pattern by eliminating some of those stresses. It seems that the truth is likely to lie in a combination of these two approaches [PRINZINGER 1996].

While it is clear that ageing has a genetic basis, there are a large number of various causes indicated as the most essential for this process. Considering this, the biological theories may be classified into three major categories: genetic cellular theories, nongenetic cellular theories, and physiological theories [SHOCK 1977]. According to the genetic cellular theories, the major causes of ageing may be perceived in the damage to the genetic information involved in the formation of cellular proteins. Several theories have argued that it is the breakdown of these basic genetic mechanisms which cause ageing [ORGEL 1963, CURTIS 1966, HAHN 1970, SINEX 1974, STREHLER 1978]. The nongenetic cellular theories focus their interest on changes that take place in the cellular proteins after they have been formed. They suggest that ageing results from the accumulation of deleterious substances in the cells of the organism, or from damage to cell proteins (HARMAN [1954], Failla 1958, Szilard 1959; cited after PRINZINGER [1996], BJØRKSTEN [1968]). A set of physiological theories suggest that ageing results from the failure of some physiologically coordinating system, such as the immunological or endocrine

systems, to integrate bodily functions properly [FINCH 1976]. While each of these theories focuses on a different aspect of the ageing process, they may be found to share some common points. According to Strehler, the integrations are as follows: the loss of the ability of a cell to divide which is programmed in the genetic code of the cell, the loss of some of the copies of a group of genes responsible for protein synthesis essential for cell repairing from environmental damage, an increased accumulation of damaged enzymes and lipofuscin which impair the cell function, and decreased normal functioning at the cellular level. These processes lead to the decreased integration among the physiological systems of the body; ultimately, the systems break down and the individual dies [STREHLER 1978].

Whatever hypothesis on ageing is taken into consideration, it is true that ageing is one of the great paradoxes of nature. As Williams states, "It is indeed remarkable that after a seemingly miraculous feat of morphogenesis a complex metazoan should be unable to perform the much simpler task of merely maintaining what is already formed" [WILLIAMS 1957:398]. An attempt at resolving this problem refers to the explanation in terms of evolutionary process, e.g. natural selection (Weismann 1891, cited after KIRKWOOD, CREMER [1982]). There are two possible explanations: either adaptive or non-adaptive in nature. If ageing is seen as a beneficial trait in its own right, the explanation can be labelled as an adaptive one. If ageing is seen as detrimental, or at best neutral, and so its evolution explained indirectly, the explanation is non-adaptive in character. Explicit formulations of the adap-

tive view are rare, despite the fact that this type of theory is probably the more popular. The difficulty is that, for the individual, ageing is clearly disadvantageous, since senescence must reduce life expectancy and hence diminish the opportunity for reproduction. Among the beneficial characteristics of ageing is the fact that this process accelerates the turnover of generations and thereby increases the chance of a species adapting to a change in its environment, whereas without ageing the species might overcrowd its environment and exhaust its resources [KIRKWOOD, CREMER 1982, STEARNS 1992, PRINZINGER 1996].

Considering non-adaptive theories of the evolution of ageing, again two lines of thought may be distinguished: one suggesting that natural selection is simply unable to prevent the deterioration of older organisms because it becomes attenuated with age, and another suggestion that ageing is a by product of selection for other beneficial traits. These two views share a common principle originally pointed out by Haldane and later clearly enunciated by Medawar which asserts that the force of natural selection reduces progressively with age [HALDANE 1941, MEDAWAR 1981]. The reason for this is that selection acting early in life will affect a greater proportion of individuals than genes acting late, when the proportion of survivors is smaller and the remaining fraction of their lifetime expectation of reproduction is less. Medawar proposed that the attenuation of the force of natural selection with age was sufficient by itself to explain the evolution of ageing. He suggested that selection acting on genes with age-specific times of expression would tend to defer the age of expression of harmful

genes, so as to minimize their potential for deleterious effects. However, there may be an accumulation of deleterious genes which in the normal environment would combine to handicap any individual severely. This is a process, as Medawar claims, which could be accounted for by the evolutionary origin of senescence.

The second sub-type of non-adaptive theory, that ageing is a by-product of selection for other beneficial traits, was formulated in general terms by WILLIAMS [1957]. Williams' theory was derived from an argument similar to Medawar's, except for the crucial difference that the genes in question were assumed to be pleiotropic; the same genes having both, good effects in early life and bad effects in late. It was argued that natural selection should favour retention of the genes on the basis of their benefits, but defer as far as possible the time of the expression of their deleterious effects to ages when survivorship was low. In Williams' theory, ageing was attributed to the positive action of selection on the pleiotropic genes, but was not in itself regarded as beneficial. In conclusion he stated that even in the losses caused by environmental mortality the events occurring at later ages had lesser evolutionary significance.

Among the non-adaptive theories, the one which offers the most specific mechanism to account for the gene evolution of ageing, is the disposal soma theory [KIRKWOOD, 1977]. This theory postulates that the optimum investment of resources in somatic maintenance and repair is always less than the minimum which would be required for indefinite somatic survival. In con-

sequence, ageing results from a progressive accumulation of somatic defects and damage.

Table 1 gives a summary of the discussed explanations of the origin and causes of ageing.

### **How do we age? A defence of a multifactorial concept of ageing**

Ageing is a multifactorial process which takes place simultaneously at each level of the living structure in the course of an individual life. The distinguished levels are: subcellular, cellular, tissue, organs, individual and population [SUSANNE 1986]. There are structure specific manifestations of the process of ageing which are presented in Table 2.

Considering some of the molecular, cellular and physiological changes that may be responsible for the growth and ageing processes, it may be noted that these changes affect virtually all major systems of the human body: skeletal, muscle, skin, pulmonary, cardiovascular, neural, endocrine, reproductive, gastrointestinal, and excretory. It is quite evident that, although biological changes associated with growth and aging are inevitable and universal they do not affect all the systems equally. There is a wide variation in the rate of growth and in the de-

Table 1. Summarized representation of the theories of causal factors and origin of ageing

1. Causes of Ageing	
Stochastic approach	Deterministic approach
<i>Thesis: Ageing is an accidental consequence of depletion and accretional processes</i>	<i>Thesis: Ageing is an innate, genetically programmed process</i>
The tear and wear theory [PEARL 1927]	The absolute metabolic theory [RUBNER 1908]
The free radicals theory [HARMAN 1956]	The cell doubling theory [HAYFLICK 1969]
The collagen-/cross-linkages theory [VERZAR 1957]	
The somatic mutation theory [FAILLA 1958, SZILARD 1959]	
The error and fidelity theory [ORGE 1963]	
The immune theory [WALFORD 1969]	

2. Origin of Ageing	
Adaptive approach	Non-adaptive approach
<i>Thesis: Ageing is a beneficial trait in its own right</i>	<i>Thesis: Ageing is detrimental, or at best neutral; its evolution must be explained indirectly</i>
	<p>The force of natural selection reduces progressively with age and is unable to prevent the deterioration of older organisms</p> <p>The evolutionary origin of senescence theory: [MEDAWAR 1982]</p>
	<p>Ageing is a by-product of selection for other beneficial traits</p> <p>The pleiotropic origin of senescence theory: [WILLIAMS 1957]</p> <p>The disposable soma theory: [KIRKWOOD, HOLLIDAY 1979]</p>

Table 2. Indicators of the process of ageing at various levels of a living organism

Level	Manifestation
Subcellular	Biochemical changes in various subcellular structures
Cell	Changes or loss of cells
Tissue	Histochemical changes in tissue
Organ	Decrease in functional ability
Individual	Decrease in the adaptive ability to stress
Population	Increased probability of death

breathing capacity, maximum work rate and maximum oxygen.

There have been numerous cross-sectional analyses of age-specific size and physique changes designated as the processes of ageing. Data from longitudinal studies indicate the fourth decade of life as the critical point when decrement

in stature begins, but there is of course considerable variability in the time of the onset of stature decline. It has been hypothesized that the decline in stature with age results from vertebral body fractures, compression of intervertebral discs, and postural changes [FRIEDLANDER et al. 1977, BORKAN et al. 1980]. There is also

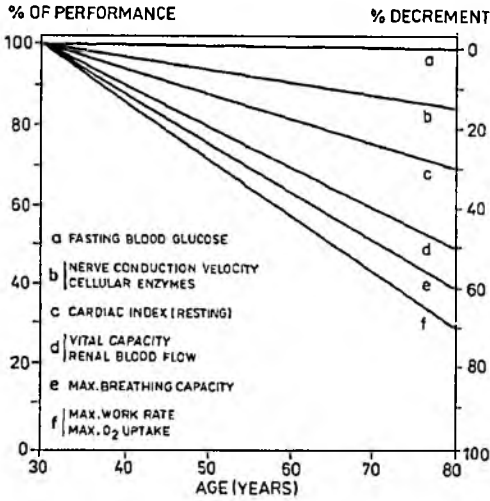


Fig. 2. Schematic linear representation of the decline in various physiological functions with age [SHOCK 1977]

a longitudinal decline with age in measurements of the extremities and other body length and breadth dimensions [DAMON 1972]. A secular trend was found in this process [HIMES, MUELLER 1982, BORKAN et al. 1983]. The effects of the secular trend are also found in body size.

Senescent changes in body weight display a clear pattern of sexual dimorphism. While in males a decrease in body weight begins after the age of 55, in females it starts at a later age and advances more slowly thereafter [DAMON et al. 1977, ROSSMAN 1977]. The percentage of body fat increases into late adulthood, although in absolute terms fat remains rather constant while the lean body mass decreases, thus accounting for the increase in relative fatness [NORRIS et al. 1963, MALINA 1969, ROSSMAN 1977, PARIZKOVA, EISELT 1980]. Despite the relative constancy of absolute fat mass, there are changes in fat distribution throughout the body. There is an age

specific tendency to a larger amount of intra-abdominal fat and a thinning of subcutaneous trunk fat [GARN & YOUNG 1956, BORKAN & NORRIS 1977, MUELLER 1982].

The results of numerous works demonstrate a substantial modification of the skeleton in late adulthood characterized by considerable loss of bone mass (osteoporosis). The causes of osteoporosis are presently unknown, although a number of explanations are being proposed. As the incidence of osteoporosis is considerably higher in females, there are some suggestions of a link between bone loss and estrogen, a link between postmenopausal calcium loss through the parathyroid hormone and between degree of bone loss and growing sensitivity to the parathyroid hormone with age [WISKE et al. 1979].

The demography of ageing is also a domain of anthropological interest. It is a specific pattern of the human demographical structure that females outlive males [BIELICKI et al. 1994]. Among living creatures, humans are those who have the highest lifespan potential. An ageing population is defined as one in which the triangle-shaped demographic pyramid moves to a more rectangular one as a consequence of the increase in the mean age of its members over time. There is a considerable variation in life expectancy (as opposed to the life span) among contemporary human populations, even when the distorting effects of infant mortality are removed. It is also claimed that the longer life span in women has no perceptible influence on the upper age limit of fertility. According to Malina, the age at menopause has remained in the 45 to 50 range at least since the Graeco-Roman times [MALINA 1979].

## **An assessment of variation in the rates of human ageing**

A major goal in biological gerontology is the description of normal ageing. The traditional belief that disease is pathological while ageing is normal is beginning to be questioned by human biologists, since the precise point at which several conditions constitute a disease is problematic and the boundary between disease and normality is very subtle (for instance atherosclerosis which is present in many individuals at the age of thirty). It is emphasized that every physiological process involved in the maintenance of homeostasis becomes less effective with increasing age. It is characteristic of biological systems that each has a certain redundancy of functional capacity or a functional reserve. There is also a minimum level required to sustain a death threshold (cut-off point). When vitality or functional capacity drop below this critical point death occurs. The level of the death threshold reflects the severity of the conditions to which an individual is exposed. As the environmental challenges and stresses that individuals cope with are never constant, the death threshold fluctuates around a mean value. It is logical, that if conditions are severe, the threshold will be higher, and life shorter. The process of ageing can be modified by an appropriate intervention, such as lifestyle, diet, or activity. The extent of interventions can influence the rate of ageing. The latter may vary considerably among individuals. The understanding of such interpersonal variations may reveal the special characteristics of individuals who retain their functional capacities to age slowly or fast. Estimating the rate of

ageing of an individual is of considerable interest because it is important for identifying basic ageing processes, and thus for preventive medicine. The most prevalent approach to the assessment of ageing in individuals is the concept of biological age, widely used by auxologists for assessing normal child growth [TANNER 1992]. The basic point of this concept is that the normal sequence of developmental processes is provided by chronological age. The index of biological age in adults has a slightly different interpretation, as it must reflect the probability of death at any chronological age; this probability increases with chronological age after about the first year of life [BROWN, FORBES 1976]. Some mathematical models, the multiple regression procedure, have been proposed which relate the progressive decline in physiological functions to the increased risk of death with age [BROWN, FORBES 1974, FURUKAWA et al. 1975]. These models are built under the assumption that there is a risk factor, corresponding to an index of biological age, the mean value of which may change with age for the population. There is also a cut-off level in the distribution of the parameter. The individuals for whom those values lie on the unfavourable side of this cut-off level are at an increased risk of disease or death. However, the multiple regression analysis approach has met with severe criticism [COSTA, MCCRAE 1980]

Another approach to the estimation of biological age of adult is to transform data from any battery test in the pattern profile system [BORKAN, NORRIS 1980]. Twenty four physiological, morphological and functional parameters were selected from the data bank as a representative subset of the characteristic changes



of ageing. The criteria for selection were that the variables had a positive or negative linear trend with age, good reliability and reflected a wide range of physical functions. Analyses were performed using data on lifestyle to delineate subgroups for comparison as mean biological age profiles. The results obtained indicate that men who engaged in organized physical activity were biologically more youthful than those who did not. Other comparison showed that being married and better educated were also associated with greater biological youthfulness [BIELICKI et al. 1988, ROGUCA 1995]. This approach, although successful in comparison between different groups is limited in use, since the data come from cross-sectional studies. The data from cross-sectional study are efficient for description of the status of biological phenomena at a particular moment of time but not for the rate of the ageing processes. The latter one requires some additional assumptions and is based on the data from longitudinal examination of actual change with age. Although, such studies are known to be difficult and time consuming but may provide evidence on how to delay deleterious changes.

Summarizing, it may be said, that the ageing processes in human species are a subject of great popular interest and anthropological approaches to the study of ageing seem to offer considerable potential for the future.

## References

- BIELICKI T., Z. WELON, W. ŻUKOWSKI, 1988, *Problemy nierównowagi biologicznej warstw społecznych*, Materiały i Prace Antropologiczne, 109, 123–140
- BIELICKI T., CZ. BRAJCZEWSKI, E. ROGUCA., Z. WELON, 1994, *Niektóre społeczne i ekologiczne uwarunkowania przedwczesnej umieralności w Polsce*, Monografie Zakładu Antropologii PAN, 12, Wrocław
- BJØRKSTEIN J., 1968, *The cross-linkage theory of aging*, Journal of the American Geriatrics Society, 16, 408–427
- BOGIN B., 1993, *Patterns of human growth*, Cambridge University Press
- BORKAN G.A., A.H. NORRIS, 1977, *Fat redistribution and the changing body dimensions of the adult male*, Human Biol. 49, 495–514
- BORKAN G.A., A.H. NORRIS, 1980, *Assessment of biological age using profile of physical parameters*, J. Geront. 35, 177–184
- BORKAN G.A., D.E. HULTS, R.J. GLYNN, 1983, *Role of longitudinal change and secular trend in age differences in male dimensions*, Human Biology, 55, 629–641
- BROWN K.S., W.S. FORBES, 1974, *A mathematical model of aging processes*, J. Geront. 29, 46–51
- BROWN K.S., W.S. FORBES, 1976, *Concerning the estimation of biological age*, Gerontology, 22, 428–437
- COMFORT A., 1979, *The Biology of Senescence*, 3rd ed. Churchill Livingstone, Edinburgh
- CURTIS H.S., 1966, *Biological mechanisms of aging*, Charles C. Thomas, Springfield, Ill
- COSTA P.T. Jr, R.R. MCGRAE, 1980, *Functional age: a conceptual and empirical critique*, [in:] S.G. Haynes, M. Feinleib (eds.), *Epidemiology of Aging*, NIH Publication No 80-969, Bethesda
- DAMON A., 1972, *Predicting age from body measurements and observations*, Aging Human Dev., 3, 169–173
- DAMON A., C.C. SELTZER, H.W. STOUTD, B. BELL, 1972, *Age and physique in healthy white veterans at Boston*, J. Gerontol. 27, 202–208
- FRIEDLANDER J.S., P.T. COSTA Jr, R. BOSSE, E. ELLIS, J.G. RHOADS, H.W. STOUTD, 1977, *Longitudinal physique changes among healthy white veterans at Boston*, Hum. Biol. 49, 541–558
- FURUKAWA T., M. INOUE, F. KAJIYA, H. INADA, S. TAKASUGI, S. FUKUI, H. TAKEDA, H. ABE, 1975, *Assessment of biological age by multiple regression analysis*, J. Geront. 30, 422–434
- GARN S.M., R.W. YOUNG, 1956, *Concurrent fat loss and fat gain*, Am. J. Phys. Anthropol. 14, 497–504

- HAHN H.P., 1970, *The regulation of protein synthesis in the aging cell*, *Experimental Gerontology*, 5, 323
- HALDANE J.B.S., 1941, *New Paths in Genetics*, Allen & Unwin, London
- HARMAN D., 1956, *Aging: a theory based on free radical and radiation chemistry*, *J. Gerontology*, 11, 298-300
- HAYFLICK C.C., 1965, *The limited in vitro lifetime of human diploid cell strains*, *Experimental Cell Research*, 37, 614-636
- KIRKWOOD T.B.L., 1977, *Evolution of Ageing*, *Nature*, 270, 301-304
- KIRKWOOD T.B.L., T. CREMER, 1982, *Cytogerontology since 1881: a reappraisal of August Weismann and a review of modern progress*, *Human Genetics*, 60, 101-121
- KIRKWOOD T.B.L., R. HOLLIDAY, 1986, *Ageing as a consequence of natural selection*, [in:] *The biology of human ageing*, A.H. Bittles and K.J. Collins (eds.), Cambridge University Press, 1-17
- MALINA R.M., 1969, *Quantification of fat, muscle and bone in man*, *Clin. Orthop. Rel. Res.* 65, 9-38
- MALINA R.M., 1979, *Secular changes in size and maturity: causes and effects*. *Mon. Soc. Res. Child Dev.* 44, 179, 59-102
- MEDAWAR P.B., 1982, *The Uniqueness of the Individual*, Dover, New York
- MUELLER W.H., 1982, *The changes with age of the anatomical distribution of fat*, *Assoc. Sci. Med.*, 16, 191-196
- NORRIS A.H., T. LUNDY, N.W. SHOCK, 1963, *Trends in selected indices of body composition in men between ages of 30 and 80 years*, *Ann. N.Y. Acad. Sci.* 110, 623-639
- ORGEL L.E., 1963, *The maintenance of the accuracy of protein synthesis and its relevance to aging*, *Biochemistry*, 49, 517-521
- PARIZKOVA J., E. EISELT, 1980, *Longitudinal changes in body build and skinfold in a group of old men over a 16 year period*, *Human Biology*, 52, 803-809
- PRINZINGER R., 1996, *Das Geheimnis des Alterns Die programmierte Lebenszeit bei Mensch, Tier und Pflanze*, Campus Verlag, Frankfurt, New York
- RILEY M.W., 1979, *Aging from birth to death: Interdisciplinary perspectives*, Westview Press, Boulder, Colo.
- ROGUCKA E., 1995, *Uwarunkowania społeczne nadumieralności mężczyzn w Polsce*, *Monografie Zakładu Antropologii PAN*, 13, Wrocław
- ROSSMAN I., 1977, *Anatomic and body composition changes with aging*, [in:] *Handbook of the Biology of Aging*, Finch CE, Hayflick (eds.) Van Nostrand Reinhold Co, New York, 189-221
- SHOCK N.W., 1977, *System Integration*, [in:] *Handbook of the Biology of Aging*, Finch CE, Hayflick (eds.) Van Nostrand Reinhold Co, New York, 26-34
- SINEX F.M., 1974, *The mutation theory of aging*, [in:] *Theoretical aspects of aging*, M. Rockstein (ed.), Academic Press, New York
- STEARNS S.C., 1992, *The evolution of life histories*, Oxford University Press
- STREHLER B.L., 1978, *The mechanism of aging*, *The Body Forum*, 3, 44-45 Oxford University Press
- SUSANNE C., 1986, *Senescence: effects anthropologiques*, [in:] *L'homme, son évolution, sa diversité*, D. Ferembach, C. Susanne, M-C. Chamla (eds.), Editions du CNRS, Paris, 369-382
- TANNER J.M., 1992, *Human growth and constitution*, [in:] *Human Biology: an introduction to human evolution, variation, growth and ecology - 3rd ed.*, G.A. Harrison, J.M. Tanner, D.R. Pilbeam, P.T. Baker (eds.), Oxford University Press, London, New York, Sydney, 339-435
- WISKE P.S., S. EPSTEIN, N.H. BELL, S.F. QUENNR, J. EDMONDSON, C.C. JOHNSTON Jr, 1979, *Increase in immunoreactive parathyroid hormone with age*, *New Engl. J. Med.*, 300, 1419-1421
- WILLIAMS G.C., 1957, *Pleiotropy, natural selection and the evolution of senescence*, *Evolution*, 11, 398-411
- WYNNE-EDWARDS V.C., 1962, *Animal Dispersion in relation to Social Behaviour*, Oliver & Boyd, Edinburgh

## Streszczenie

Pragnienie nieśmiertelności i tęsknota za wieczną młodością wyrażona, między innymi w zacytowanym na wstępie fragmencie mitologii greckiej, a równocześnie świadomość nieuchronności procesów starzenia się i śmierci pasjonowały ludzi od zarania dziejów. Proces starzenia się jest doskonale znany we wszystkich populacjach ludzkich, a ujęcie antropologiczne w ramach gerontologii, nauki o starzeniu się i starości żywych organizmów, pozwala poznać naturę i osobliwości procesu starzenia się człowieka, jej źródła i przyczyny oraz implikacje tego procesu dla jednostek i społeczeństwa. Celem niniejszej pracy jest przedstawienie koncepcji wyjaśniających źródła i przyczyny procesu starzenia się organizmów w ujęciu antropologicznym.

Organizm w toku ontogenezy podlega ciągłym zmianom, których przejawem jest wzrost, rozwój, dojrzałość, starzenie się. Wprawdzie procesy te przebiegają równocześnie, jednakże ze względu na istotę leżących u ich podstaw zjawisk biologicznych przyjęto dla nich odrębne definicje. Wzrost określa się jako nieodwracalne zmiany natury ilościowej, włączając w to produkcję plazmy, prowadzące do zwiększania się masy lub wymiarów danego organizmu. Rozwój jest określanym jako progresywne zmiany ilościowe lub jakościowe, które prowadzą od prostej formy do wysoko zorganizowanej formy dorosłej. Stan, w którym organizm osiąga formę dorosłą nazywa się dojrzałością. Tak więc, dojrzałość oznacza funkcjonalną zdolność organów i całego organizmu do pełnienia określonych funkcji. Starzenie się żywego organizmu określa się jako powolne, nieodwracalne zmiany koloidalnej struktury materii. Wyrazem tych zmian są procesy degeneracyjne powodujące osłabienie czynności układów enzymatycznych i hormonalnych. Objawem tych zaburzeń są zmiany czynności wszystkich układów ustroju, takich jak układ nerwowy, hormonalny, krążenia, oddychania. Ujmując naturę procesów starzenia się w perspektywie antropologicznej, zjawisko to opisuje się i wyjaśnia na poziomie osobniczym oraz na poziomie populacyjnym. Starzenie się osobnika określa się na podstawie zmian degeneracyjnych, które pojawiają się przede wszystkim w okresie postreprodukcyjnym. Przejawiają się one stopniowym spadkiem sprawności różnych funkcji życiowych. Drugie ujęcie odwołuje się do poziomu populacyjnego i zgodnie z tym podejściem starzenie się oznacza całość procesów destrukcyjnych, które w efekcie prowadzą do zwiększania się prawdopodobieństwa śmierci. Starzenie się nie jest właściwością immanentną wszystkim organizmom żywym. Prokaryota i proste Eucariota nie podlegają procesom starzenia się, gdyż wytworzyły mechanizmy regeneracji, które umożliwiają im nieograniczony rozwój. Populacyjną koncepcję procesu starzenia się organizmu można przedstawić graficznie jako krzywe przeżywania i umieralności. Na rys. 1 zaprezentowano krzywe przeżywania i wymierania, w zależności od wieku, dla przeciętnej populacji ludzkiej. Krzywe te odzwierciedlają specyficzne dla określonych kategorii wieku tempo wymierania oraz przeżywania; w pierwszym okresie życia człowieka charakteryzuje się ono wysokim stopniem przeżywalności i niską wymieralnością, następnie stopniowym i stałym spadkiem przeżywalności oraz wzrostem umieralności aż do wieku, w którym przeżywalność osiąga zero a wymieralność osiąga swoje maksimum. Ten wiek ma stałą wartość dla określonych gatunków. Można więc powiedzieć, że starzenie się jest procesem przebiegającym w ciągu całego życia osobniczego, wraz z wiekiem rośnie prawdopodobieństwo śmierci osobnika. Zaproponowano wiele teorii objaśniających przyczyny procesu starzenia się organizmu, jednak żadna z proponowanych teorii nie jest kompletna i nie wyjaśnia tego zjawiska w pełni. Wszystkie proponowane wyjaśnienia łączy to, że odwołują się do genetycznej determinacji procesu starzenia się, przy czym jedne z nich ujmują ten proces jako deterministyczny, a więc nieunikniony, inne jako stochastyczny. Zgodnie z pierwszym podejściem badawczym proces starzenia się nie jest modyfikowalny i jakiegokolwiek działania w kierunku opóźnienia procesu starzenia się człowieka skazane są na niepowodzenie. Przyjmując, iż proces starzenia się ma charakter stochastyczny, możliwa jest wówczas jego modyfikacja; zależy ona od stopnia zagrożenia organizmu przez warunki środowiska, w jakim ten organizm się rozwija. Wśród postulowanych teorii wyjaśniających źródła i przyczyny starzenia się organizmu można wyróżnić trzy kategorie: teorie genetyczne, niegenetyczne i fizjologiczne. Teorie genetyczne starzenia się organizmu upatrują źródło przyczyn starzenia się w uszkodzeniach materiału genetycznego, teorie niegenetyczne natomiast postulują, iż przyczyną starzenia się jest nagromadzenie substancji szkodliwych dla organizmu, które w dawkach w jakich występują przyczyniają się do rozpadu organizmu, teorie fizjologiczne sugerują że starzenie się jest wynikiem degeneracji systemów fizjologicznych (immunologicznego, endokrynnego).

W ujęciu ewolucyjnym starzenie się jest zjawiskiem paradoksalnym, którego biologia ewolucyjna nie potrafi przekonująco wyjaśnić. Istnieją dwa możliwe wyjaśnienia: adaptacyjne lub nieadaptacyjne. Jeśli przyjąć, że proces starzenia się jest korzystny dla populacji, wyjaśnienie będzie miało charakter adaptacyjny. Jeśli u podstaw wyjaśniania leży założenie, że starzenie się jest procesem szkodliwym lub co najwyżej neutralnym, wówczas jest to wyjaśnianie nieadaptacyjne. Wyjaśnianie adaptacyjne opiera się na stwierdzeniu, że starzenie się przyspiesza wymianę pokoleń, tym samym zapobiega przeludnieniu i wyczerpaniu zasobów środowiska. Teorie nieadaptacyjne postulują, że starzenie się jest ubocznym produktem selekcji dla innych korzystnych cech (teorie Williama, Medawara). Interesującą kon-

cepcja jest propozycja Kirkwooda („disposable soma theory”), w której postuluje się, że optymalna inwestycja w zachowanie somatycznej równowagi i reperacji szkód jest zawsze mniejsza niż minimum, które byłoby wymagane do nieskończonego trwania organizmu. Omawiane wyżej teorie starzenia się prezentuje tabela 1.

Proces starzenia się organizmu przebiega w różnym tempie i na różnych poziomach, od struktur komórkowych do poziomu populacyjnego. Tę wielopoziomowość procesu starzenia się ilustruje tabela 2. Tempo procesu starzenia się organizmu ocenia się na podstawie kryterium morfologicznego, fizjologicznego i funkcjonalnego, na przykład, spadek wydolności organizmu z wiekiem określany na podstawie funkcji fizjologicznych poszczególnych układów przedstawiono schematycznie na rys. 2. Stopień wyrażenia cech stanowiących podstawę wymienionych kryteriów oceny procesu starzenia się bada się w ujęciu przekrojowym lub podczas obserwacji długofalowych. Badania przekrojowe pozwalają wnioskować o stanie organizmu w określonym momencie czasowym, natomiast dynamikę procesu starzenia się można określić tylko i wyłącznie na podstawie obserwacji z badań długofalowych. Są one trudne do realizacji, czasochłonne i kosztowne ale coraz częściej postuluje się tego typu badania aby móc odpowiedzieć na pytania, na przykład w jakim stopniu określony styl życia przyczynia się do przyspieszania lub opóźniania procesów starzenia się.

Podstawę teoretyczną oceny procesu starzenia się stanowi koncepcja wieku biologicznego, który w odniesieniu do fazy stabilnej i inwolucyjnej ma inne znaczenie niż w odniesieniu do fazy progresywnej. Wiek rozwojowy w odniesieniu do fazy stabilnej i inwolucyjnej ontogenezy człowieka pojmuje się jako zwiększające się prawdopodobieństwo śmierci osobnika, empirycznie wyznaczane za pomocą modelu regresji wielokrotnej. Ten sposób postępowania poddano krytyce [Costa i McCrae 1980] a Borkan i Norris [1980] zaproponowali metodę tak zwanych profili. W metodzie tej wykorzystuje się kilka z określonego zestawu cech fizjologicznych, dla których wykreśla się profile grupując osobników w zależności od interesującego nas aspektu (np. aktywni fizycznie, nieaktywni).

W podsumowaniu powyższych rozważań należy stwierdzić, że mimo niedoskonałości koncepcji oraz metod służących do oceny stanu i dynamiki procesu starzenia się człowieka, antropologia oferuje nowe perspektywy badawcze tego fascynującego zjawiska.