PSYC 336
Developmental Psychology II

Session 2 – Theories of ageing

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Session Overview

- Several theories have attempted to provide explanation for why we age. Although many theories are not mutually exclusive, they enhance our understanding of reasons behind ageing. In this session, the focus will be on theories such as the Rate-of-living theories, Cellular theories and Programmed cell death theories. We will also discuss the implications of these theories on some age-related diseases.

Session Outline

The key topics to be covered in the session are as follows:

- Introduction to theories of ageing
- Rate-of-Living Theories
- Cellular Theories
- Programmed-cell-death theories
- Other theories

Reading List

- Read Chapter 2 of Recommended Text – Adult development and aging, Cavanaugh & Blanchard-Fields (2006).

Topic One

INTRODUCTION TO THEORIES OF AGEING

Introduction to theories of aging

- Can you give me examples of physical changes people undergo as they age?
- What are some of the impacts of those changes?
- Why do we age?
- What are some normal and abnormal physical changes associated with aging?
- What factors influence how we age?
- Many theories that are **not mutually exclusive.**

Theories of ageing

- Theories of ageing attempt to explain the phenomenon of aging as it occurs over the lifespan
  - aging is viewed as a total process that begins at conception
  - *senescence:* a change in the behavior of an organism with age leading to a decreased power of survival and adjustment
- According to Madison (2002), theories of ageing can be grouped into three; namely, sociological, psychological and biological, theories:
- **Sociological**: e.g., disengagement theory, activity/development task theory, continuity theory, age stratification theory, person-environment fit theory.
- **Psychological:** e.g., Maslow's Hierarchy of human needs, Jung's theory of individuation, Erikson's eight stages of life, Peck's expansion of Erikson's theory and selective optimism with compensation

Theories of ageing

- **Biological**: focus on answering basic questions regarding the physiological processes that occur in all living organisms as they chronologically age.
- Madison (2002) categorises biological theories into two:
  - ***Stochastic****:* Explain aging as events that occur randomly and accumulate over time. E.g., error theory, free radical theory, cross-linkage theory and wear and tear theory
  - ***Non-stochastic****:* View aging as certain predetermined, timed phenomena. E.g., programmed theory and immunity theory
- Other researchers such as Cavanaugh and Blanchard-fields (2006) group biological theories into three; namely, rate-of-living theories, cellular theories and programmed-cell-death theories
- In this session, only the biological theories will be emphasized and Cavanaugh and Blanchard-fields' (2006) categorization will be used. However, other theories will be highlighted briefly

Topic Two

RATE-OF-LIVING THEORIES

Rate-of-living theories

- **Originally proposed in the 1920s**
- **Rate-of-living** is an assumption that every organism has a set metabolic potential.
- Higher metabolic rate is associated with shorter lifespan and *vice versa*
- These theories suggest that people have limited energy to expend in a lifetime, hence, the more energy being used (which amounts to higher metabolism), the quicker one ages and the shorter the lifespan and *vice versa*

Rate-of-living theories

- Focus on the overall contribution of systems in the body:
  - Metabolic rates: we can only burn so much energy before too much damage. The higher the metabolism rate, the shorter the lifespan
    - E.g., bigger animals live longer than smaller animals due to the rate of metabolism between the two
  - Reduced calorie consumption is associated with longer lifespan in animals and humans
    - Evidence from experiments with rodents and rhesus monkeys suggests that reducing caloric intake lowers the risk of premature death, slows down a wide range of normative age-related changes, and in some cases results in longer life spans than do normal diets (Hayflick, 1996).
    - In humans, the Okinawans, who consume only 60% of the calories in a normal Japanese diet, have 40 times as many centenarians (people who are at least 100 years old) per capita as there are in the rest of Japan. Moreover, the Okinawan incidence of cardiovascular disease, diabetes, and cancer is half that in the rest of Japan (Monczunski, 1991).
  - Adaptation to stress is known to decrease as we age (Finch & Seeman, 1999).
    - These changes are associated with several diseases, e.g., Atherosclerosis, hypertension, diabetes, osteoporosis, and cognitive deficits

Rate-of-living theories

- Clever experiments over the last century have cast some doubt on this theory.
- Experiments where animals had their metabolic rate artificially heightened did not die sooner. This means that, while we can say that in general larger animals live longer and the larger animals have a slower metabolism, this does not always directly translate into a longer or shorter lifespan.

Topic Three

CELLULAR THEORIES

Cellular theories: Hayflick limit

- Cellular theories focus on ageing at the cellular level as opposed to systems.
- One cellular theory focuses on the number of times cell can divide which limits the life span of complex organism
- Cells are limited in the number of divisions they can undergo as they age (called the Hayflick limit).
  - Number of possible division decline with age E.g., human fetal tissues are capable of 40 to 60 divisions; human adult only about 20.
  - Evidence suggests that the tips of the chromosomes, called **telomeres,** play a major role in determining the limit of cell divisions
  - An enzyme called *telomerase* is needed in DNA replication to fully replicate the telomeres.
  - But telomerase normally is not present in cells, so with each replication the telomeres become shorter.
  - Eventually, the chromosomes become unstable and cannot replicate because the telomeres become too short (Saretzki & Zglinicki, 2002)
  - Telomeres and Aging _ Isagenix.mp4

Cellular theories: Cross-linkage

- Another cellular theory is based on a process called cross-linking.
- Cross-linking occurs when *certain proteins in human cells interact randomly and produce molecules that are linked in such a way as to make the body stiffer* (Cavanaugh, 1999c).
  - The proteins involved are called **collagen**
  - The more cross-links there are, the stiffer the tissue
- Cross-linking of proteins increase with age making the body stiffer.
  - Cross-linking may explain why muscles such as heart and arteries become stiffer with age.
  - Cross-linking impedes metabolic processes or cause faulty molecules to form resulting in aging
- However, few scientific data demonstrate that cross-linking impedes metabolic processes or causes the formation of faulty molecules that would constitute a fundamental cause of aging (Hayflick, 1998). Thus, even though crosslinking occurs, it probably is not an adequate explanation of aging.

Cellular theories: Free radicals

- A third type of cellular theory proposes that ageing is caused by unstable molecules called **free radicals,** which are highly reactive chemicals produced randomly in normal metabolism (Cristofalo et al., 1999).
- When free radicals interact with nearby molecules, problems may result.
  - For example, free radicals may cause cell damage, which in turn impairs the functioning of the organ, or may block the effects of important molecules.
- Evidence of the link between free radicals and ageing comes from research on antioxidants which prevent oxygen from combining with susceptible molecules to form free radicals
  - Antioxidants (Vitamins A, C and E, and coenzyme Q) are frequently used to counteract their effects.
  - A growing body of evidence shows that ingesting antioxidants postpones the appearance of age-related diseases such as cancer, cardiovascular disease, and immune system dysfunction (Hayflick, 1996)
  - But there is no evidence that taking antioxidants actually increases the life span (Cristofalo et al., 1999).
  - 'Free Radicals' or 'Oxidative Stress' will age our body's cells.mp4

Cellular theories: Wear and tear theory

- Proposed first in 1882
- Cells simply wear out over time because of continued use--rather like a machine
- Would seem to be refuted by the fact that exercise in OA's actually makes them more functional, not less

Cellular theories: Immunity theory

- **Immunosenescence**: Age-related functional diminution of the immune system
- Lower rate of T-lymphocyte ("killer cells") proliferation in response to a stimulus & therefore a decrease in the body's defense against foreign pathogens
- Change include a decrease in humoral immune response, often predisposing older adults to:
  - 1)decreased resistance to a tumor cell challenge and the development of cancer
  - 2) decreased ability to initiate the immune process and mobilize defenses in aggressively attaching pathogens
  - 3) increased susceptibility to auto-immune diseases

Cellular theories: Error theory

- Originally proposed in 1963
- **Damage/error theories** focus on how cells accumulate damage to their structure or errors to their DNA or mitochondria, which results in cell death.
- Basis:
  - 1)errors can occur in the transcription in any step of the protein synthesis of DNA
  - 2) error causes the reproduction of an enzyme or protein that is not an exact copy
  - 3) As transcription errors to occur, the end product would not even resemble the original cell, thereby compromising its functional ability
- More recently the theory has not been supported by research
  - not all aged cells contain altered or misspecified proteins
  - nor is aging automatically or necessarily accelerated if misspecified proteins or enzymes are introduced into a cell

Topic Four

PROGRAMMED-CELL-DEATH THEORIES

Programmed-cell-death theories

- Theories focusing on genetic programming of human DNA.
  - Is aging programmed into the genetic code?
  - Cells appear to receive signals to self-destruct.
- Argue that cells natural maturation follows a timetable with the death of the cell imminent.
- Programmed cell death appears to be a function of physiological processes, the innate ability of cells to self-destruct, and the ability of dying cells to trigger key processes in other cells.
  - Currently no information on how self-destruct programme is activated or how it works
- Genetic pathologies may be caused by genetic programming
- Evidence show that osteoarthritis, changes in the brain cells, Alzheimer's disease and Parkinson's disease have key genetic underpinnings

Programmed-cell-death theories

- The evidence supporting this theory is that there is not a great deal of variation in lifespan within species.
  - E.g., Elephants die at around 70 years old, spider, monkeys die at around 25 years old and humans die around the age of 80, on average.
  - Some changes can be made based on nutrition, medical care and other demographic factors, but overall lifespan within species is fairly constant

Sample Questions

- _____ theories stipulate that people have limited energy in lifetime and age-related changes come about when people use up their limited energy.
- One of the theories on aging propose that aging is caused by unstable molecules called _____ that produced in metabolism.

References