NRO D67 H3S PSYCHOBIOLOGY OF AGING AND SELECTED DISEASE STATES

SPRING, 2006 ROOM AA204, THURS.5-7 P.M.

INSTRUCTOR: PROFESSOR GWEN O. IVY

OFFICE: S-569, PHONE: 287-7438

EMAIL: ivv@utsc.utoronto.ca

OFFICE HOURS: THURSDAY AFTER CLASS UNTIL?, OR BY APPOINTMENT

COURSE DESCRIPTION:

Aging is a complex biological phenomenon which is, at present, a universal and inevitable fact of life. The goal of this course is to characterize the morphological, biochemical and physiological changes in aging neural tissue, as well as to delineate various behavioral and cognitive deficits that occur with age. We will explore the association between cognitive deterioration and age related neuropathologies. Further, we will define the relationship of numerous and diverse phenomena which occur during normal aging to those which occur in a number of related disease states, such as Alzheimer disease, Down syndrome, Parkinson disease, Neuronal Ceroid Lipofuscinosis and Progeria, with the aim of achieving a better understanding of factors which may cause aging at the cellular as well as the organismic level. Finally, we will evaluate the validity of several current unifying hypotheses of aging and will elucidate current and future prospects for research on the underlying mechanisms of aging using animal models.

ORGANIZATION:

The course will meet weekly for two hours and will consist of lectures by the instructor, as well as oral presentations by members of the class. Discussion will follow each presentation. Each person will select a topic from a list compiled by the instructor and will write a 20 page term paper (typed, double spaced, submitted in duplicate) and present a 15 to 20 minute talk to the class on that topic with a typed summary to be distributed to the class. Students should present talks in Powerpoint format if possible, and should provide handouts of the presentation talk for class and prof, as well as a copy of the Powerpoint (or other) presentation for the prof. Approximately 10 minutes of questions and discussion will follow. The student will be required to place one representative research or review article on his or her topic on reserve in the library (for the class) and to provide one copy of same for the instructor at least one week prior to the oral presentation. Note: Copies may be made for individual class members. There is no textbook. (Literally, NO comprehensive text on Aging, which includes up-to-date material on the variety of theories, experimental approaches, related disease states and numerous and diverse cellular and organismic phenomena that we cover in this course, exists!)

EVALUATION:

Term Paper (2 copies due April 7 th):	25%
Oral Presentation, includes summary handout, article placed on reserve and copy of PPT	15%
Midterm (essay, short answer): 2 hrs.	15% Week of Feb 20, TBA by Registrar
Midterm (essay, take home portion) (Critique of Protease Inhibitor Model)	10% Due in class March 9, 2006
Final Exam (essay, short answer): 3 hrs. During Final Exam Period	20%
Final Exam (essay, take home portion) (What is Aging?)	10% Due at time of Final Exam
Class Participation: (Includes questions, comments and presence in class)	5%

SCHEDULE OF TOPICS TO BE COVERED

<u>DATE</u>	GENERAL TOPIC	SPECIFIC TOPIC
Jan. 12	Introduction	Course overview/Selection of paper topics
Jan. 19	Morphological and some biochemical and physiological	 A. Age pigments (Lipofuscin & other pigments: ceroids) B. Dolichols C. Gross brain changes D. Vascular System Changes E. Dendritic changes F. Synaptic changes
Jan. 26	changes in neural tissue with age, and in certain diseases with some similarities to aging: Lectures	G. Changes in glial cells H. Cell loss I. Amyloid and senile plaques J. NFT, PHF K. Progeria and NCL
Feb. 2	bv G.O. Ivv	L. The Proteuse Inhibitor Model of Aging

Feb. 9 Class Discussion

Aging in Canada: Excerpts from "Alive" Magazine

Seniors hooked on Drugs (P. 52)

Eye Disease (P. 58) Retirement (P. 62) Incontinence (P. 68)

Healthy Longevity (P. 106) Foods for Longevity (P. 116)

Feb. 13 Class Discussion

Selected Articles from Scientific American:

The Oldest Old; Spare Parts for Vital Organs;

Aging: Promised Land or Purgatory?

How Research on Aging Progresses:

Interviews with Denham Harman and Ken Kitani

Feb. 20-24

READING WEEK !!!! NO CLASS !!!

Student Oral presentations:

- Mar. 2 Sensory motor and anatomical changes with age and disease
 - 1. Sensory deficits, aging of sense organs
 - 2. AD & Down's syndrome: morphological changes in brain, descriptive
 - 3. Pathogenesis of Amyloid Plagues and NFT
 - 4. Risk factors and Safety factors in AD etiology, familial vs. sporadic
- March 2-10 Midterm, 2 hrs, TBA by Registrar
- Mar 9 Changes in anatomy and neurotransmitter systems in aging, AD and PD; immune system
 - 5. Dopaminergic Systems in Aging and PD; PD genesis
 - 6. Cholinergic systems in aging and AD
 - 7. Immune system changes in aging how to prevent
- Mar 12 March 12= Last Day to Drop
- Mar. 16 Theories on mechanisms underlying the aging process
 - 8. Free radical theory of aging (FRTA)
 - 9. Mitochondria and aging
 - 10. Genetic theories of aging: cell culture
- Mar. 23 Theories (cont'd.)
 - 11. Changes in DNA: damage and repair
 - 12. Telomere shortening and telomerase intervention for life extension
 - 13. Caloric Restriction and its mechanisms for prolonging life

Mar. 30 Other factors in Aging:

- 14. Dietary antioxidants and Longevity
- 15. Adrenocortical axis in aging: stress and glucocorticoids
- 16. Changes in Sex hormones with age

Apr 6 Other factors of Aging (con'd)

- 17. Exercise and mitochondria in skeletal muscle (athletes)
- 18. Biological Clocks: Can we slow them?
- 19. Worms: What they tell us about living longer and better
- 20. Flies: What they tell us about living longer and better
- 21.

Summary, Conclusions - Ivy and Class

NROD67H3S

GWEN O. IVY

MIDTERM EXAM

Take Home Portion
10% of Grade
Due at time of Midterm Exam

Critique the Protease Inhibitor Model of Aging.

Delineate its strengths and weaknesses. What, if anything, can it tell us about primary vs. secondary effects of aging. Are defective thiol proteinases likely to cause aging? Why or why not? What is, or would be a better model of aging? (BE REASLISTIC!) Is there any better way to investigate the underlying cause(s) of aging?

Seven typed pages, double spaced is maximum length.

FINAL EXAM

Take Home Portion 10% of Grade Due at time of Final Exam

What causes AGING?

Utilizing your knowledge of the various manifestations of aging along with what you know about the various mechanisms which may cause these manifestations, create your own hierarchical scheme of aging. From lipofuscin to physiological and behavioural changes, how does it all happen? What does the cascade of 1° to 2° to 3° (etc.) changes look like? How do you know? Back up your ideas with data!

Seven typed pages, double spaced is maximum length. A grade of "A" probably cannot be achieved in fewer than 5 pages.