Syllabus for Biology 372: Evolutionary Ecology of Disease
Fall Semester 2015

Professor:
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Office hours: Tuesdays 4:00-5:00, Thursdays 1:00-2:00; other times by appointment.

Goal of course: to provide an overview of health and disease from evolutionary and ecological perspectives, with a particular emphasis on diseases of humans.

Target audience: The course is designed for students who want to understand how biological principles, particularly the concept of evolution, can be applied to issues of health and disease. I am trying to reach students who are in one or more the following situations: (i) students who are interested in pursuing a career in the health or agricultural sciences and in obtaining a broader sense of the ecological and evolutionary framework within which disease processes arise, (ii) students who have taken some general science courses and are interested in understanding how biological principles are relevant to the shaping of public policy (e.g., students who may eventually pursue a career in law or government), (iii) students who are interested in understanding the interplay between disease and history, and (iv) students who would like to understand how basic principles of evolution may be relevant to daily life regardless of their eventual profession.

Hierarchical structure: The course delves into fairly complicated aspects of biology (e.g., antigen presentation and leukocyte function) but will develop each concept from basic principles with a restricted scope, so that students receive what they need to understand the arguments that are being made. I assume only that students have a working knowledge of the basic concepts that were covered in introductory biology (the meaning and significance of terms such as genes and alleles, the differences between viruses, bacteria, protozoa, etc.); course material will, however, be specialized and detailed. Even those students who have completed substantial coursework in biology will therefore be exposed to a great amount of information that they have not encountered in other courses.

Student Learning Objectives

- *Integrative thinking:* Large amounts of information will be presented. Lectures and exam questions are designed to develop and test not only a mastery of this information but the ability to integrate this information into broadly relevant conceptual frameworks.

- *Critical thinking*: Information will be presented alternative hypothesis which students will need to evaluate by considering both supportive and contradictory evidence.

Prerequisites: Biology 240 & 242, or equivalent, or permission.

Readings

No textbook is required for the course. A few mandatory readings will be assigned during the

semester. They will be posted on blackboard prior to the lectures to which they correspond.

Optional background readings are listed in the lecture schedule below. These readings are from the following books:
-Ewald, P.W. 2002. *Plague Time*. Anchor Press: New York. (paperback) [ISBN:0684869004]
-Nesse, R.M. & Williams, G.C. 1996. *Why We Get Sick*. Vintage Books/Random House: New York. [ISBN: 0679746749 (paperback)
New and used copies are available at amazon.com, barnesandnoble.com, etc. Used copies are available at abebooks.com. The hardback and softcover editions of Plague Time have different subtitles: "How stealth infections cause cancers, heart disease and other deadly ailments" and "The new germ theory of disease" respectively.

Grading:
Exams

Two exams, each worth 100 points each (total points=200): Sept 29th & Nov 5 (second exam is on material since first exam)

Comprehensive final exam worth 200 points: Wed., Dec. 9, 11:30AM - 2:00PM (in our normal classroom)

*Final letter grades*Pluses and minuses will be assigned for each letter grade according to the standard breakdown of points:

A+	**A**	**A-**	**B+**	**B**	**B-**	**C+**	**C**	**C-**	**D**	**F**
96.7-100%	93.3-96.7%	90.0-93.3%	86.7-90.0%	83.3-86.7%	80.0-83.3%	77.6-80.0%	73.3-77.6%	70.0-73.3%	60.0-70.0%	0.0-60.0%

Last day to withdraw: October 26th (set by College of Arts and Sciences)

Lecture notes, visuals, and all other documents will be posted on Blackboard in the Course Content section.

Lecture Schedule (About three lectures will be given on each topic. *Optional* background readings from *Why We Get Sick* (WS) and Plague Time (PT) are given in parentheses.

The evolutionary framework for understanding disease
-Proximate vs ultimate questions
-Natural selection: levels, power, and outcomes
-The ecological stage of the evolutionary play
-What is disease?
-Epidemiology: the ecological study of disease
-Evolutionary epidemiology: the study of disease processes from ecological and evolutionary perspectives
-Darwinian medicine: the study of the causes, consequences and medical implications of human and veterinary diseases from an evolutionary perspective.

Causes of disease (WS Chapter 1-2; PT Introduction, pp.1-6)
-Agents of disease: infectious, genetic, and noninfectious environmental causes–relative contributions and joint actions
-Acute vs chronic disease–the artificial dichotomization of a continuum

Disease manifestations: signs and symptoms (WS Chapters 3, 5 & 6)
-Evolutionary categories of manifestations: defenses, manipulations, & side effects
-Implications of categories for symptomatic treatment
    --Noninfectious diseases: evolutionary implications are relatively straight forward because manipulation category is almost always irrelevant and long evolutionary histories imply of damaging side effects.
    --Infectious diseases: evolutionary implications are complex because defenses and manipulations are both relevant.
-Examples:
    --Physical trauma, (e.g., sprains)
    --Manifestations of infectious diseases (e.g., fever, diarrhea, anorexia, vomiting)

The immune system (WS, pp. 49-52, 61-62, Chapter 11; PT Chapter 4)
-the immune system as a police force
-the immune system as a system for storing information and making decisions
-evasion of the immune system
-arms races with the immune system
-the immune system gone wrong?--autoimmunity, inflammation, allergy and the hygiene hypothesis

Genetic diseases (WS, Chapter 7)
-the big ones (e.g., sickle cell anemia, cystic fibrosis) all appear to be crude defenses against infection.
-the moderately big ones (thalassemias, G6PD deficiency, hemochromatosis) are probably also defenses against infection
-the nasty but very rare ones (e.g., Huntington's Disease) may be maintainable by mutation rate and may therefore be considered purely genetic diseases.

The evolution of virulence
-The parasitism/mutualism continuum **(WS, pp. 57-61; PT, Chapter 1)**
-The traditional view of medicine: natural selection favors evolution of parasitism toward benignity
-The modern view from evolutionary biology: natural selection can favor evolution toward any point on the continuum from virtually perfect mutualism from lethal parasitism
-Ecological factors favoring evolution of high virulence
    --vectorborne transmission
    --waterborne transmission of diarrheal diseases
    --attendant borne transmission (e.g., in hospitals)
    --sit-and-wait transmission of durable pathogens

--agricultural vectors
--complex life histories involving predation
--parasitoid life cycles

-Evolutionary virulence management of acute infectious disease **(PT pp. 195-206, 213-219)**
--Altering the environment to make host health a prerequisite for transmission: vector-proofing houses, cleaning up water supplies, improving hygienic practices in hospitals, staying home when sick and working on the Internet.
--Tipping the balance in favor of benign pathogens using virulence-antigen vaccines
--Economic, social and political implications

The evolution of antibiotic resistance (WS, pp. 52-57; PT Chapter 5 & pp. 219-226)

-The biological problem: antibiotic resistance typically evolves within a time period of a few weeks to a few decades.
-The socioeconomic constraints: antibiotic usage is good for the patient and the physician over the short term but bad for the society over the long term (Tragedy of the Commons).
-Long term strategies recognize this conflict of interest and work within its constraints
--reduce unnecessary usage (e.g., by coupling narrow spectrum antibiotics with rapid diagnostics)
--reduce usage by favoring evolution of reduced virulence
--reduce selection for resistance by restricting some antibiotics for dangerous dead-end infections

The major bridge between acute and chronic infectious diseases: STDs (PT Chapters 2, 3, 7 & 9, & pp 206-11)

-The key attribute for sexually transmitted pathogens: long persistence within a host
-Mechanisms of persistence
-Natural selection acting on persistent pathogens: high potential for sexual transmission should favor evolution toward high virulence
-Test cases: HIV, HPV, HTLV, genital herpes simplex, *Chlamydia* and other bacterial pathogens

Emerging diseases and biosecurity (PT Chapter 6 & 12)

-Threat posed by emergence of different infectious diseases
--diseases that have recently entered human populations from animal populations (i.e., zoonotic diseases)
--diseases that are expanding from one human population into another
--diseases that are newly recognized as being caused by infection
-Threat of pathogens as biological weapons