Focus of this course:
to understand the interactions of insects, fire and disease on forest community structure
and dynamics, and to understand the effects of human actions on these processes.

Main emphasis will be on California forest systems

What is a community?: assemblage of species and their interactions

Parameters that define a community
Composition (species present and relative abundance); "stocking level"
relationships among species
dynamics

Climax view ala Clements (1905-1930) - Succession and Climax; communities are real
entities.

Two main processes explain community dynamics
1) competition, especially for light, water, & nutrients
2) periodic disturbance

The central ideas of climax theory are:
1) Given enough time without disturbance all plant communities reach a climax,
and in a forest community this climax would be dominated by shade tolerant
plants.

2) For a given climate and soil type there is a definable climax community
(="potential vegetation")

Climatic effects on community structure:
Whittaker diagram - effects of mean temp and ppt.

Effect of altitude: Figure from Arno 1973,(attached)
Fig. 17-1 from Terrestrial Vegetation of California

Relationship of Temp and ppt. to altitude in Sierras
Fig. 2-12 from Terrestrial Vegetation of California

In the mixed conifer forests of the Sierras the level of shade tolerance looks something
like this:

```
 increasing shade tolerance
    giant sequoia    true firs
  most pines  Douglas-fir  incense cedars
  most oaks  sugar pine  hemlock
```

Problems with climax view - long lived "pioneers", inevitable disturbance
'Modern view' - (with roots in the work of A.G. Tansley, and H. A. Gleason ca. 1917-1930s)

1) Communities are collections of individual species. These collections are unique in time and space and defined by competitive interactions among the species.

2) Equilibrium is seldom if ever reached, because the frequency of disturbance is very high.

3) Plant diversity results primarily from the patchy distribution of disturbance in time and space (Pickett 1980) - see figures 2.9 and 2.10 in text.

Problems with modern view

Although the importance of disturbance is realized the agents involved in it are often misunderstood, ignored, and underestimated. Examples:

Insects and pathogens are seldom mentioned, even though "gaps" in the forest are frequently caused by them.

The importance of density dependent selection is appreciated but the roles of disease and to a lesser extent insects are grossly understudied.

Trees do not die of old age - they are actively killed either by abiotic or biotic agents or a combination of both. Predisposition is important.

Although fire has been recognized as a significant disturbance agent it is often taught as an all or nothing phenomena - the differential effects of various fire regimes and their specific interactions with plants, plant diseases, and insects are not commonly taught.

The role of human actions are usually ignored or viewed as unnatural intrusions into a natural system, but today these are some of the dominant influences.

Disturbance regimes (table 2.2 in Edmond et al 2000): Type, Frequency, Magnitude, Predictability, extent, timing, synergy, and severity.

Compound disturbances (Paine et al 1998) - shift to a different state?

Disease Definition (from Fields Cobb):
"abnormal and harmful physiology resulting from a sustained interaction between a plant and its biotic or abiotic environment"

The disease "triangle":

Host - genetics (most potential hosts are resistant), Age & organ, physiological status (wounds).

Environment - Temperature, moisture, other organisms

Pathogen - genetics, other hosts, physiological status, inoculum (amount, proximity)
How do pathogens enter?
- Wounds - fire-scars, branch stubs, damage from insects or other pathogens
- Direct penetration - leaves or succulent tissue (stomates) or thin bark, root contact or graphs, branch contact,
- Vectored by insects - on surface, mycetangia

Plant resistance to pathogens
- Horizontal vs vertical resistance (tolerance vs “all or nothing”) – graph

Horizontal defenses (= tolerance)
- Slow rusting, tolerance of various sorts
- Heartwood - extractives
- Tyloses, resin
- Tannin, other Phenolics, various toxic compounds

Systemic Acquired resistance SAR
- Attack at some part of the plant increases resistance at other unattached parts via salicylic acid signal.

Similar independent pathway is based on jasmonic acid and ethylene signals (ISR - induced systemic resistance)

Vertical - Gene-for-Gene Model: for each host resistance gene there is an avirulence gene in the pathogen
- Host reaction is usually expressed as a hypersensitive response
- Resistance is usually dominant
- Virulence is usually recessive (see tables)
- A simple model - resistance gene is a receptor that turns on defense response when it binds the avirulence gene A product

Single factor model

<table>
<thead>
<tr>
<th>Pathogen genotypes</th>
<th>Host genotypes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RR</td>
</tr>
<tr>
<td>AA</td>
<td>-</td>
</tr>
<tr>
<td>Aa</td>
<td>-</td>
</tr>
<tr>
<td>aa</td>
<td>+</td>
</tr>
</tbody>
</table>
ESPM 134  Introduction  Spring 2009

Two-factor model

<table>
<thead>
<tr>
<th>pathogen genotypes</th>
<th>R1- R2-</th>
<th>R1- r2r2</th>
<th>r1r1 R2-</th>
<th>r1r1 r2r2</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1- A2-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>A1- a2a2</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>a1a1 A2-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>a1a1 a2a2</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

+ = disease, - = resistance

Importance for epidemics

**R genes** - several classes known, class with Lucine Rich Repeats and Nucleotide binding sequences are abundant

**Avirulence genes** – no common motifs, but often appear to be involved in altering plant disease resistance. - Misnamed because they may actually increase virulence in plant lacking vertical resistance.

Effects of Diseases and Insects in forests

Selection of species best suited for a site (maintenance of geographic ranges - e.g. Monterey pine, Monterey cypress)

Maintenance of species diversity (forest composition - selection against monocultures - e.g. blackstain root disease)

Selects for genetic diversity within species (via density dependent selection)

Maintenance of forest structure (stocking density as well as composition: root diseases, mistletoe)

Driving force in succession

Who are the Pathogens:

**abiotic**: air pollution - O3 and SO2, salt damage (for others see chapter 9, table 9.1)

SO₂ - point source: mining and heavy industry; acute effects (e.g. Sudbury Ontario) vs. chronic effects (acid rain).

$$O_2 + NO_3 \rightleftharpoons O_3 + NO$$

$$O_3 + NO + hydrocarbons \rightleftharpoons PAN \text{ (peroxyacetyl nitrate)}$$
Sources - many: automobiles, farming, etc.  
O3 and PAN cause acute effects (e.g. chlorosis)  

N deposition (10-20 kg/H) may cause chronic effects: acidification, species shifts in plants and ectomycorrhizal fungi, forest decline in Europe?  

Tree species and genotypes are differentially susceptible to many airborne pollutants.  

biotic:  
higher plants (mistletoes, dodder)  
Bacteria & mycoplasmas - crown gall, leaf spots, yellows & scorches (ash yellows in East)  
Viruses - blackline on walnuts  
Fungi - virtually all important diseases of forest trees  

Readings:  


Other references for your interest only  
(not on reserve)  

Classic Climax  

non-equilibrium models of diversity:  

For general definitions and measures of communites:  

For California Plant Communities  
Barbour, M. G. and Major, J. (eds) 1977. Terrestrial Vegetation of California, California native plant society. - Chapter 17 is especially useful for the Sierra field trips., and chapters 9 and 21 for Pt. Reyes.  

Ornduff, R., Faber, P.M. and Keller-Wolf, T. 2003 *Introduction to California Plant Life*, University of California Press. 341 p. **At $16.95 this book is a must own for anyone with a long-term interest in plant communities in California.**

For molecular genetics of R genes try the following two reviews, but the detail in them is beyond my expectations for this course.:
