

Genetics – for week 7, read gene cloning (285-297), Reconstructing the Genome chapter; parts of ch. 11 assigned week 4 but not yet discussed

Week 6

1. Discussion of the normal control of cell division – pg. 618-627 plus fig. 17.4-17.5 (regulation)
Terms: cell cycle phases G1, S, G2, M – which do you expect to be most variable in length?; **cyclins** and **cyclin dependent protein kinases**; advantages of budding yeast (*Saccharomyces – brewer's yeast*) in identifying genes controlling cell division – even genes of humans! [see fig. 18.3-18.10 and pg. 745]; key checkpoints and how they are studied (pg. 624-626); apoptosis

2. basic immunogenetics: Trevor – see Fig. 21.21-23; pg. 445-447; read: 728-734 in Hartwell

3. GENETICS AND CANCER –

Discuss fig. 18.15 to 18.24 and Watson Cancer chapter

4. Reassemble; discuss questions raised in small groups

My supplement to the book section :The word "Cancer" evokes images of alien invasion, betrayal by our own body, cells totally out of control, fear of the unknown. Until very recently, our only ways to fight these cells gone astray involved **massive frontal attack** -- surgery as long as it was still (largely) in one place and accessible, radiation and chemicals that would damage or **destroy any rapidly-growing cells**, including, unfortunately, the linings of our body and the natural defenses provided by the **immune system**. We knew no way to treat our cancer cells except as **incurable criminals**, enemies capable of infiltrating and destroying every aspect of our system, evil traitors. Especially frightening was the realization that the **initial events in tumor formation had generally happened 25 years earlier, and this potential betrayal is going on in all of us all the time**, with no way to know when or where our immune systems will fail in their work to keep such delinquent cells in check. Nixon's "war on cancer" led to little change in the mortality rates for most cancers, despite aggressive treatment. Our understanding of the cell changes involved in the development of cancer (and the normal regulation of growth) is increasing enormously using the techniques of molecular biology. This increased understanding is now affecting both efforts at cancer prevention and the development of much more tightly targeted methods of cancer detection and treatment.

As a society, we are learning to reframe our understanding of cancer. Cancer does not involve cells that are inherently alien and bad, but **cells that are working as intensely as they can to respond appropriately to the signals they are receiving** – the **problems** relate to specific **errors** in the nature and receipt of those signals.

The **challenge** is to look for specific ways to **reprogram** early on in the development of a specific tumor so that it will not become seriously destructive and we will not have to use methods that are terribly destructive to the whole system to eliminate it. Here, it parallels social concerns.

Self versus non-self – and the relationship of being able to develop this sense effectively to the preservation of the community

Effective communication among members of a community – involving appropriate **sending and receiving of signals** and the **ability to respond** to them in appropriate ways

Early research on cancer focussed on the role of viruses – and though they generally are not a primary cause, our understanding of the cancer process, and of cell division in general, has largely been gained through work with oncogenic viruses. The major causes of cancer are in fact things where each of us has substantial control, such as smoking, dietary fats and lack of antioxidants, alcohol and hormone use. These act by causing **mutations** (i. e., free radicals and radiation), **stimulating cell division directly** (hormones), or both (**tobacco**), with broader environmental problems playing a smaller role.

MODEL:

CARCINOGENESIS IS A MULTISTEP PROCESS.

IT INVOLVES A SERIES OF **DEFINABLE GENETIC CHANGES** THAT ACCUMULATE IN THE