Skin:
Reading Chapter 22
SKIN
at risk because its the main interface with the outside world.
skin is formed of flat layers of cells and contains much keratin, which makes it tough;
there are seven layers, growth is continuous.
the outer surface is very dry, which makes direct entry difficult for pathogens

Pathogens
capable of gaining entrance through:
– injuries
– sweat glands, -sweat, which moistens the skin
– sebaceous glands-sebum, also moistens the skin
– hair follicles

Normal biota 3
1. Diphtheroids, gram+ rods
Propioni acnes is an organism that can be found on people without disease [acne]
lives on the skin
anaerobic, oil-coated - in the sebum
breaks down oil into fatty acids which protect against pathogens; create a buffer zone
2. Staphylococci
ex: Staphylococci Epidermidis is a gram-positive aerobic organism, found in clusters
does not compete for living space with Propioni acnes
can inhibit growth of Propioni acnes ; as well as inhibiting pathogens from growing in its place.
3. Yeasts
ex: Malassezia is an example of a yeast
need oils for growth
capable of making substances which can protect against other

Pathogens
characteristics:
Bacterial Infections give spotty infections

vs

Fungal Infections which localize or
go across or under the layers of skin
spread uniformly in a coat
- often enter at points other than the skin, but manifest in the skin

Virulent Staphylococci aureus

Staph = cluster ; aureus = golden
-Infests hair follicles to cause :
– furuncles (boils)-which are an extension of folliculitis; [inflammation of the hair follicle]
– carbuncles - which result from the coalescence of furuncles and extend to the deeper subcutaneous tissue; dangerous because the infection can spread through the blood to the brain, bone, or heart.

Virulence Factors of Staph aureus

coaagulase positive and often resists penicillin and other antibiotics
bonds to the hair follicle then enters the skin matrix and goes deep to cause an inflammatory response
a polysaccharide capsule helps inhibit phagocytosis
teichoic acid binds fibronectin - located extracellularly
fibronectin is known as "glue" between the cells; secreted by the host cell
Protein A helps Staph A. survive attack

bonds on Fc portion of an antibody, this inhibits the induction of phagocytosis and C’ attack of the antibodies

Leukocydin is a poison it "drills" holes in granulocytes
- Exfoliative toxin breaks the tight connection between skin cells that holds the skin together in layers, then the skin falls off in loose bits which results in Scalded Skin Syndrome
- if the toxin is -only- localized then skin blisters occur , which results in impetigo (S. Pyogenes can do this too)
Toxic Shock Syndrome Toxin-1 is an exotoxin secreted during growth. Causes a general turn on of all T cells by binding them--and thus inappropriate inflammation! Without a specific target.
Enzymes of Staph.
- Hyaluronidase breaks down connective tissue; hydrolyzes hyaluronic acid present in the acellular matrix of connective tissue
- Coagulase may cause the formation of a clot --that is, a fibrin layer around the abscess which localizes the infection and protects the organism from phagocytosis. Coagulase positive organisms tend to form focal infections.
  – coagulase-negative spreads to the organs; transmitted by the inoculation of organisms onto a damaged or artificial heart valve (Endocarditis-the swelling of internal heart tissue). These infections are seen at places where the immune system isn’t up to par.
  – coagulase-positive Staph is present as normal flora in the stomach and vagina; local infections produce toxins that spread thru system. Toxic shock syndrome.

Streptococci pyogenes
causes similar diseases of the skin, such as impetigo
Streptococcus pyogenes is known as the "pus maker"; infects the throat and causes respiratory diseases.
glomerulonephritis after Strep

Borrelia burgdorferi
a spirochete
also causes skin infections
discovered in Lyme, Connecticut, cause Lyme Disease

see a map of its distribution currently
enters the skin through bite of hard shell ticks from deer, white foot mice, thus, a zoonoses
- at risk: hunters and hikers
After infection:
sometimes a local red patch at the bite site =
"erythema migrans" which means moving redness; this can be long or short term
- other early signs and symptoms exist as well, such as malaise, severe fatigue, headache, fever, chills, musculoskeletal pains = myalgias, and lymphadenopathy.
later manifestations can be seen as neurological symptoms and cardiac dysfunction
Difficult to control:
East Coast...Connecticut- 80% of hard ticks that bite humans have the disease (mice ticks)
West Coast...California - pack rats have ticks and a similar disease but these ticks do not bite humans, so there are fewer cases of the disease

RICKETTSIA
discovered by Henry Rickett in 1904, he later died from typhus
intracellular obligates-tiny bacteria
cause of Rocky Mountain Spotted Fever.
transfer is from blood by living cell of ticks into humans.
growth is inside vessel walls, this causes blood vessel damage seen on skin and in organs below.

map

symptoms

FUNGAL SKIN INFECTIONS
– fungal infections are classified according to the tissues that are initially colonized.
1. Superficial Mycoses - found on the top layer of skin and hair Maleszia furfur is the organism responsible for the disease Pityriasis versicolor. (involves the skin)
Exophiala werneckii an organism which causes the disease
Tinea nigra (involves the skin)
Note: These infections are not painful because they do not occur near live tissue.
– Piedra - an infection in a hair shaft.
– Tinea - an infection in the skin; causes symptoms like dark to light or light to dark

2. Cutaneous Mycoses
involve diseases that are deeper in the skin, hair, and nails
painful, due to the occurrence near live tissue. This also triggers an immune response.
clinically termed ringworm or tinea, depending on the anatomical site involved:
capitus = head
pedis = feet
corporis = body
manus = hands
unguium = nails
exothrix = hair surface
endothrix = deep in the hair
favus = (a fungal inf. that hurts) both hair surface and deep.

Traditionally these fungi are termed dermatophytes [skin plants] although they are not
phylogenetically related to plants.
Often classed according to their source/origin:
geophilic - soil source
zoophilic - animals
anthropophilic - ONLY from other people

Skin and Viruses

Basement Membranes- in the skin these are very thick
nutrients that feed the skin cells above come from these basement membranes
– good site for parasites NO DEFENSES, because blood, PMNs are out of reach here
unless a break is present
viruses can not go through the basement membrane because a host cell is necessary but
they can cause a persistent infection.
Defenses
Ideal Circumstance-a permanent soldier above the basement membrane that could
distinguish between infected tissue [and not], and be able to kill the infected directly.
Actual Circumstance- an infected cell makes stress proteins which include heat shock
proteins {HSP}. HSP is turned on during stress. There are gamma-delta T-cells which use
gamma -delta T cell Receptors. These T-cells are seeded early in life to coverings of the
body. These T cells are called dendritic because they have long projections--a good
property to check out lots of cells. These T cells function as killer cells.
Viral Pathogens

Measles, standard- rubeola
the first site: Koplik’s spots will be present approx. 24-48 hrs before the rash
infection enters through the respiratory system
easily diagnosed once it reaches the level in the skin where it causes local bleeds
casted by rubeola
a negative single-stranded RNA and of the family of paramyxovirus morbillivirus.
– does not kill infected host cells but instead they bud off the surface taking protein,
lipids etc. which leaves the host cell sick.
A common problem with budding viruses it that the immune system can end up making
mistakes---see below
BEST: before any cell is infected a neutralizing antibody which will coat the virus prior
to infection and neutralize it. This can result from vaccination.
Antibody is especially ineffective after infection: the virus can pass directly from cell to
cell and escape antibody control due to its ability to cause cell fusion, resulting in giant
cells. F protein
Infection with measles virus through
CD46 = complement regulatory protein located on cell surface proteins. CD46 prevents
lysis of our own cells by complement.
– the measles virus uses CD46 for a means of attachment to cell surfaces which allows
the virus to infect. Fortunately, when there is active infection the amount of CD46
decreases, then C’ lysis of infected cells will occur.
– CD46 is only found in humans and monkeys, therefore = the only possible hosts for this
measles virus
- this virus is spread through respiratory droplets in the air. The virus once in the airway
infests the cells of the airway, and spreads to the lymph nodes where viral replication
occurs, then it can get into the blood circulation and symptoms are seen.
- Virulence
hemaglutinin = H protein, this provides binding to CD46
Fusion protein (F) causes cells to fuse together with the virus and also causes eukaryotic
cell to fuse with another cell within the body. Results in giant cell inflammation which
can cause Giant cell pneumonia
- for adaptive immunity to occur one must have T-helper cells, T-killer cells and
antibody.
If any one of these is absent the infection will not be cleared.
Sometimes the immune system gets out of control.
There can be mis-recognition caused by self proteins being on the budded viral particles.
budding allows self-recognition--there are lots of particles that have self protein that now
have access to macrophages and B cells--self-proteins that otherwise never end up as
peptides on class II molecules--but now they are.
The long term effects of measles can be extensive if the central nervous system is
effected.
SSPE = sub-acute sclerosing panencephalitis; a neurological disorder caused by a defective [non-replicating] measles virus which persists in the brain and acts slowly. Over time the patient will develop changes in personality, behavior, and memory. the measles virus has been associated with multiple sclerosis [MS], but has not been proven causitive for the disease.

Immunization is very important to
AVOID the long term effects, which are incurable,
prevent secondary bacterial infections, which can cause deafness, and
prevent death from measles itself
Worldwide, measles [rubeola] is a major killer in children.
if malnurished one-fifth of the infected children will die, if complicated by a lung bacterial infection then 3/5 will die.

GERMAN MEASLES
- caused by Rubelia, which is a major cause of birth defects in the first trimester
- vaccines during pregnancy could be a greater health risk than the virus in some instances.
- antibodies can interfere and block the virus during the following points in the pregnancy
  primary viremia, secondary viremia, and
to the placenta . NOTE: Antibody may be achieved by any means available.
Rubella
envelope virus, Positive ss RNA virus
replicates in the cytoplasm, then buds.
incubation period is 14-21 days,
spreads through respiratory tract, a rash will be observable in 3 days and swollen glands will occur.
Congenital Rubella Syndrome
- Cataracts and other ocular defects
- Heart defects
- Deafness
- Intrauterine growth retardation: failure to thrive, mortality within the first year
- Microencephaly: Mental Retardation

cases

WOUNDS Reading Chapter 24
Staphylococcus aureus (coagulase + Staph) is the most common cause of infections in surgical wounds.
The development and type of surgical wound infection can be predicted based on the duration of surgery, presence of foreign material, status of host defenses. A major problem is the resistance which is arising to antimicrobial medicines.
Strep pyogenes -flesh eating disease

Pseudomonas aeruginosa is a common cause of burn infections. They make the burns green because they make a green pigment. P. aeruginosa is usually resistant to most antibiotics and they also release a potent toxin. Pyocyanin—produce free radicals
polysach
endotoxin
elastase
phospholipase C
alkaline protease
toxins
exotoxin A
exoenzyme S
Both A and S ADP ribotransferases alter a G protein

Clostridial wound infections:
Tetanus is spastic paralysis caused by a toxin produced by Clostridium tetani—a motile anaerobic Gram-positive rod with a terminal spore. Colonization of wounds by C. tetani can result in tetanus. Immunization and care of wounds prevents this otherwise fatal disease. Tetanospasmin
– Binding is to a ganglioside on neurons
– release of neurotransmitters blocked by light chain
– GABA gamma amino butyric acid
– block is of inhibitory synapses does this by cleaving --synaptobrevins

Gas gangrene is caused by the anaerobe Clostridium perfringens. It causes necrosis (death) of muscle tissue.

Actinomyces—sulfur in drainage

cat scratch fever

garden pokes and scratches—rose gardener's disease
and lastly,

human bites causing infections