Superficial and Cutaneous Mycoses
Superficial Mycoses

- Limited to the outermost layer of the skin
- 4 Infections
  - 1. Pityriasis versicolor
  - 2. Tinea nigra
  - 3. Black piedra
  - 4. White piedra
Superficial

- Do not elicit immune response
- No discomfort
- Cosmetic problems
- Limited to stratum corneum
Pityriasis Versicolor

- Malassezia furfur (Pityrosporum orbiculare)
- Lipophilic yeast like organism
- Rich in sebaceous glands
- Media is supplemented with fatty acids
- Exist in budding yeast, occasionally hyphal
Pityriasis versicolor (An-an)
Pityriasis

- Lesions are found in torso, arms and abdomen
- Scale very easily → chalky appearance
- Rarely, papular or grow like folliculitis
Pityriasis

- Clinical Diagnosis: KOH- Spaghetti and meatballs
- Treatment: Azoles
Tinea Nigra

- *Exophiala werneckii*
- Produce melanin → black or brown color
- Grows as yeast → Older hyphae with mycelia and conidia
Tinea nigra

- Lesion - gray to black macular palms
- Diagnosis - Skin scrapings with alkali stain
- Cultures - Sabouraud’s media pigmented yeast and hyphae
Black Piedra

- Piedraia hortae - exist in teleomorph state
- Cultures – asexual state
  - older cultures → teleomorph (asci, which contain spindle shape ascosporas)
Black piedra

- Clinical feature: presence of hard nodules found along the infected hair shaft
- Nodules contain asci
White Piedra

- Trichosporon beigelii
- Grows in media without cyclohexamide
- Cultures are pasty and white→ developed deep radiating furrows and become yellow and creamy
White Piedra

- Microscopic examination septate hypae that develops into arthroconidia
- Hair - soft, pasty, cream colored growth
Treatment

• Skin
removal of the organism by:
1. Selenium sulfide
2. Thiosulfate
3. Salicylic acid
4. Hyposulfite
inhibition of ergosterol by:
1. Miconazole
Cutaneous mycoses

- Skin
- Hair
- Nails
- Evoke cellular immune response
- Dermatophytes
- Clinical manifestations → ringworm or tinea
Cutaneous mycoses

- Etiology
  - Microsporum
  - Trichophyton
  - Epidermophyton
Cutaneous mycoses

- Classifications:
  - Anatomic location
    - Tinea pedis
    - Tinea corporis
    - Tinea cruris
  - Ecologic location
    - Geophilic
    - Zoophilic
    - Anthrophilic
Cutaneous mycoses

- Keratophilic – use keratin as subject to live (parasites)
- Keratinases - invade only keratinized layers
2 basic types of dermatophytic infection:

1. The acute or inflammatory type of infection, which is associated with CMI to the fungus, generally heals spontaneously or responds nicely to treatment.
2. The chronic or non-inflammatory types of infection, which is associated with a failure to express CMI to the fungus at the site of infection, is relapsing and responds poorly to treatment.
Cutaneous mycoses
Tinea corporis
Cutaneous mycoses

- **THE IDENTIFICATION REACTION (ID)**
- Patients infected with a dermatophyte may show a lesion, often on the hands, from which no fungi can be recovered or demonstrated.
- It is believed that these lesions, which often occur on the dominant hand (i.e. right-handed or left-handed), are secondary to immunological sensitization to a primary (and often unnoticed) infection located somewhere else (e.g. feet).
- These secondary lesions will not respond to topical treatment but will resolve if the primary infection is successfully treated.
Cutaneous mycoses

- Laboratory diagnosis: scrapings from clinical specimens

- Hair – endothrix (spores inside the hair shaft)
  - ectothrix
  - exception: T.schoenleinii
  - Disease-favus-waxy
  - mass of hyphal elements (scutulum)
  - microscopic – degenerated hyphal elements
Cutaneous mycoses

- Cultures
- Selective media – containing cycloheximide and chloramphenicol → incubate at 25 C.
- Identification based on the conidia
Ringworm culture
Microsporum

[Image of septate hyphae labeled with "Septate hyphae"]

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<table>
<thead>
<tr>
<th>Genus</th>
<th>Macroconidia</th>
<th>Microconidia</th>
</tr>
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<tbody>
<tr>
<td>Microsporum</td>
<td>Numerous, thick walled, rough</td>
<td>Rare</td>
</tr>
<tr>
<td>Epidermophyton</td>
<td>Numerous, smooth walled</td>
<td>Absent</td>
</tr>
<tr>
<td>Trichophyton</td>
<td>Rare, thin walled, smooth</td>
<td>Abundant</td>
</tr>
</tbody>
</table>
Microsporum
Trichophyton
Microsporum
Epidermophyton floccusom
Diagnosis

- Diagnosis is based upon:
  1. Anatomical site infected
  2. Type of lesion
  3. Examination with a Woods lamp (366 Å)
  4. Examination of KOH-treated skin scales from the infected area
  5. Culture of the organism (not too important)
Differential diagnosis

- In a differential diagnosis you must consider:
  1. Leprosy
  2. Secondary syphilis
  3. Pityriasis rosea
  4. Psoriasis
  5. Nummular eczema
  6. Lichen planus
  7. Alopecia areata
  8. Trichotillomania
  9. Dyshidrosis
  10. Contact dermatitis.
Treatment

- Skin – azoles, inhibits cytochrome 450 dependent enzyme systems at the demethylation step from lanosterol to ergosterol
- Hair – Griseofulvin, oral, affects microtubular system
DIMORPHIC FUNGI

BLASTOMYCOSIS
(*Blastomyces dermatitidis*)

As was noted earlier, most of the systemic fungi have a specific niche in nature where they are commonly found. *Blastomyces dermatitidis* survives in soil that contains organic debris (rotting wood, animal droppings, plant material) and infects people collecting firewood, tearing down old buildings or engaged in other outdoor activities which disrupt the soil. In addition to an ecological niche, most fungi that cause systemic infections have a limited geographic distribution where they occur most frequently.
FIGURE 44-7 Schematic illustration of the natural history of the saprobic and parasitic cycle of *Blastomyces dermatitidis.*
FIGURE 44-8 Broad-based budding yeast cells of *Blastomyces dermatitidis* seen in purulent material expressed from a microabcess.
Blastomycosis is a chronic granulomatous disease which means that it progresses slowly. Although the pulmonary and skin involvement is the most common, *B. dermatitidis* frequently affects bone, prostate and other organs. More frequently blastomycosis presents as a cutaneous or a respiratory disease. The cutaneous lesions may be primary (usually self-limiting) or secondary (a manifestation of systemic disease). The patient who presents with a complaint of respiratory symptoms will frequently remark about loss of appetite, loss of weight, fever, productive cough, and night sweats. While these symptoms resemble those of TB, it is not this disease. The X-ray shows obvious pulmonary disease. To make the specific diagnosis, the physician must be aware of blastomycosis.
Sputum sent to the lab for "culture" will not grow the organism. The lab must be alerted to look for fungal organisms or to look specifically for blastomyces. Some patients have a sub-clinical or "flu-like" response to infection. *B. dermatitidis* can frequently be demonstrated in a KOH preparation of pus from a skin lesion. A typical cutaneous lesion shows central healing with micro-abscesses at the periphery. A pus specimen may be obtained by nicking the top of a microabscess with a scalpel, obtaining the purulent material and making the diagnosis in 5 min. by microscopic examination with KOH. This organism has a characteristic appearance of a double contoured wall with a single bud on a wide base.
There are no specific virulence factors for *B. dermatitidis*. Laboratory specimens depend on the manifestation of the disease: If there are skin lesions, send skin scrapings or pus. If there is pulmonary involvement, send sputum. Other specimens include biopsy material and urine. Occasionally, the organism can be isolated from urine as it often infects the prostate.
**Mycology**

If you request a fungus culture from the microbiology lab, they will incubate the cultures at 37º C and at 25º C because most of the significant pathogenic fungi are dimorphic. A culture of *B. dermatitidis* takes 2 to 3 weeks to grow at 25 º C. It appears as a white, cottony mold (mycelium) on Sabouraud dextrose agar. Most specimens for fungus culture are plated on Sabouraud's dextrose agar.
**Microscopically**, the mycelia and the fruiting bodies are evident. However, the mold cannot be identified by its fruiting bodies. The fruiting bodies are called **microconidia**, but they are not distinctive. Other fungal saprophytes and pathogens have similar conidia. At 37°C the yeast form grows in about 7-10 days. It appears as a buttery-like, soft colony with a tan color. Microscopically, we see the typical yeast form of a thick wall and a single bud with a WIDE BASE. This wide base is characteristic of *B. dermatitidis*, and it is important to be able to recognize this. The cells are 12-15 µ in diameter. The yeast will convert to the mycelial form when incubated at 25º C, taking from 3 to 4 days up to a few weeks. Similarly, the mycelial growth can be converted to yeast form when incubated at 37ºC. In the past, the only way to identify the dimorphic fungi was to convert from one form to the other, but now it is possible to take the mycelial growth (which is the easiest to grow), and confirm the isolate with a DNA probe in a matter of hours.
**Histopathology**

*B. dermatitidis* produces both a granulomatous and suppurative tissue reaction

**Serology**

There are 3 serological tests used for blastomycosis:

- **Immunodiffusion test (precipitin).** This requires 2 to 3 weeks to become positive. This test is positive in about 80% of the patients with blastomycosis. When it is positive, there is close to 100% specificity.

- **Complement fixation (CF) test.** This test requires 2 to 3 months after the onset of disease to develop detectable antibody. Besides the long delay before there is measurable antibody, another disadvantage of the C-F is that it cross reacts with other fungal infections (coccidioidomycosis and histoplasmosis). The advantage is that it is a quantitative test. The physician can follow the patient's response to the disease by monitoring the antibody titer.

- **Enzyme Immunoassay (EIA).** The latter test has met with mixed acceptance by mycologists. However it is easy to perform and antibody is detected early in the disease process.
Amphotericin B remains is the drug of choice (DOC) although it is very toxic and must be administered intravenously for several weeks. Ketoconazole is also being used in mild cases.
Thank you very much!!!