

Before you begin: This is a big topic, and big topics beget big slide-sets. There's a natural break near slide 205; a *Break time!* slide has been placed at that point to mark it.

Toxoplasmosis: Basics

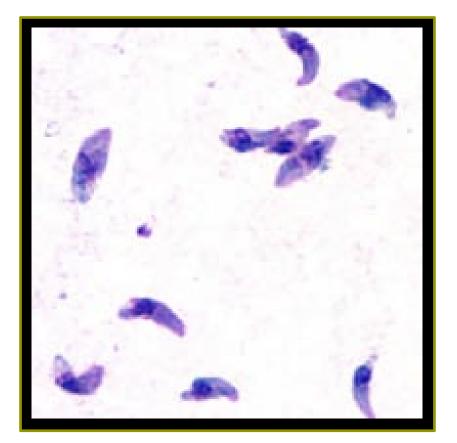
What is the causative organism in ocular toxoplasmosis?



Toxoplasmosis: Basics

What is the causative organism in ocular toxoplasmosis? *Toxoplasma gondii*







Toxoplasma gondii

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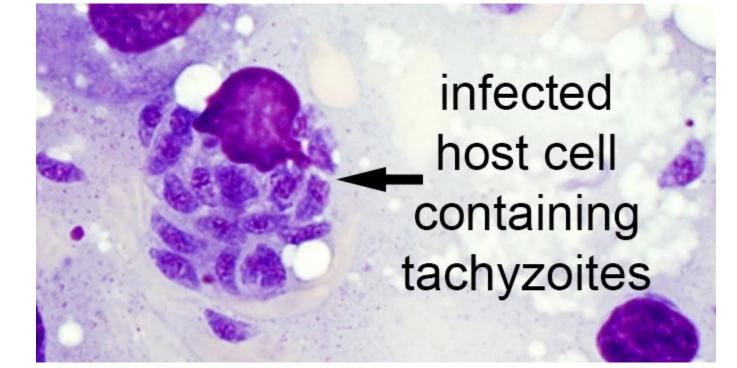
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Toxoplasma gondii intracellular

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A cat

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What percent of the IgG-positive US population have signs of ocular involvement? About 2



Toxoplasmosis: Basics

T gondii has a complex life cycle, existing in ____ forms.



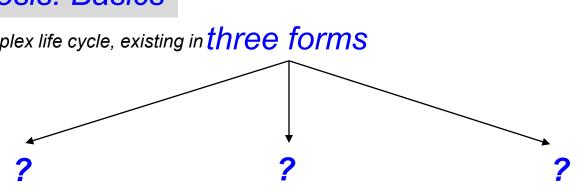
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T gondii has a complex life cycle, existing in three forms.

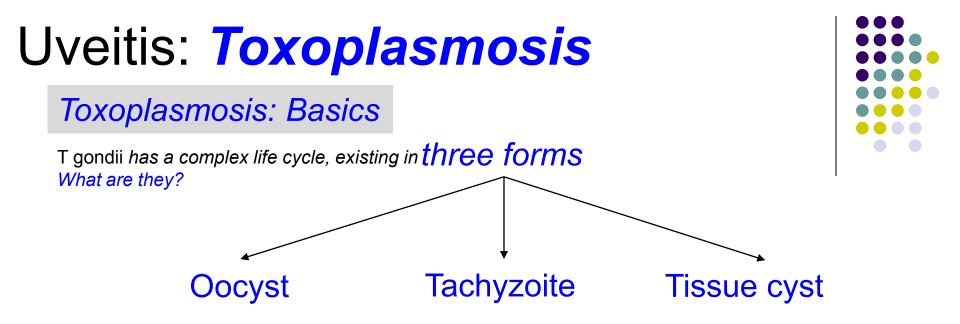


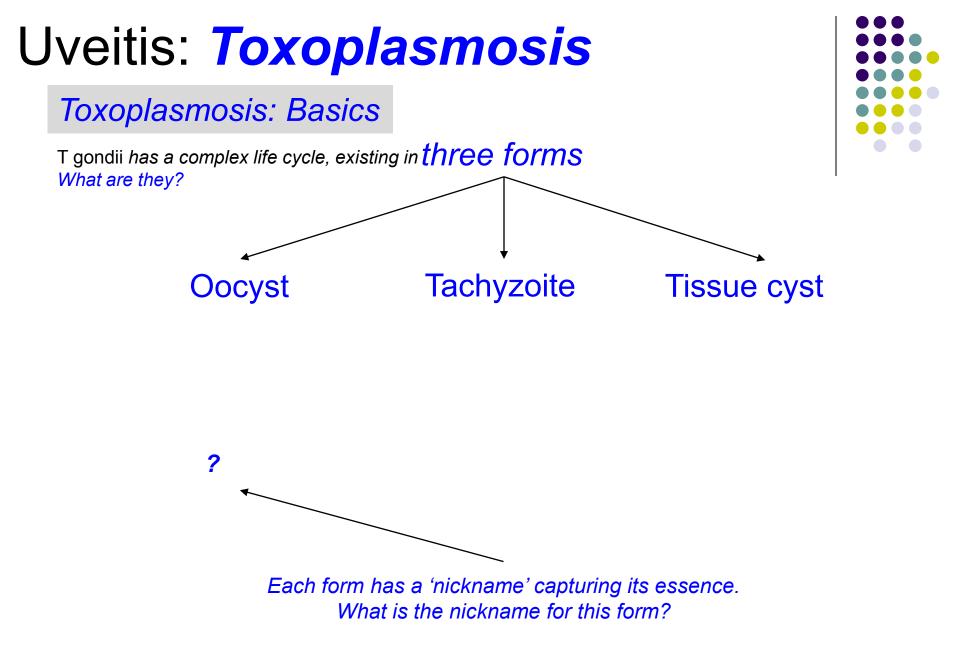
Toxoplasmosis: Basics

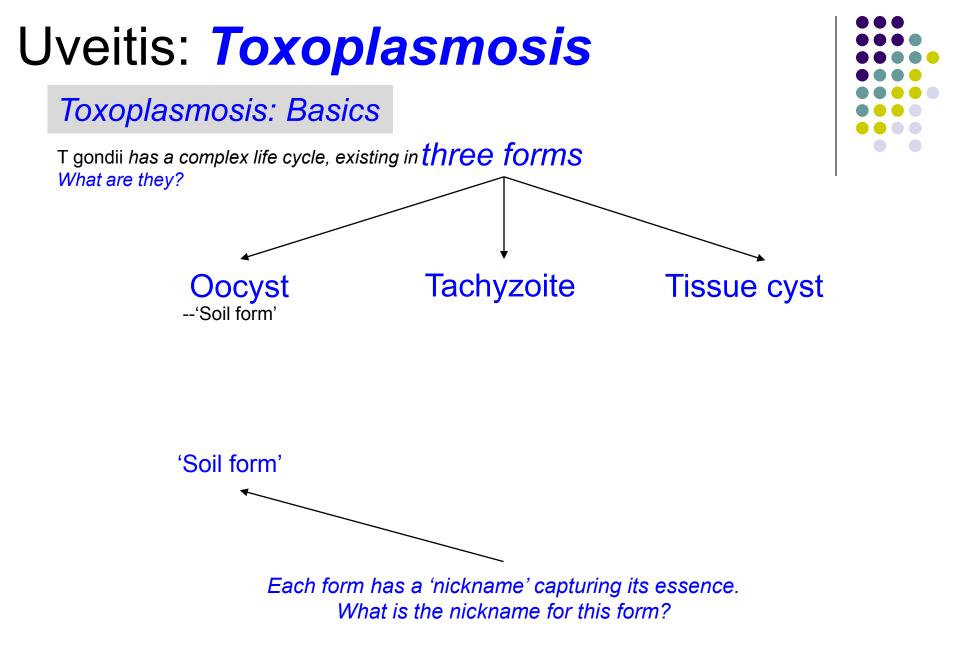
T gondii has a complex life cycle, existing in three forms What are they?

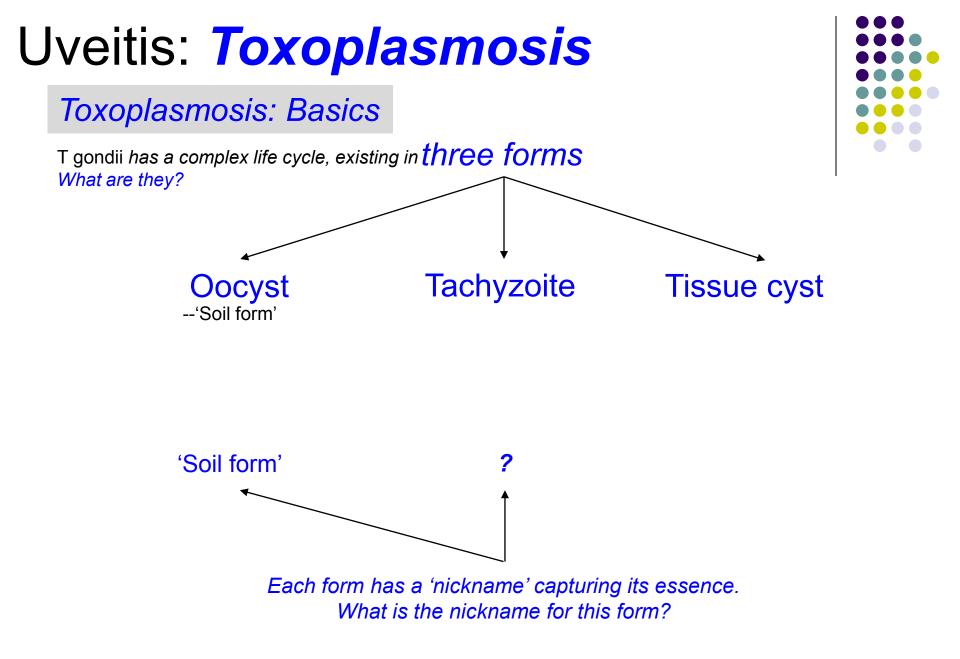


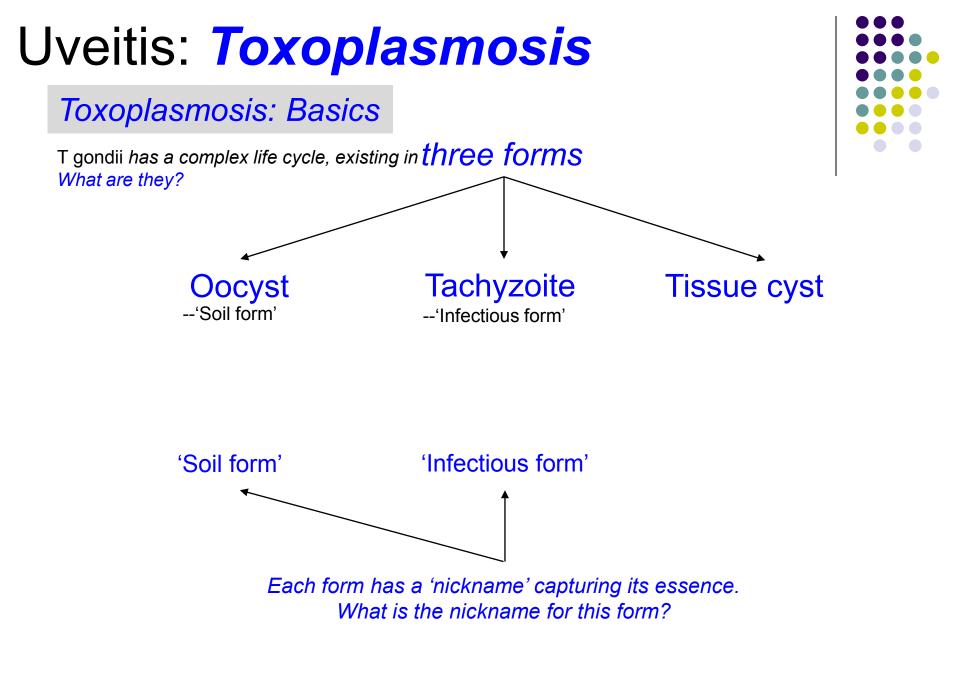


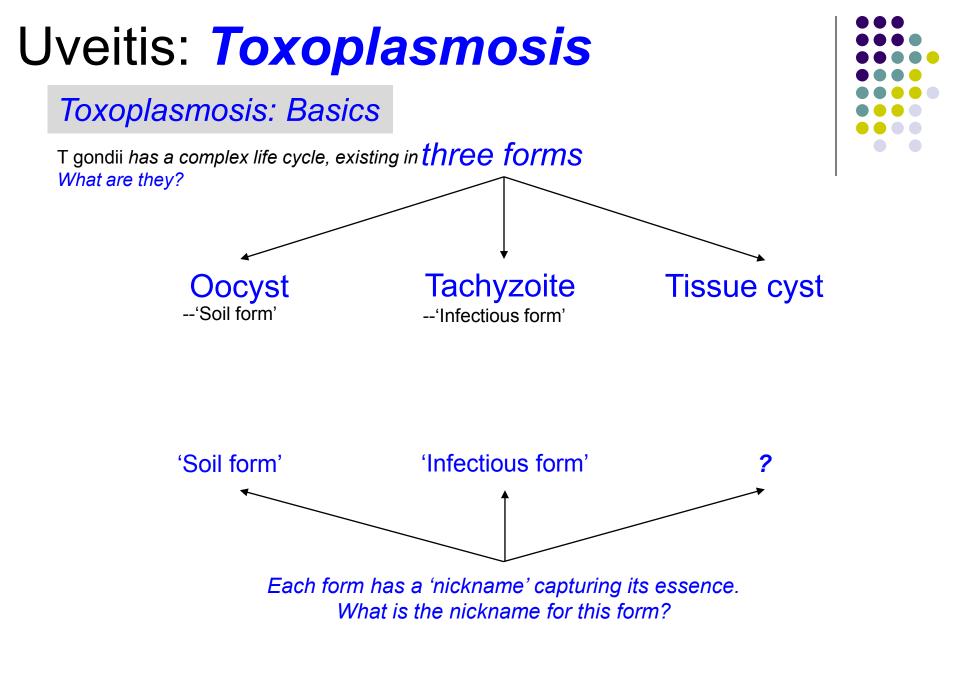


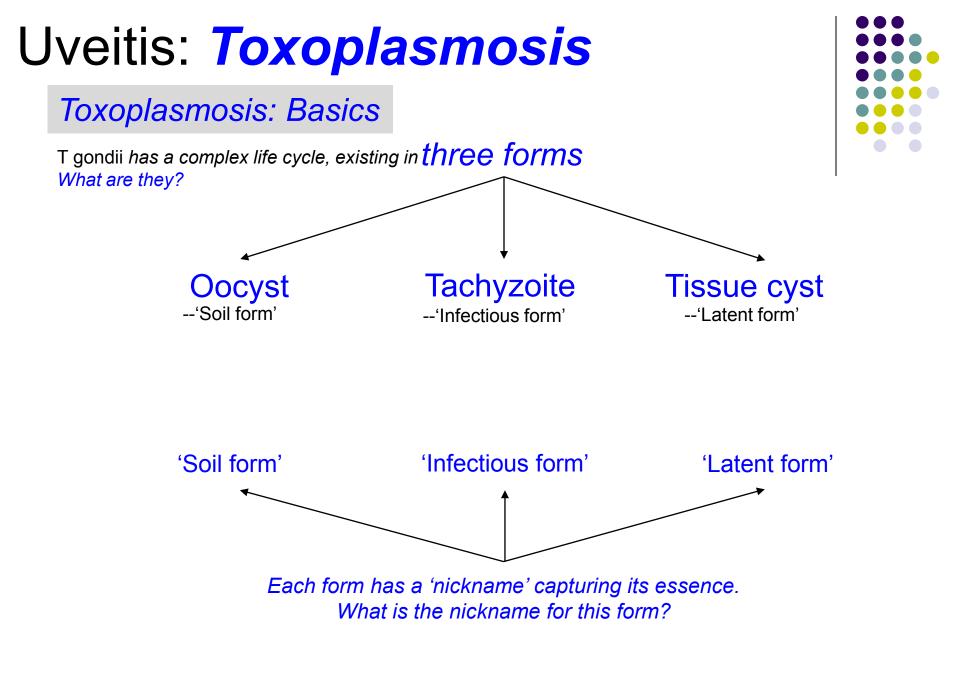


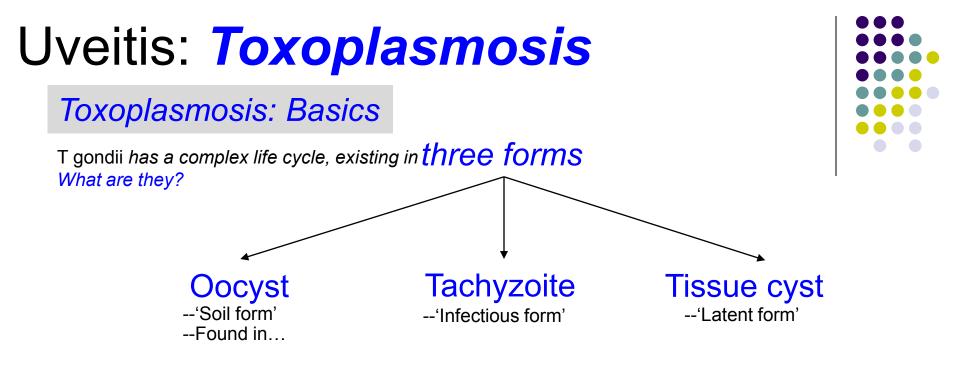


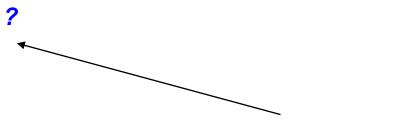




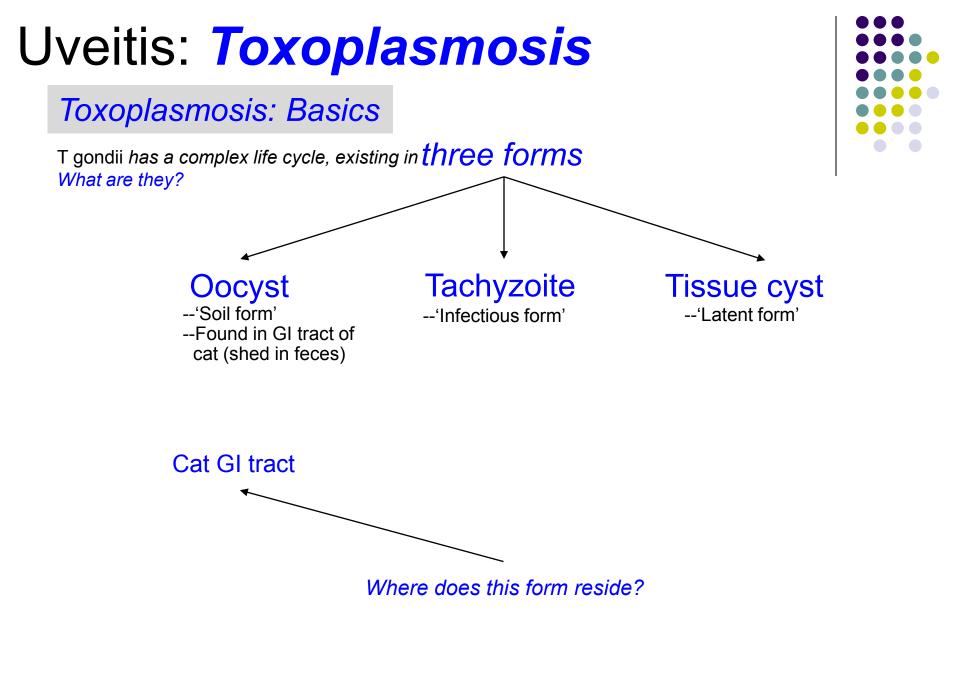


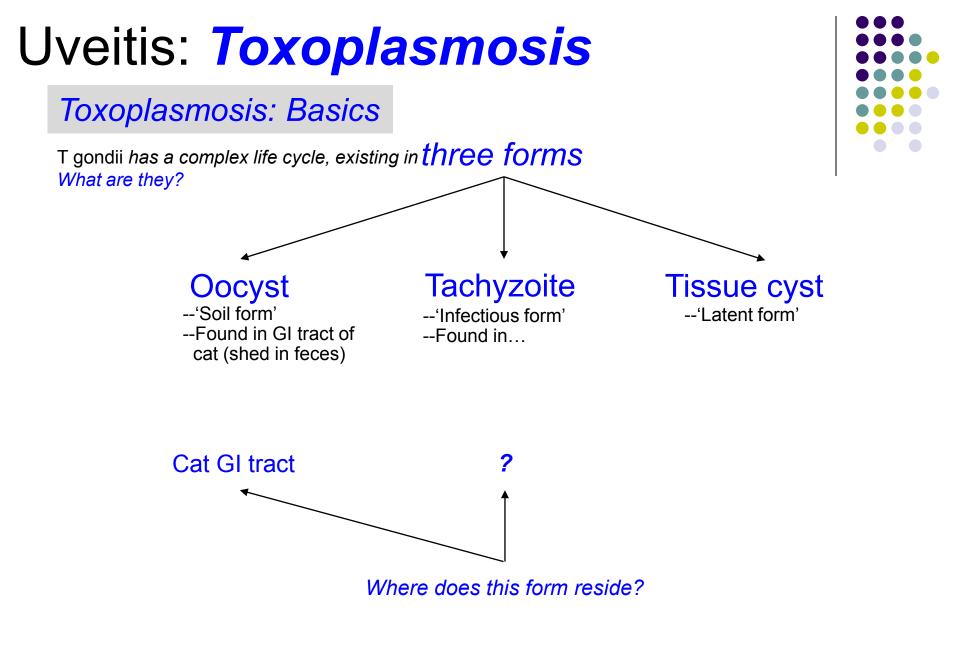


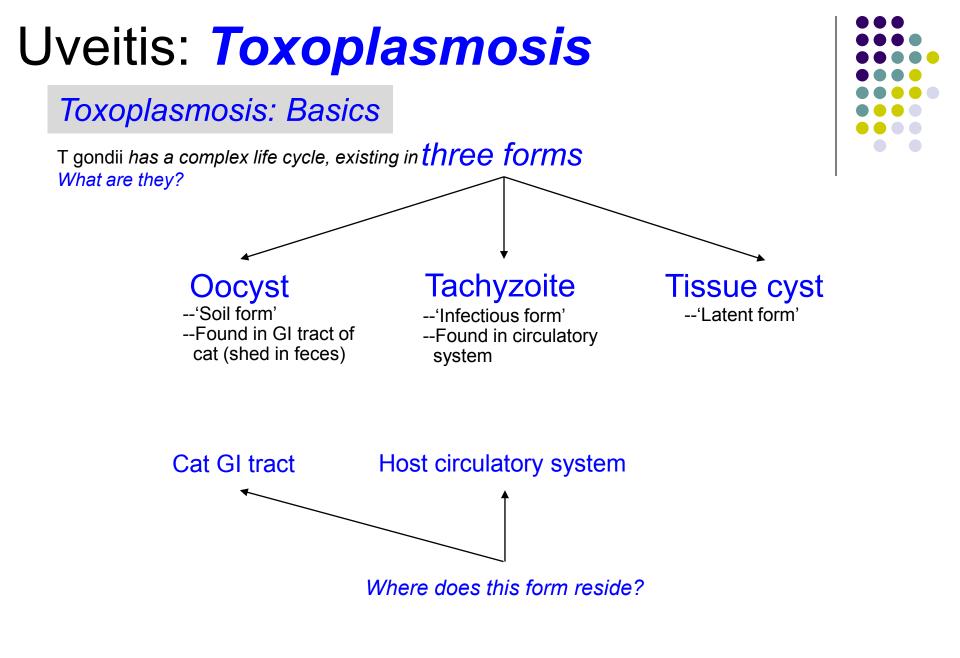


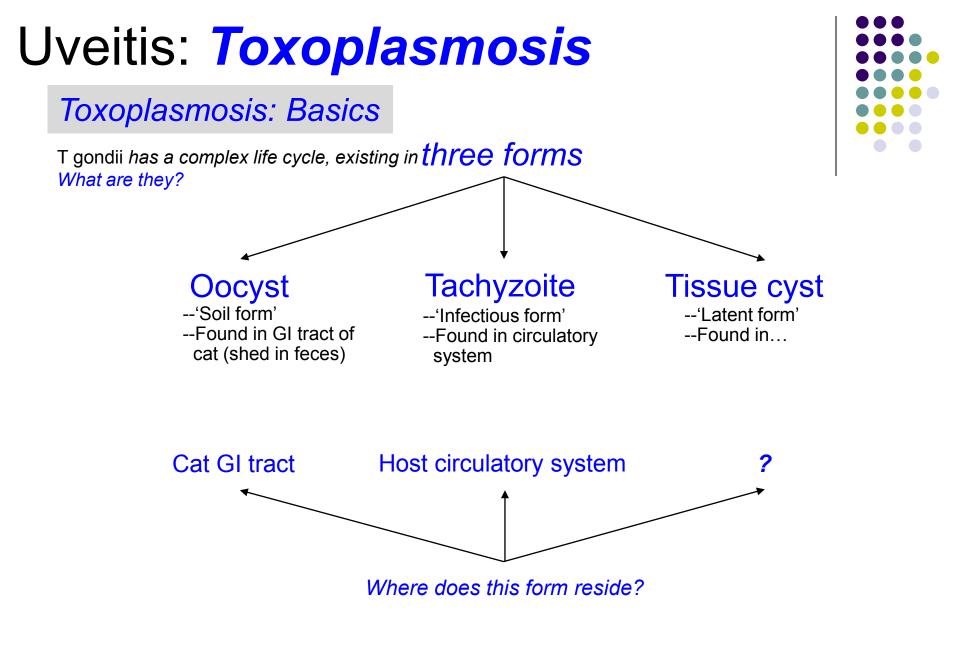


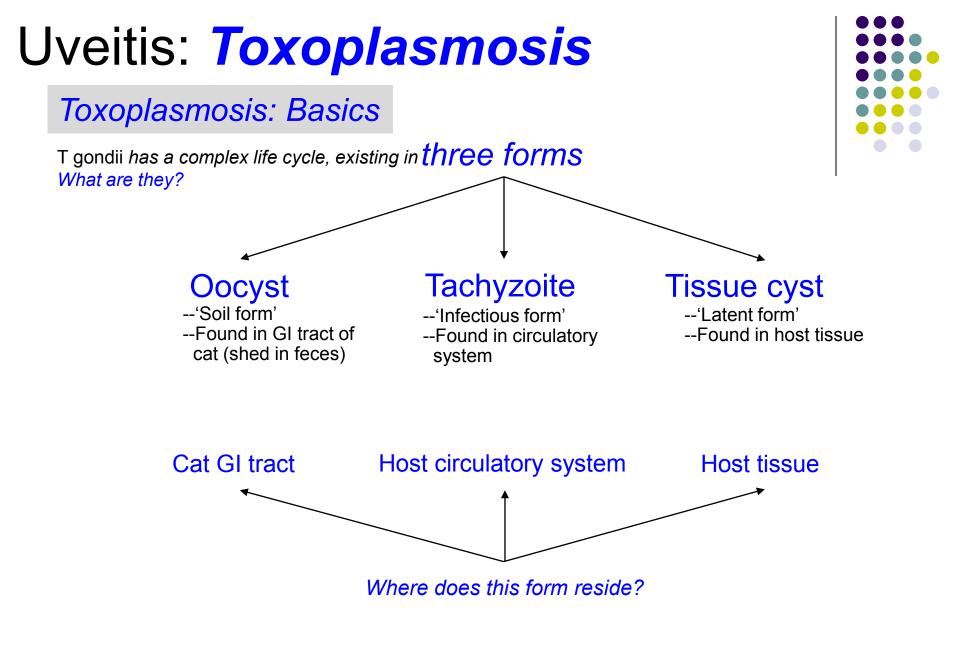
Where does this form reside?

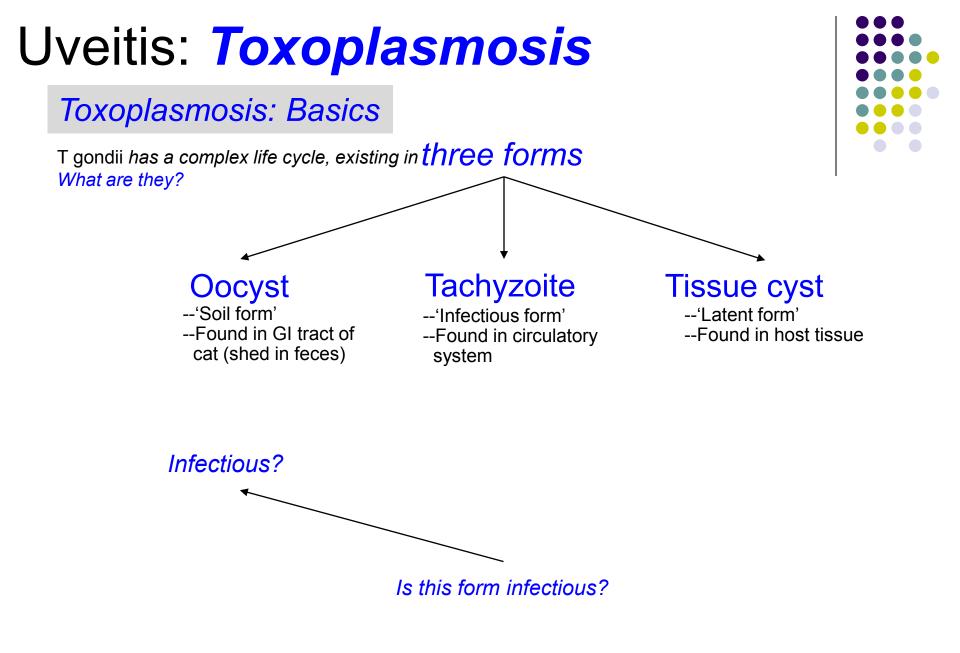


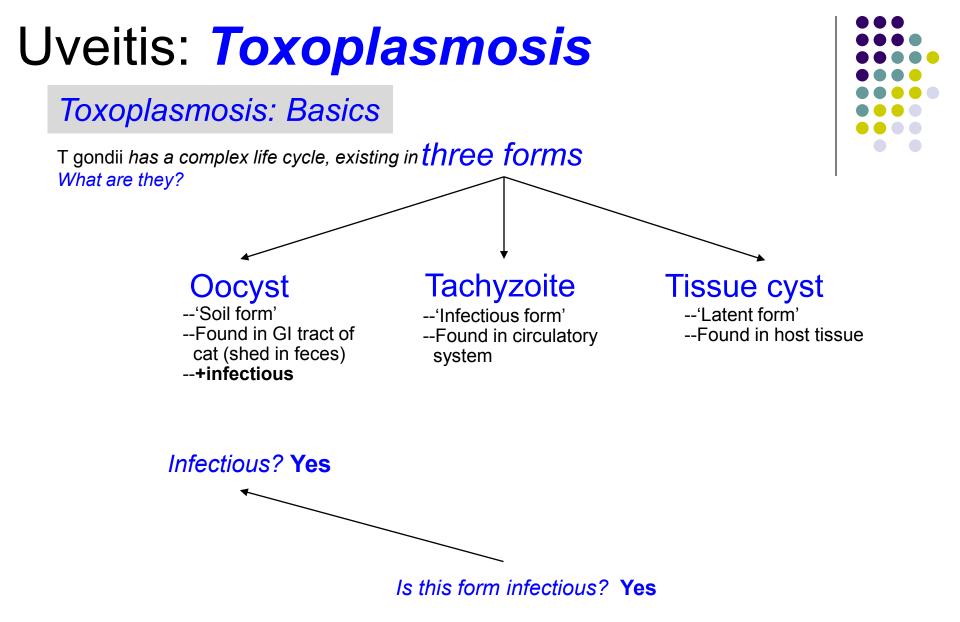


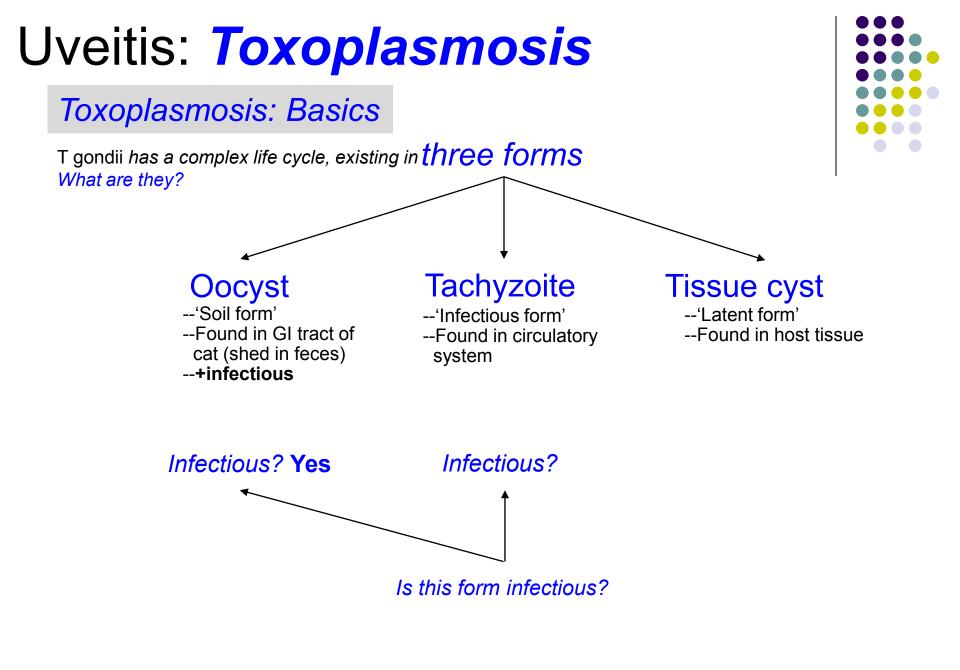


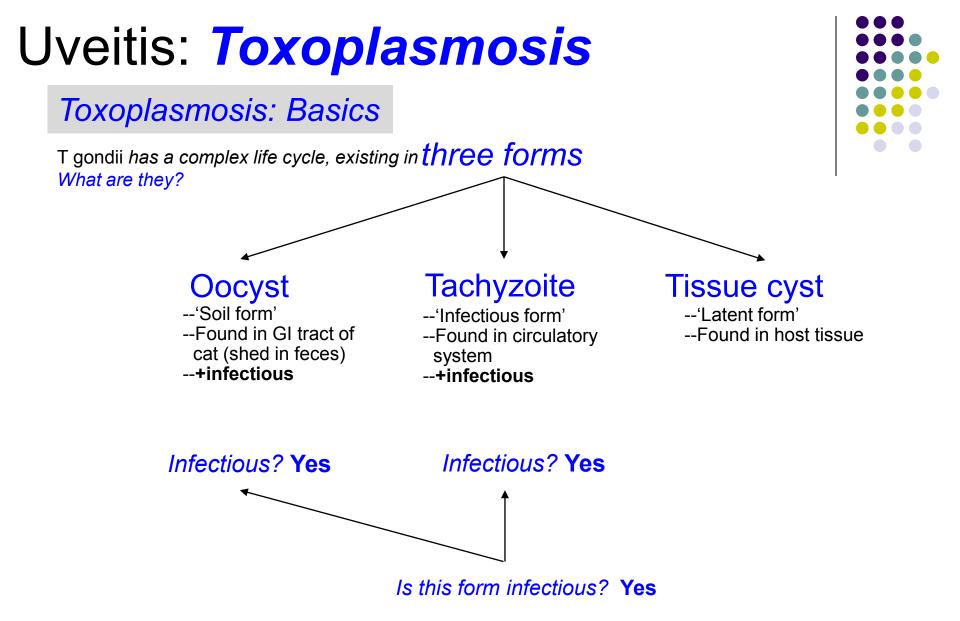


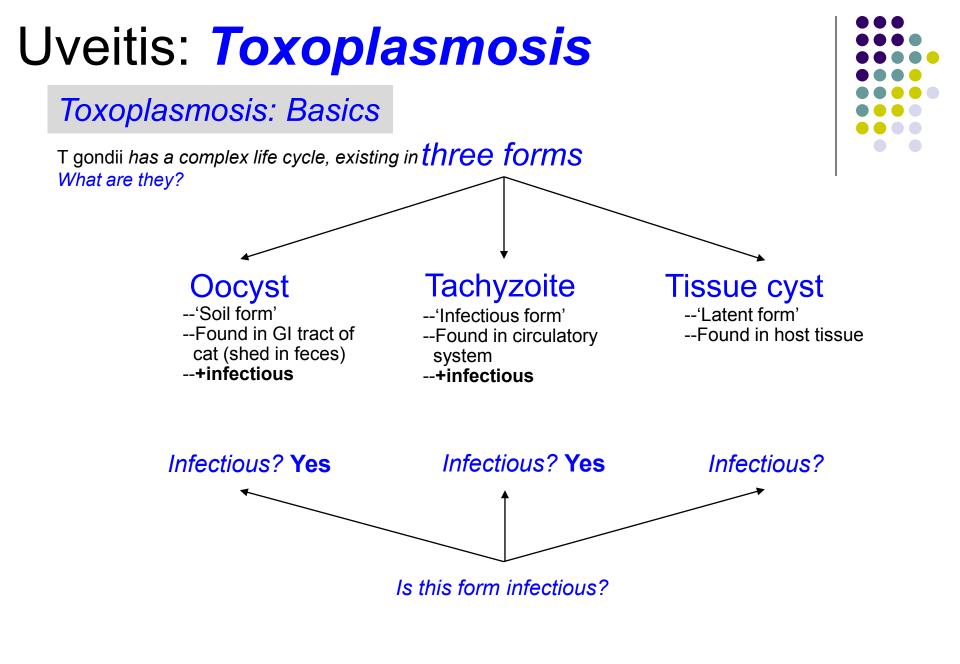


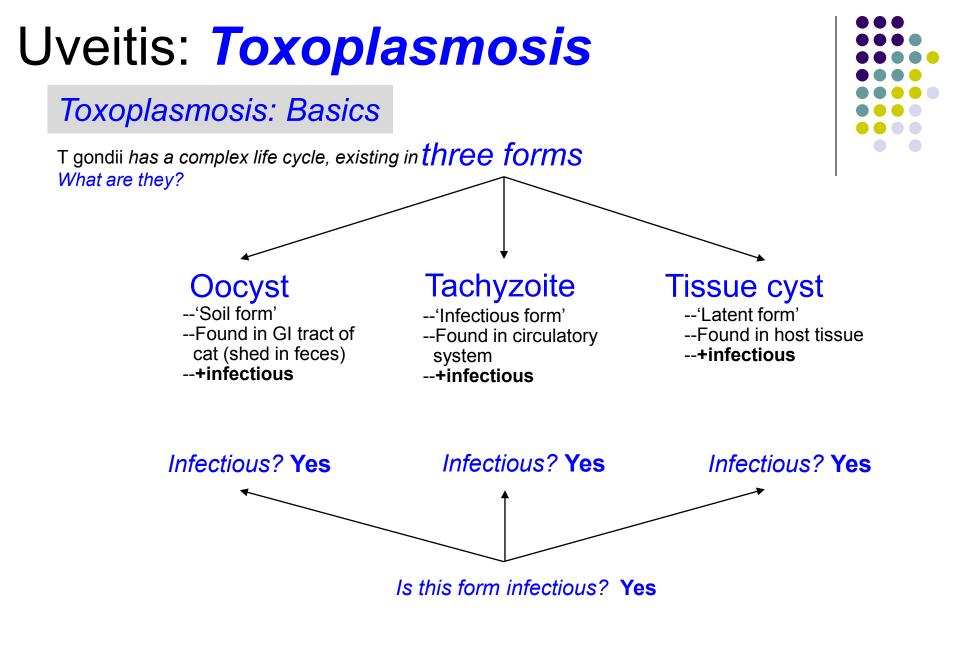


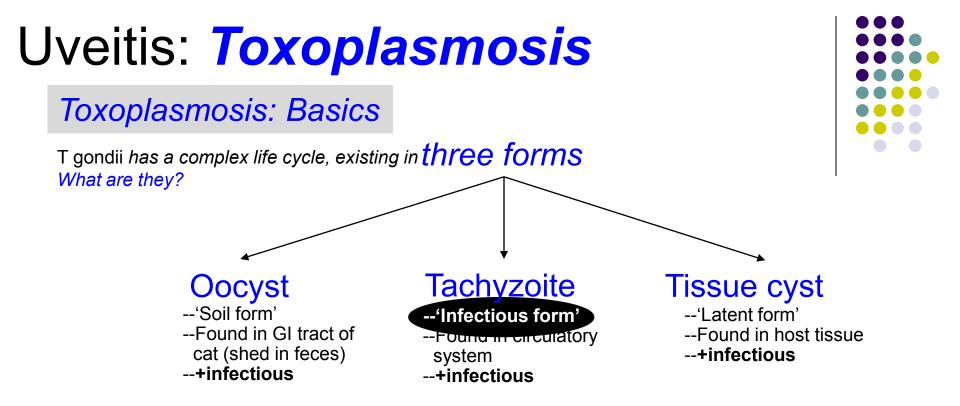






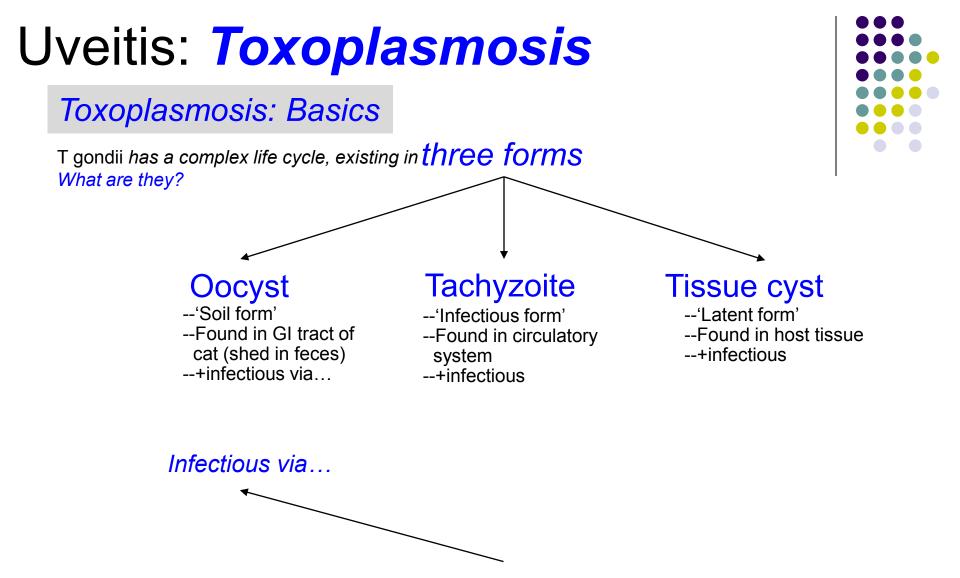




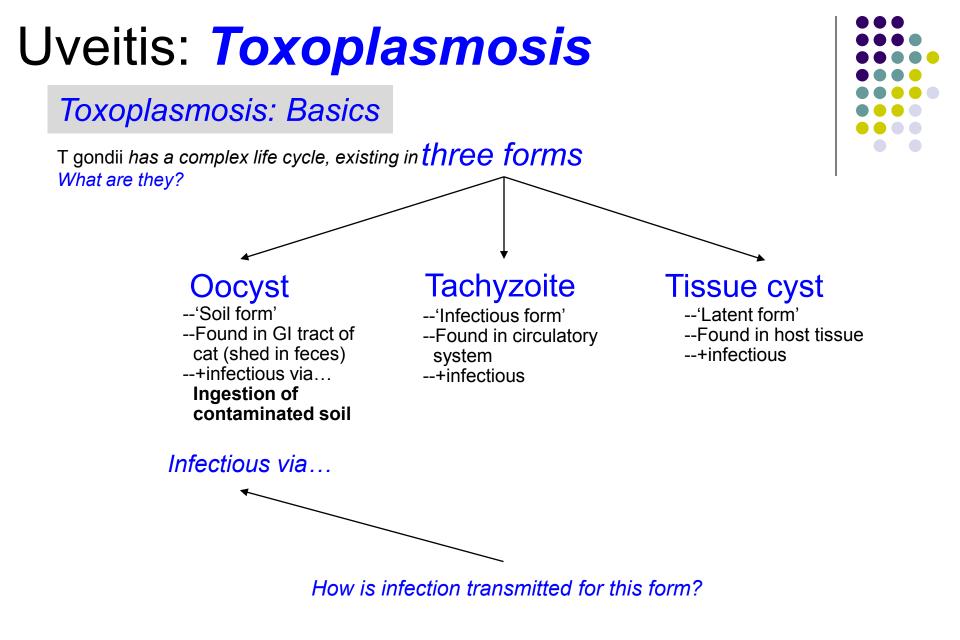


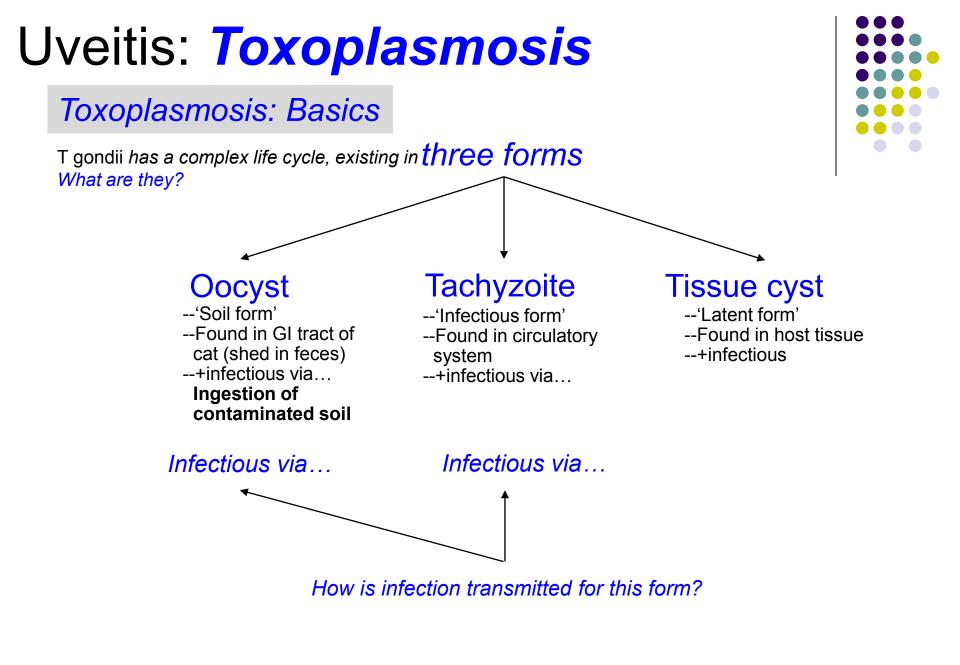
Infectious? Yes Infectious? Yes Infectious? Yes

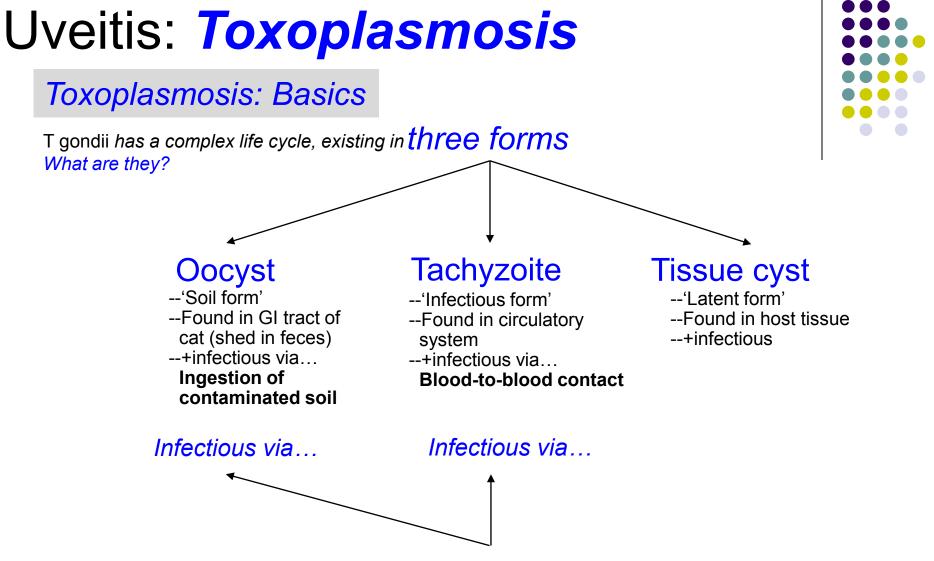
All forms are infectious under the right circumstances! Don't let the nickname of the tachyzoite form fool you into thinking it's the only one.



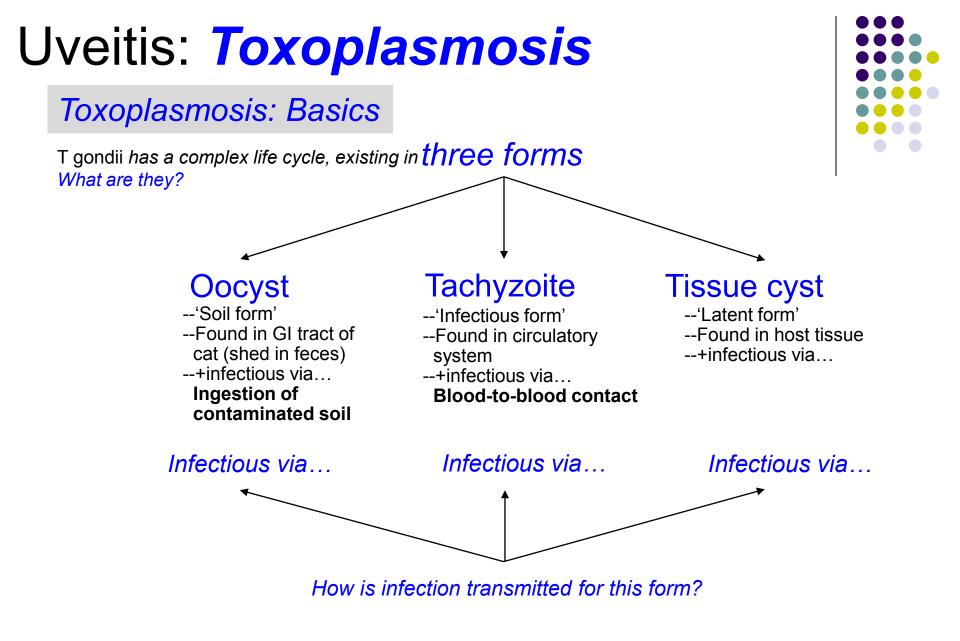
How is infection transmitted for this form?

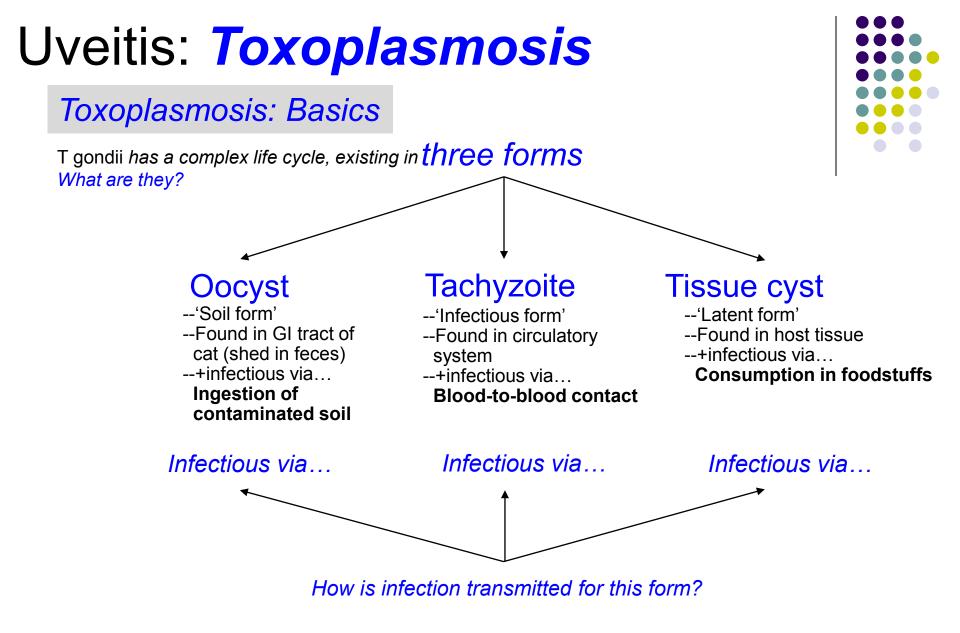


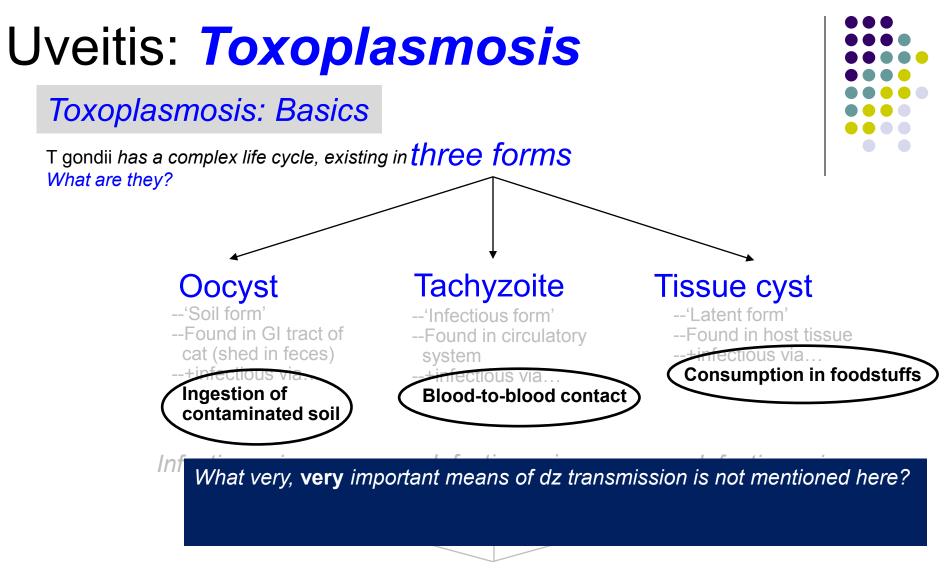




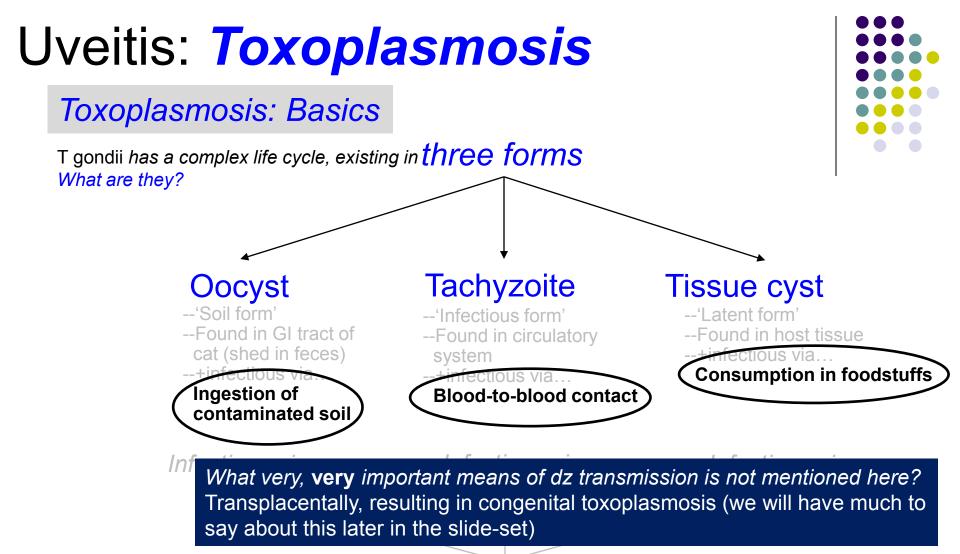
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Uveitis: Toxoplasmosis Toxoplasmosis: Basics T gondii has a complex life cycle, existing in three forms What are they? **Tachyzoite Tissue cyst** Oocyst --'Soil form' --'Infectious form' --- 'Latent form' --Found in GI tract of --Found in host tissue --Found in circulatory cat (shed in feces) --+infectious via... system --+infectious via... --+infectious via... Consumption in foodstuffs Ingestion of Blood-to-blood contact contaminated soil --? In a nutshell...

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In a nutshell, how should we think of each form? --Oocysts...

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--Found in circulatory

Blood-to-blood contact

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In a nutshell, how should we think of each form? --Oocysts...are toxo eggs or 'spores' (the bug is a

--Found in GI tract of

cat (shed in feces)

contaminated soil

--+infectious via...

Ingestion of

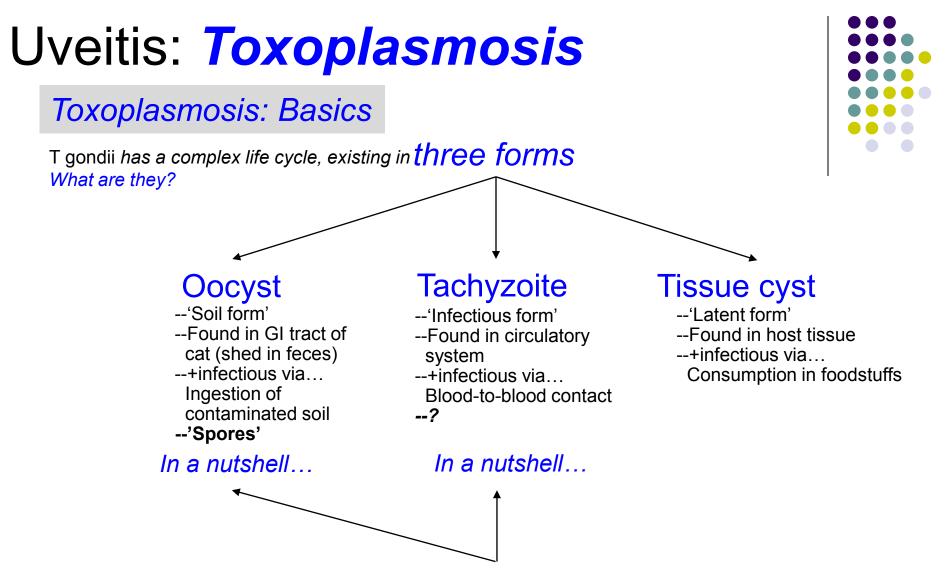
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--Oocysts...are toxo eggs or 'spores' (the bug is a *sporozoite* at this stage) --Tachyzoites...

Toxoplasmosis: Basics

T gondii has a complex life cycle, existing in **three forms** What are they?



Oocyst

---'Soil form' --Found in GI tract of cat (shed in feces) --+infectious via... Ingestion of contaminated soil --'Spores'

Tachyzoite

- --'Infectious form'
- --Found in circulatory
 - system
- --+infectious via...
- Blood-to-blood contact
- --'Active adult'

Tissue cyst

- --'Latent form'
- --Found in host tissue
- --+infectious via... Consumption in foodstuffs

In a nutshell...

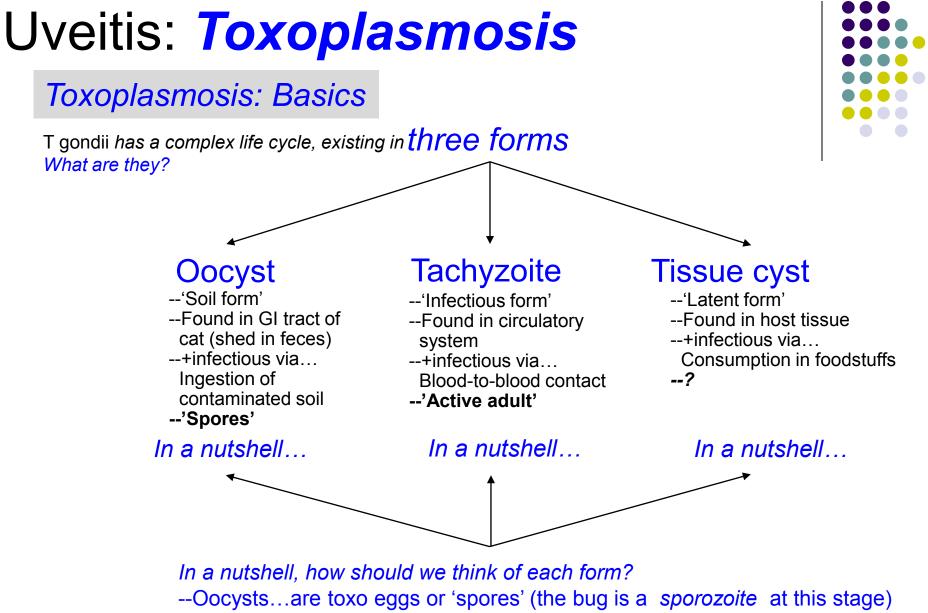
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Tissue cyst

- --- 'Latent form'
- --Found in host tissue
- --+infectious via...
- Consumption in foodstuffs
- --'Dormant adult'

In a nutshell...

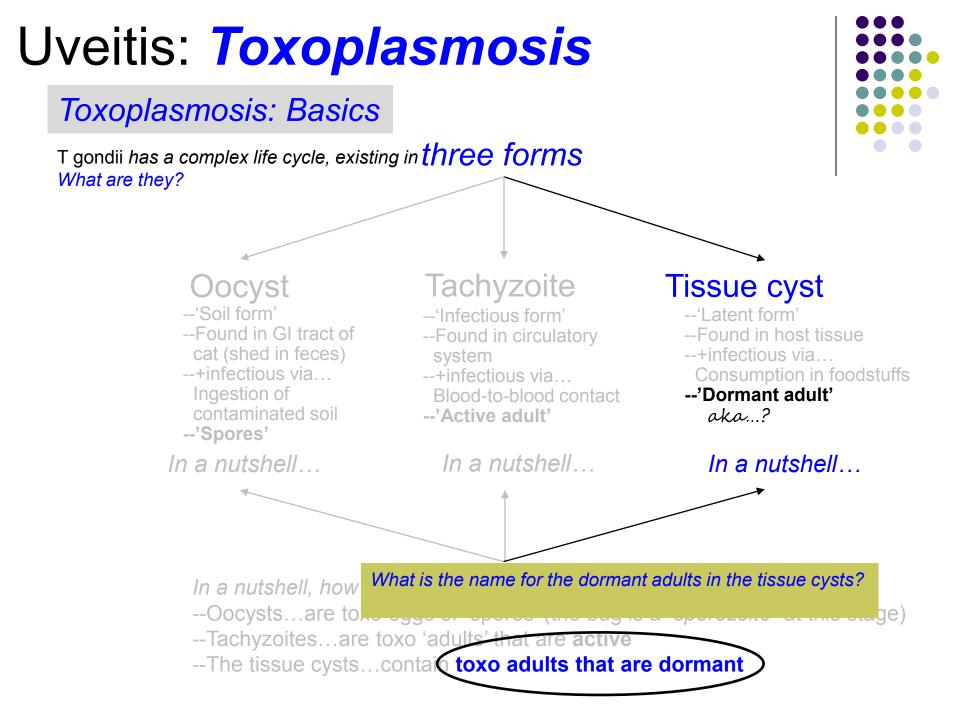
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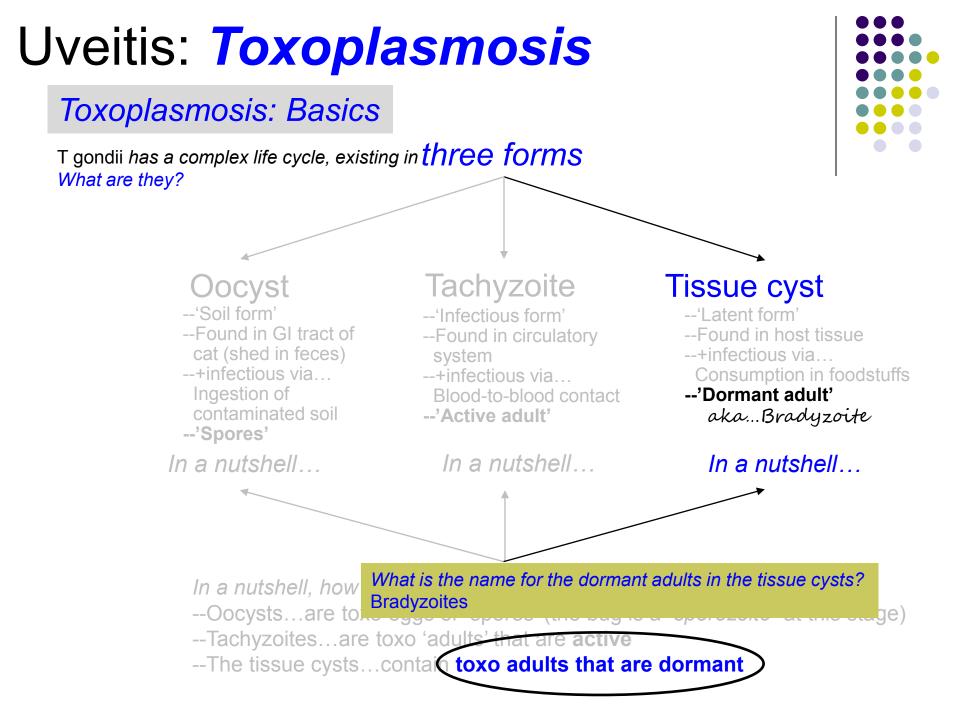
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Toxoplasmosis: Basics

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Tachyzoite

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- --+infectious via
- --+INIECLIOUS VIA...
- Blood-to-blood contact
- --'Active adult'

In a nutshell...

Tissue cyst

- --'Latent form'
- --Found in host tissue
- --+infectious via...
- Consumption in foodstuffs
- --'Dormant adult' aka...Bradyzoite



It's not a coincidence that the *dormant* adults are identified as *brady* ('slow') –zoites...

nt adults in the tissue cysts?

--Oocvsts...are to Bradyzoites

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It's not a coincidence that the *dormant* adults are identified as *brady* ('slow') –zoites...whereas the *active* adults are *tachy* ('fast') –zoites

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Blood-to-blood contact

In a nutshell...

--'Active adult'

In a nutshell...

--'Dormant adult'

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Ingestion of

In a nutshell...

--'Spores'

contaminated soil

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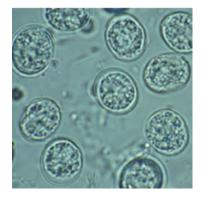
★ Summary slide—no question ★

Toxoplasmosis: Basics

Toxoplasmosis Basics tl;dr

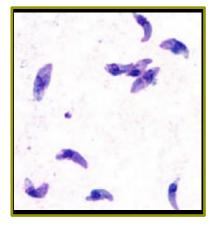
The person consumes either oocysts (wash your hands!) or tissue cysts (cook your meat!).

Oocyst ---'Soil form' --Found in GI tract of cat (shed in feces) ---+infectious via... Ingestion of contaminated soil ---'Spores'



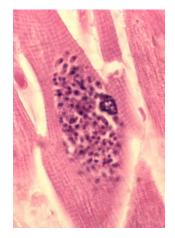
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Toxoplasmosis: Basics

Toxoplasmosis Basics tl;dr

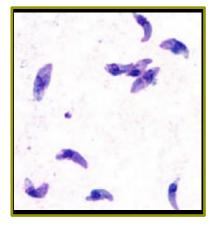
The person consumes either oocysts (wash your hands!) or tissue cysts (cook your meat!). The consumed bugs transform into tachyzoites, enter the bloodstream, then disseminate throughout the body.

Oocyst --'Soil form' --Found in GI tract of cat (shed in feces) --+infectious via... Ingestion of contaminated soil --'Spores'



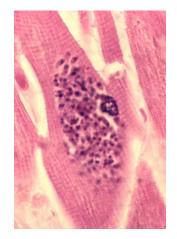
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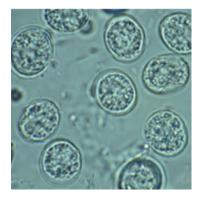


Toxoplasmosis: Basics

Toxoplasmosis Basics tl;dr

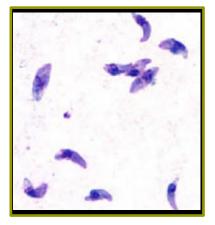
The person consumes either oocysts (wash your hands!) or tissue cysts (cook your meat!). The consumed bugs transform into tachyzoites, enter the bloodstream, then disseminate throughout the body. The immune system quickly clears the circulating parasites, but not before some get encased in tissue cysts, which are impervious to the host's immune system.

> Oocyst ---'Soil form' --Found in GI tract of cat (shed in feces) ---+infectious via... Ingestion of contaminated soil ---'Spores'



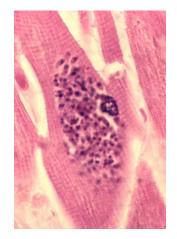
Tachyzoite

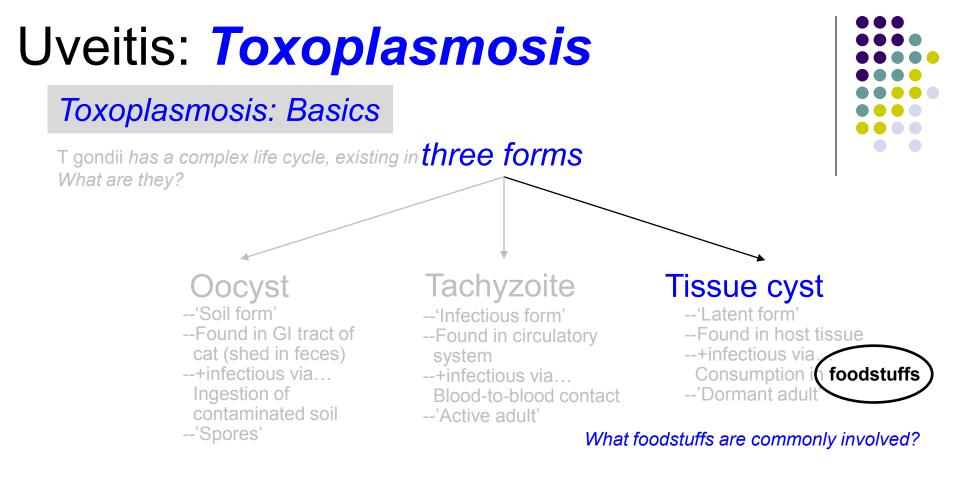
- --- 'Infectious form'
- --Found in circulatory system
- --+infectious via...
- Blood-to-blood contact
- --'Active adult'

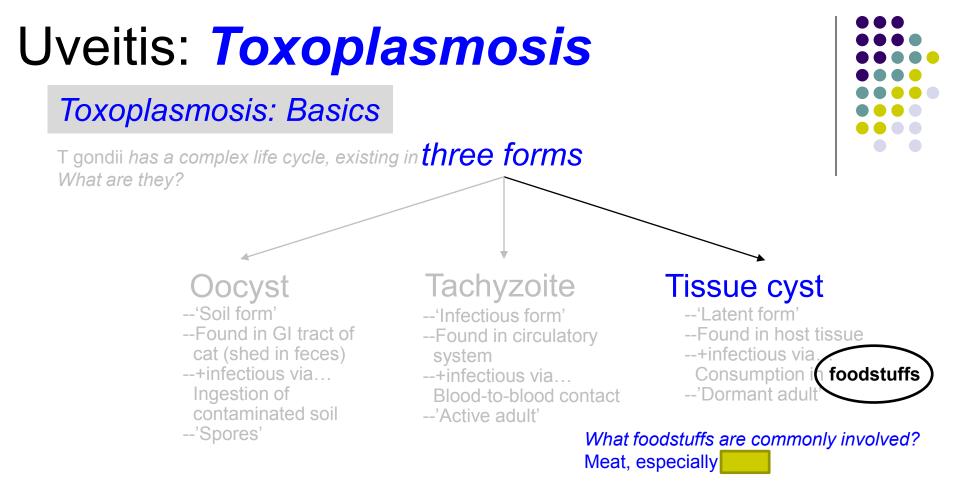


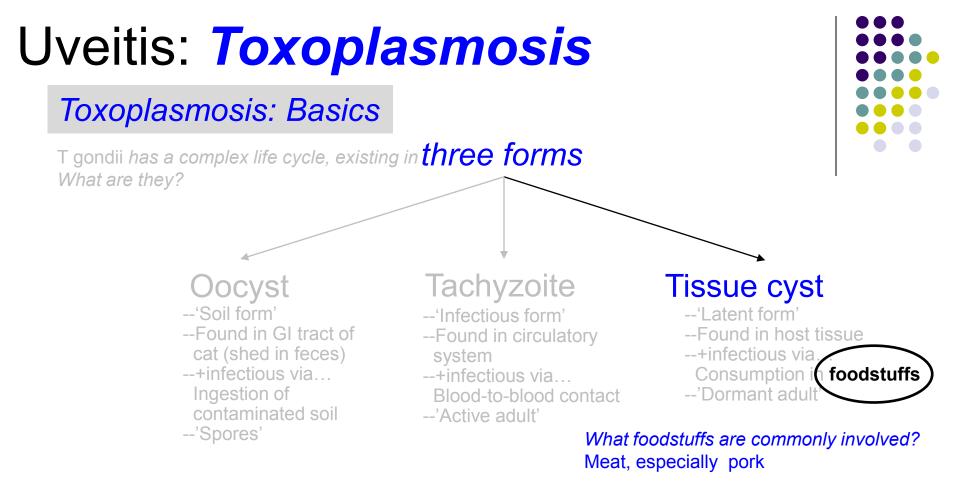
Tissue cyst

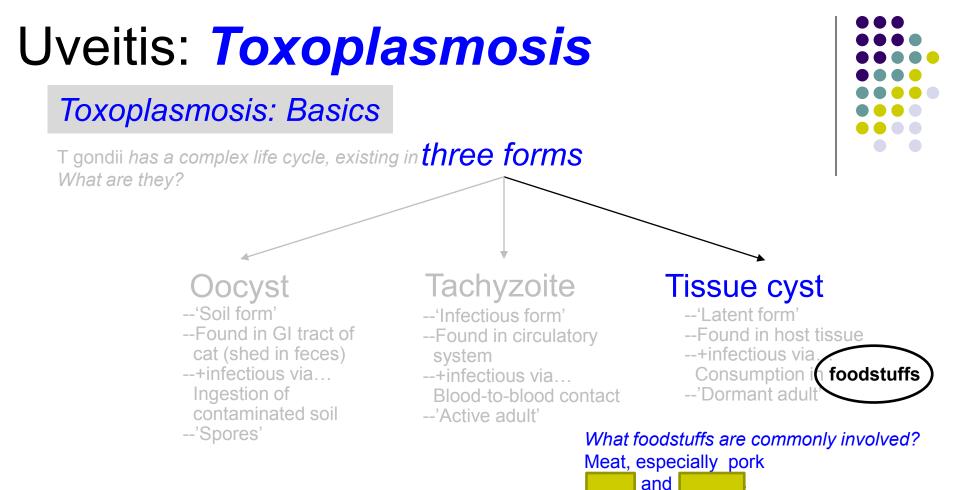
- --- 'Latent form'
- --Found in host tissue
- --+infectious via...
- Consumption in foodstuffs
- --'Dormant adult'

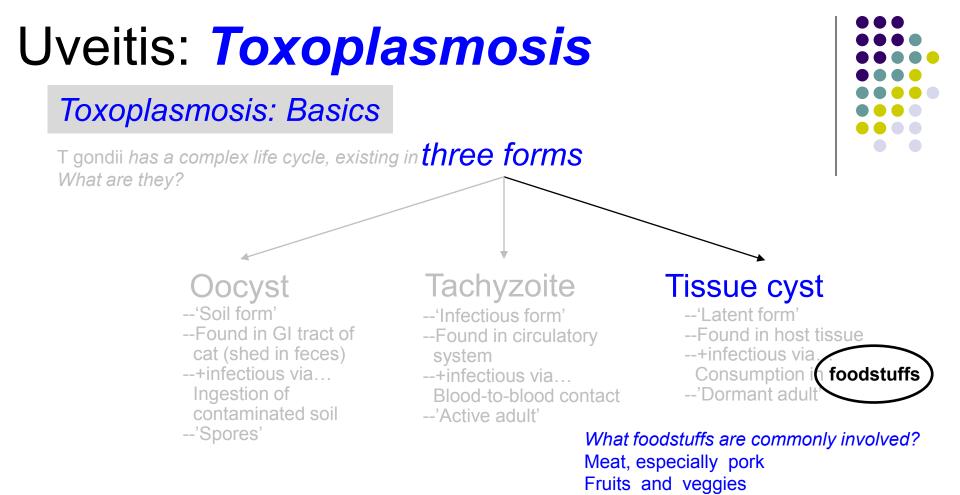


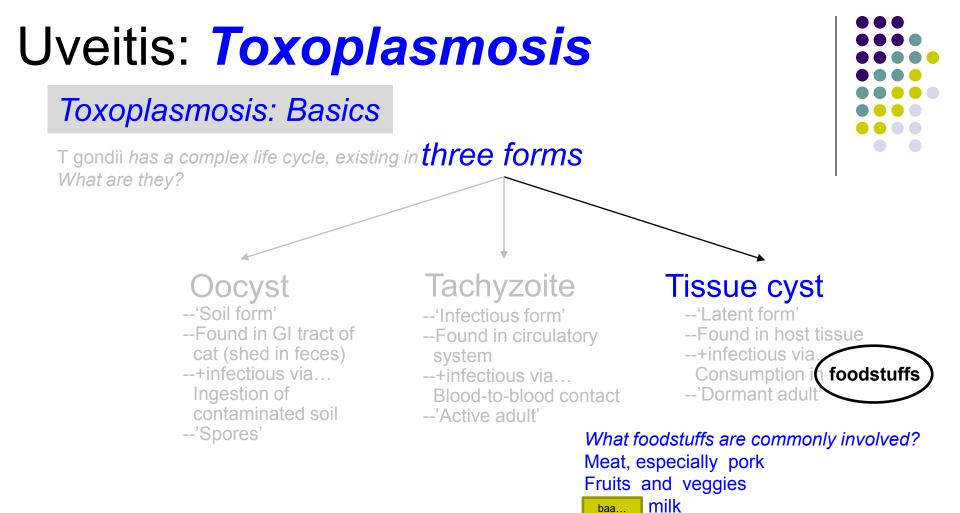


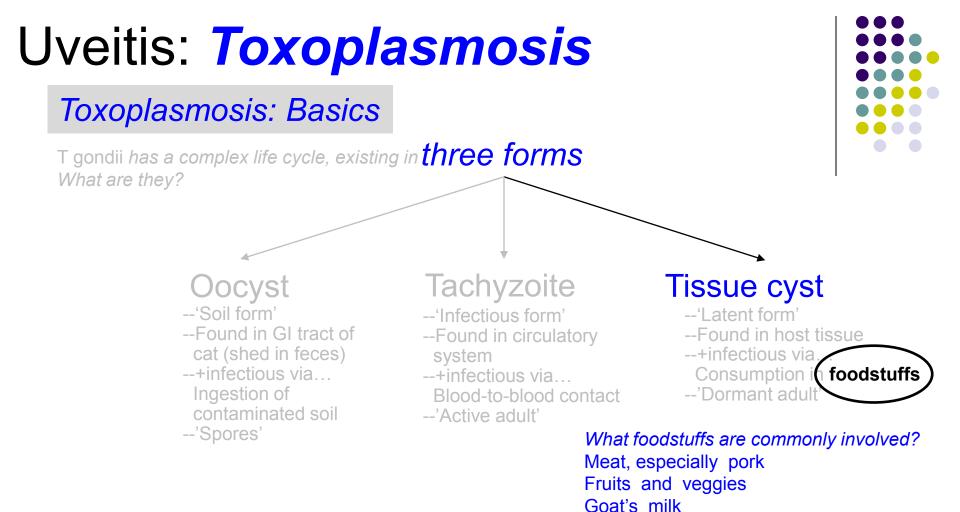


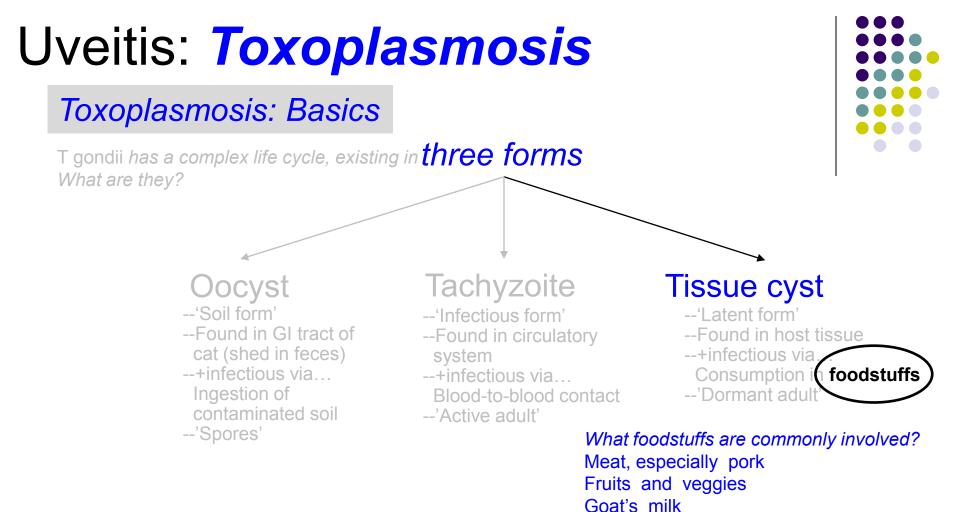




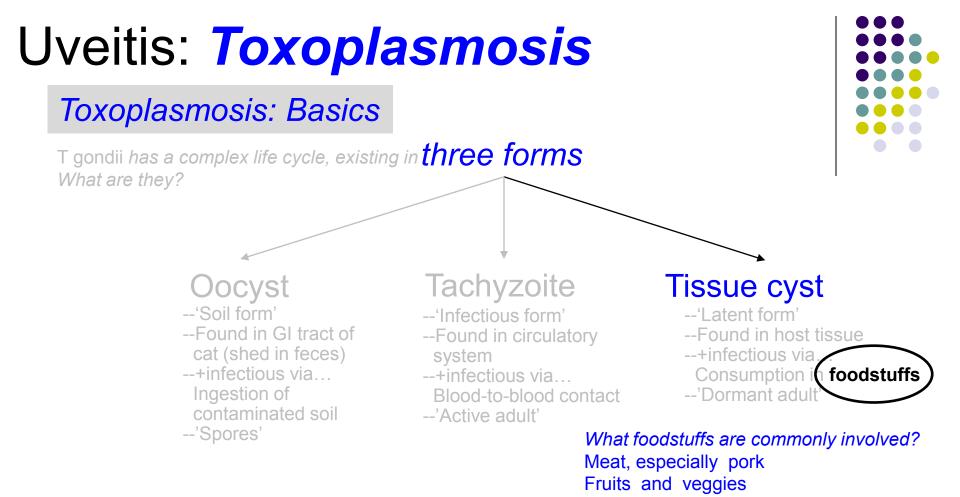






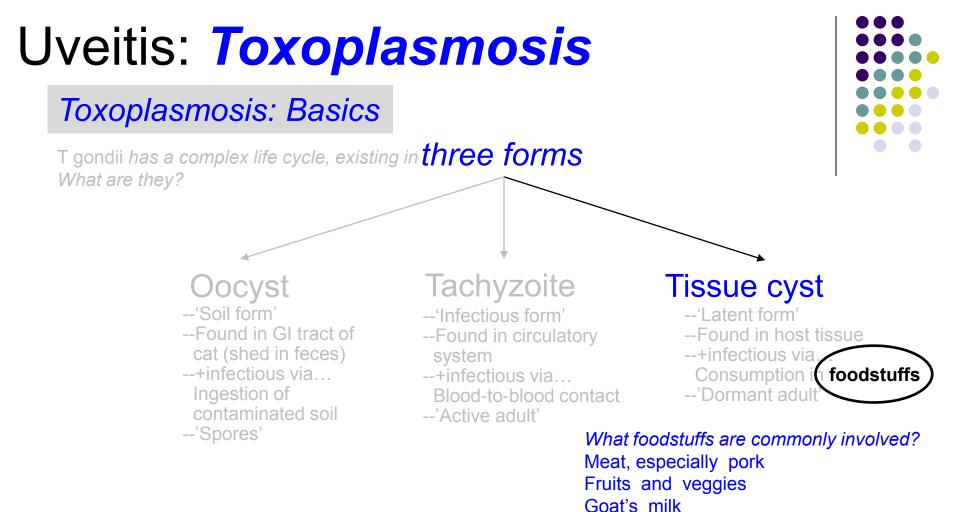


How does toxo get into the animals and/or onto the fruits and veggies?



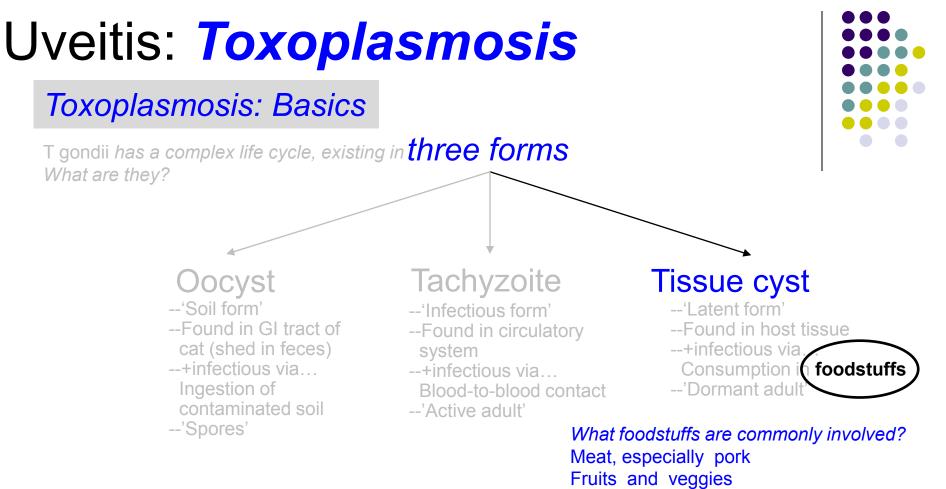
How does toxo get into the animals and/or onto the fruits and veggies? It gets into animals when they eat feed that has been pooped on by infected cats (ie, that contains oocysts)

Goat's milk



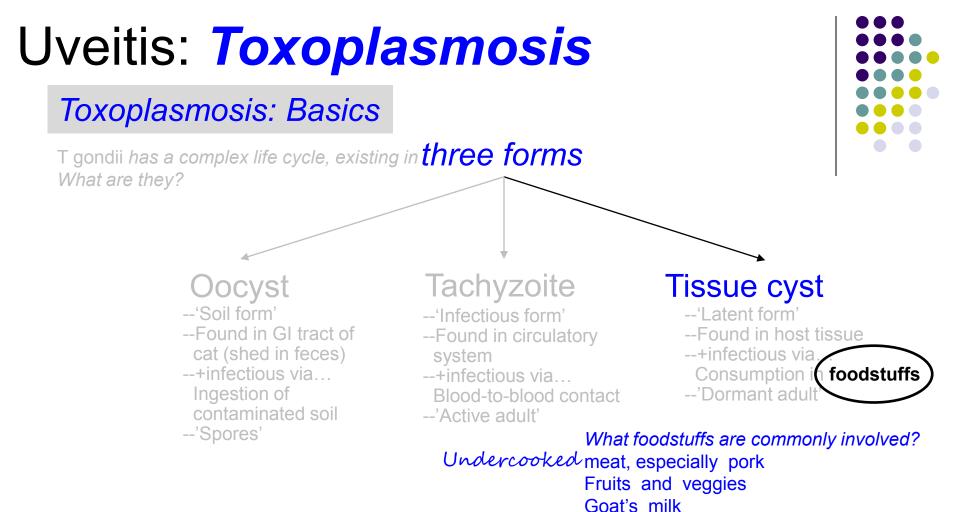
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How is toxo able to get into humans from the animals?



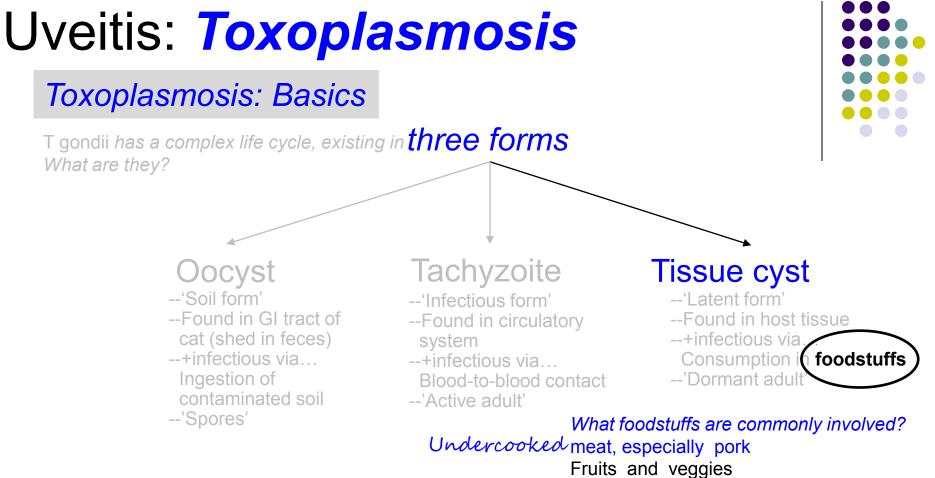
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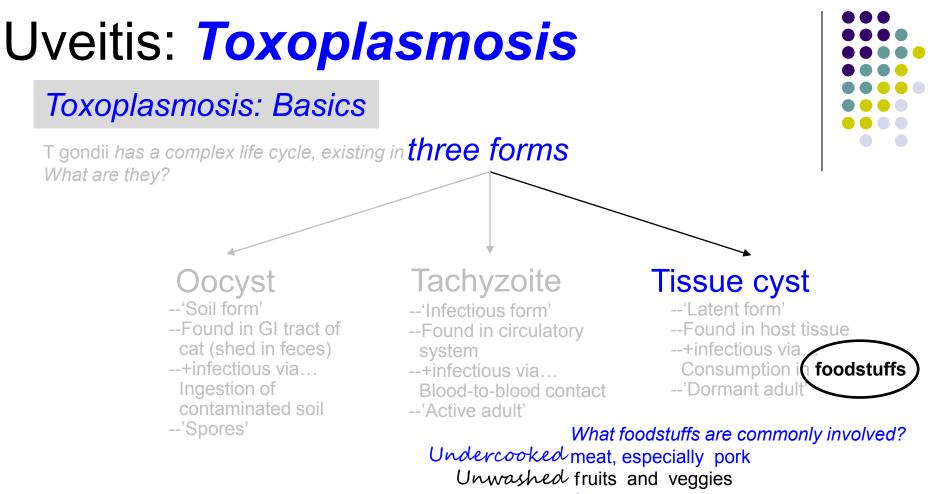
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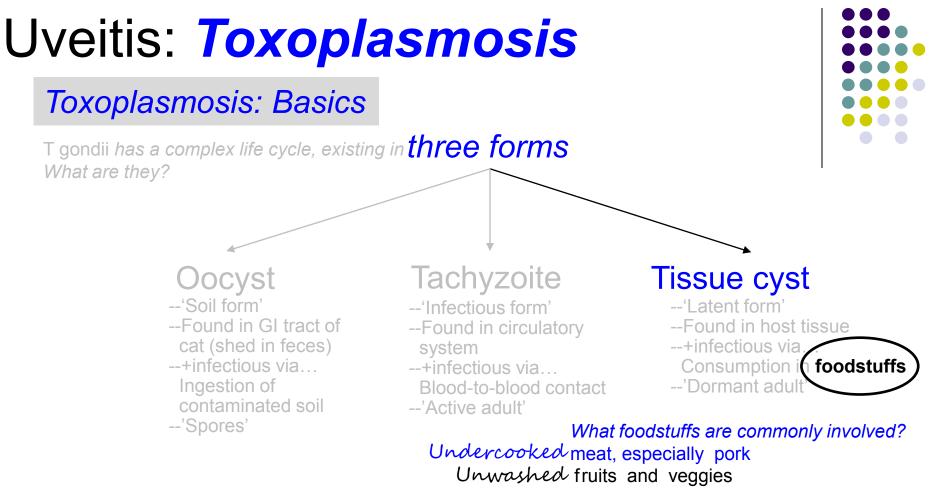
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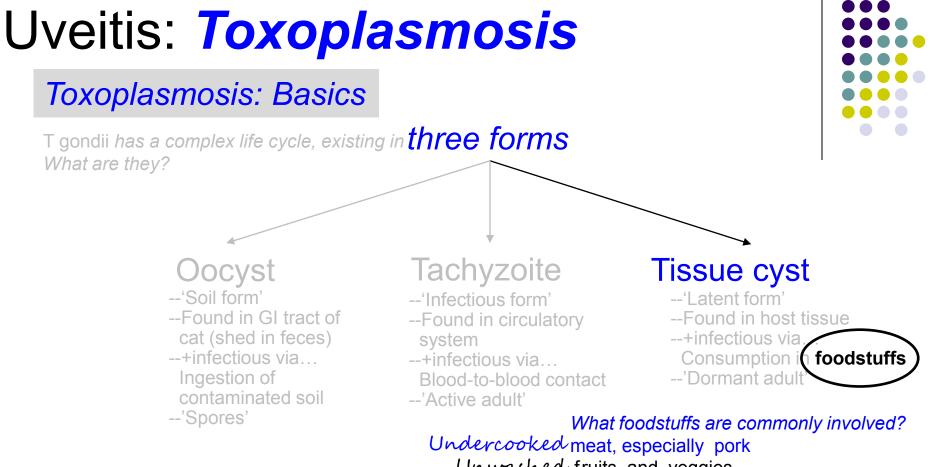
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How does toxo get into the animals and/or onto the fruits and veggies? It gets into animals when they eat feed that has been pooped on by infected cats (ie, that contains oocysts)

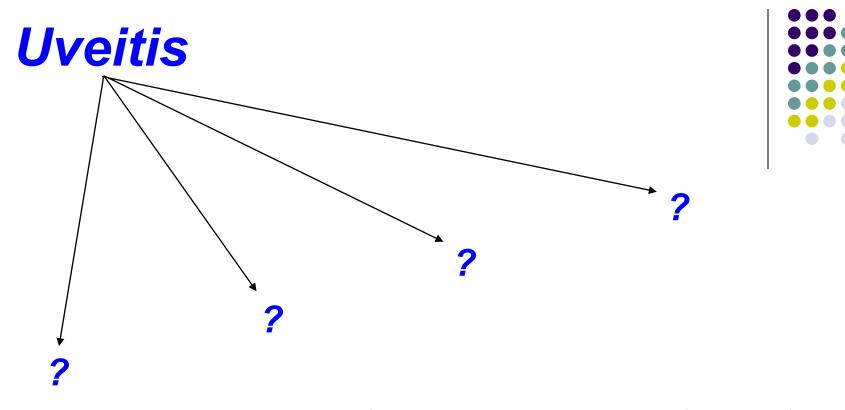
How is toxo able to get into humans from the animals? From fruits/veggies? From goat's milk? It gets into them when they eat meat that is undercooked...eat fruits/veggies that are unwashed... or drink goat's milk that is



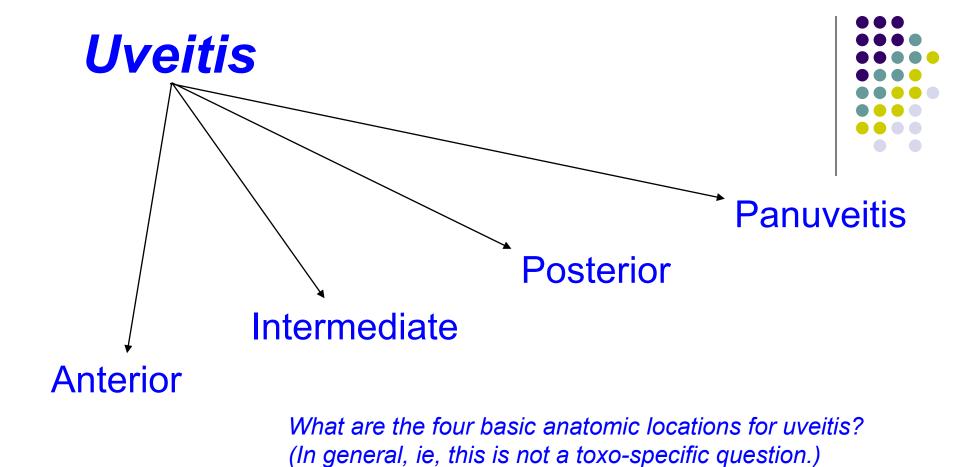
Unwashed fruits and veggies Unpasteurized goat's milk

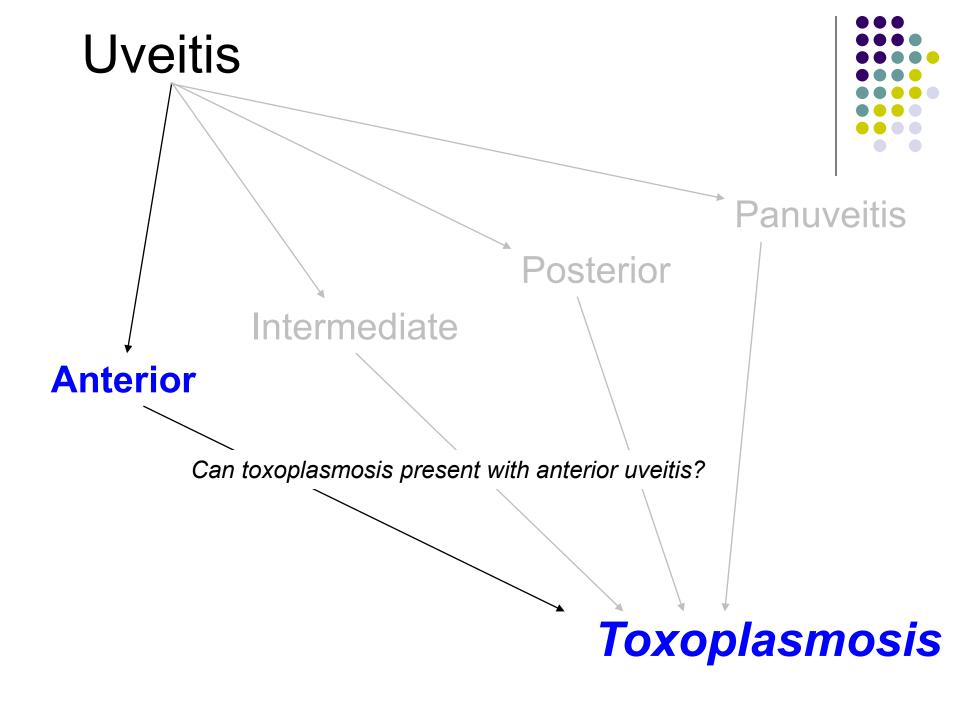
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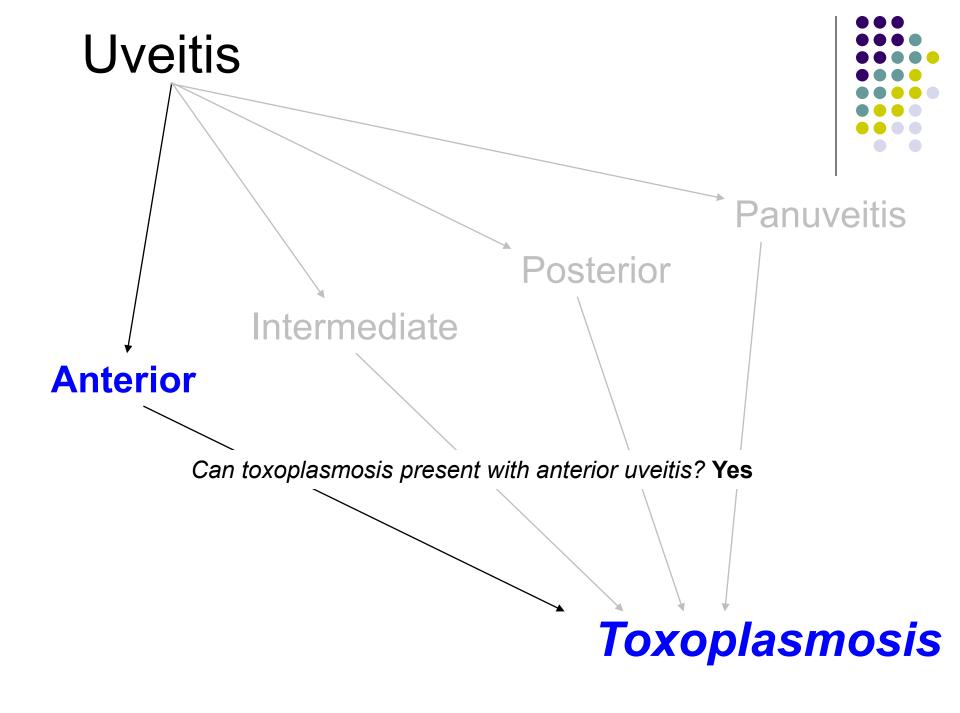
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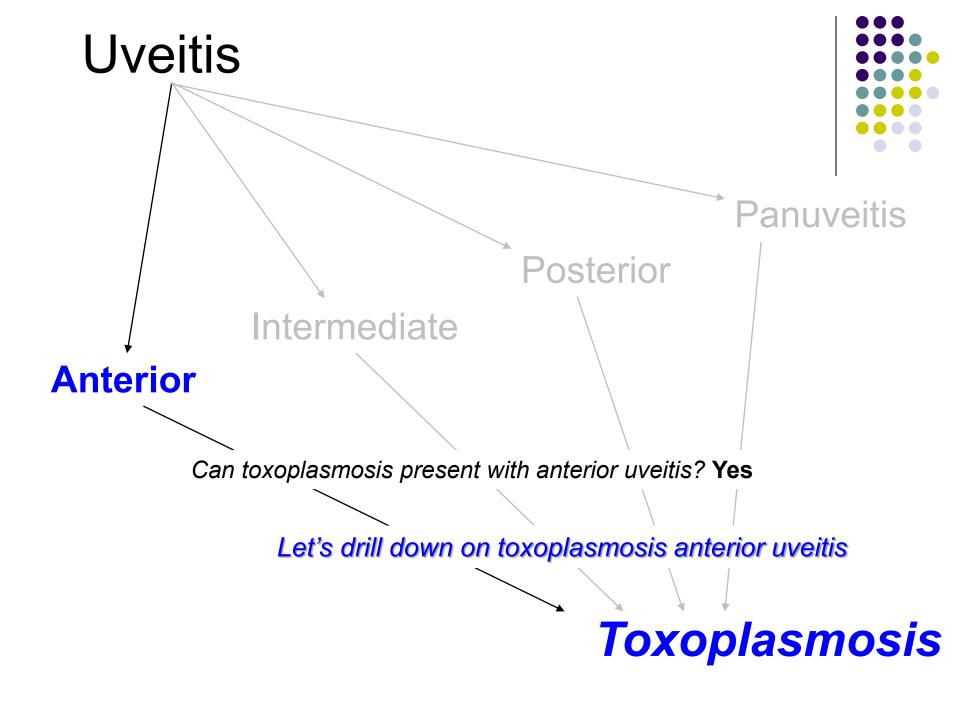


What are the four basic anatomic locations for uveitis? (In general, ie, this is not a toxo-specific question.)



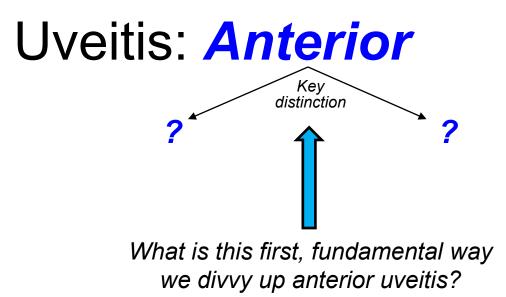




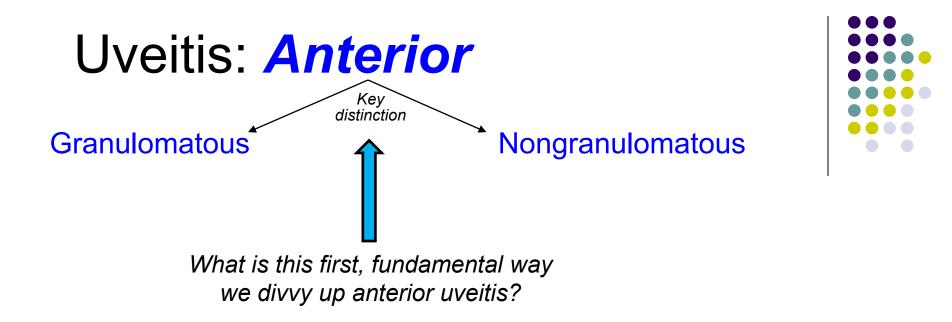


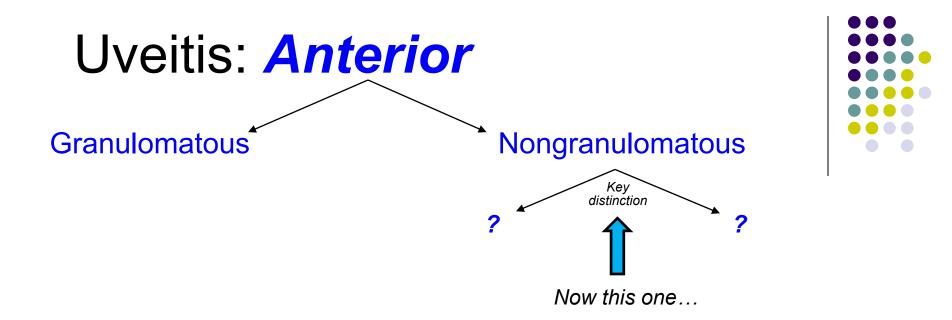
Uveitis: Anterior

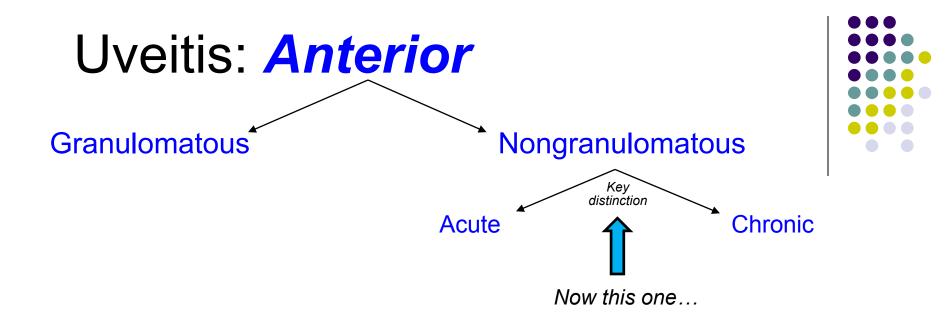


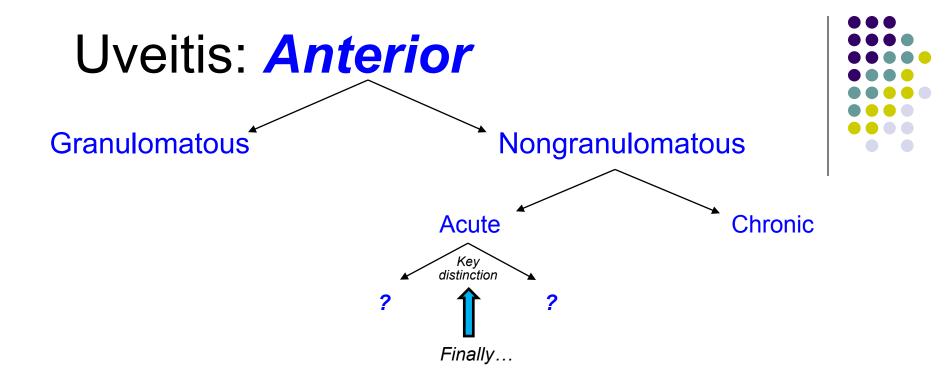


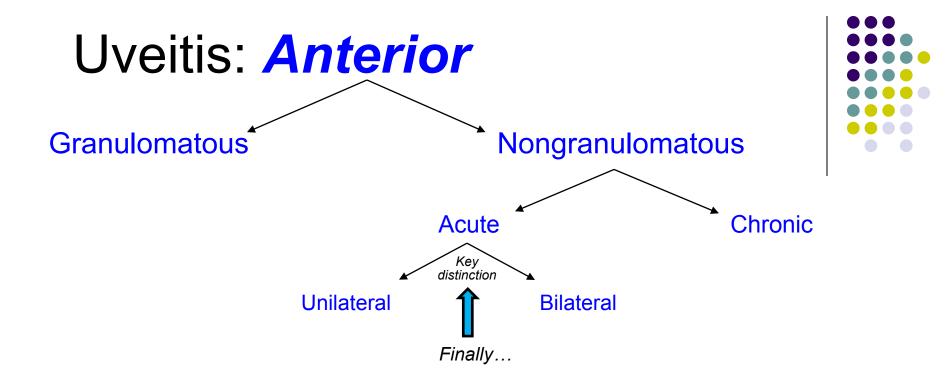


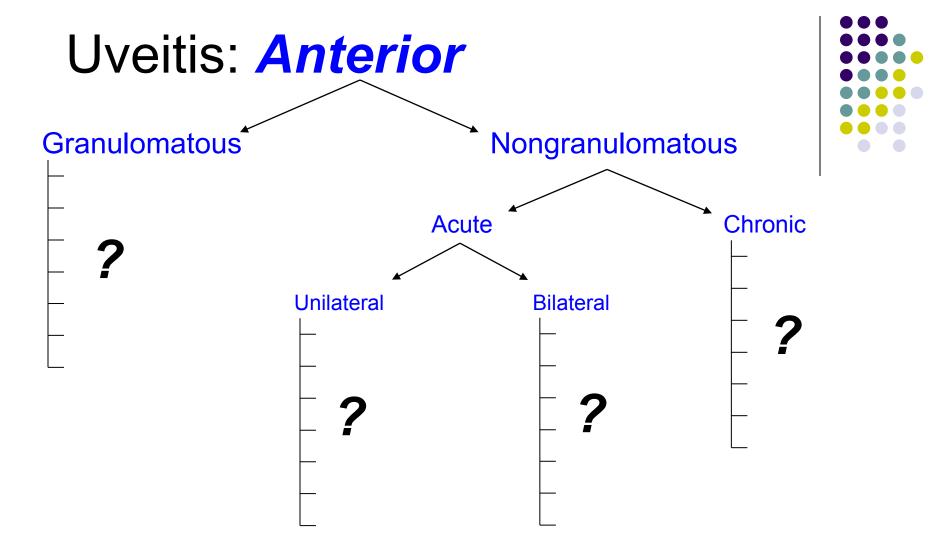




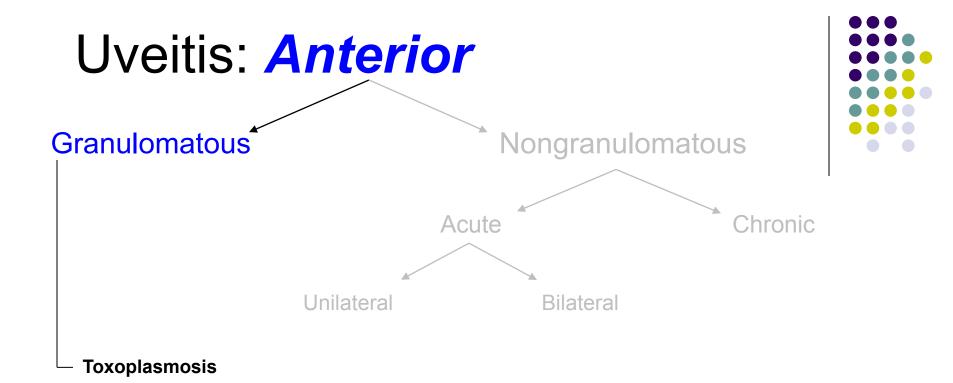




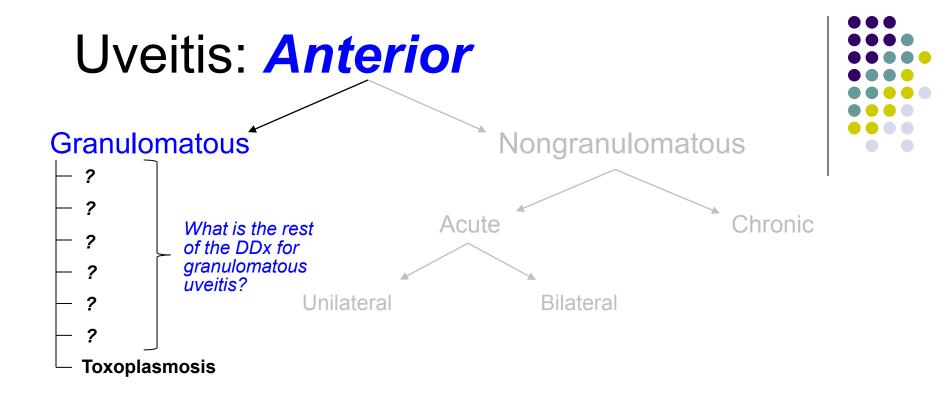




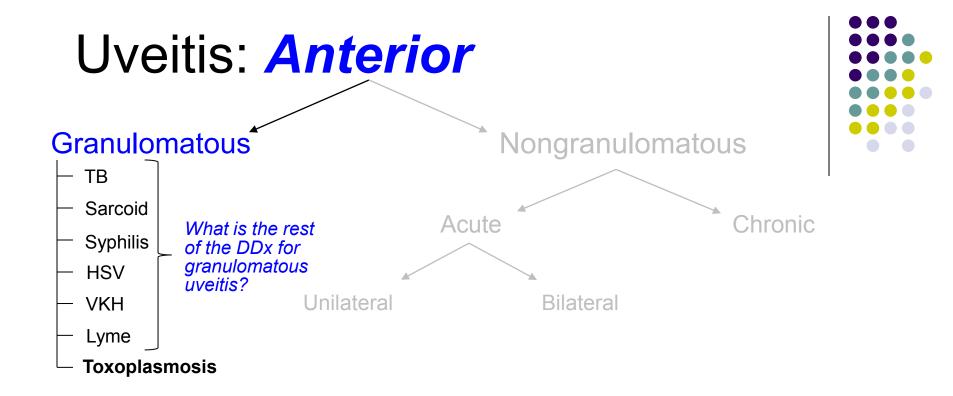
Now that the taxonomy is laid out: *Where does toxoplasmosis reside?*



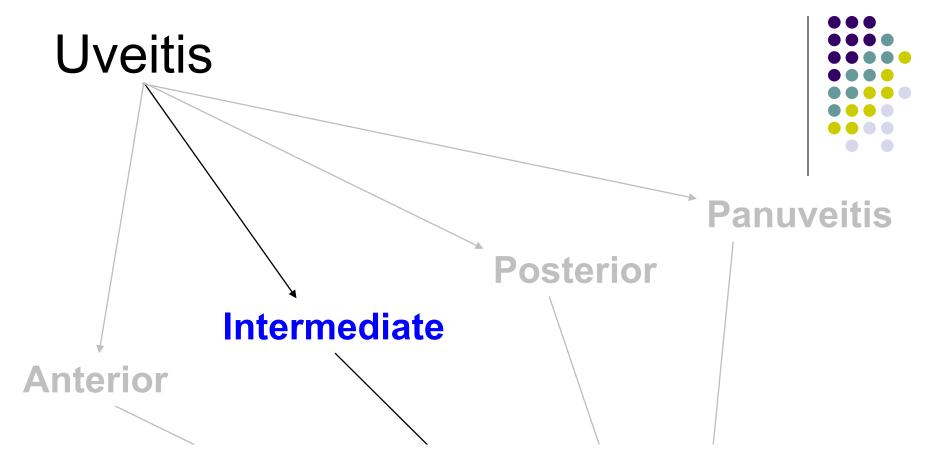
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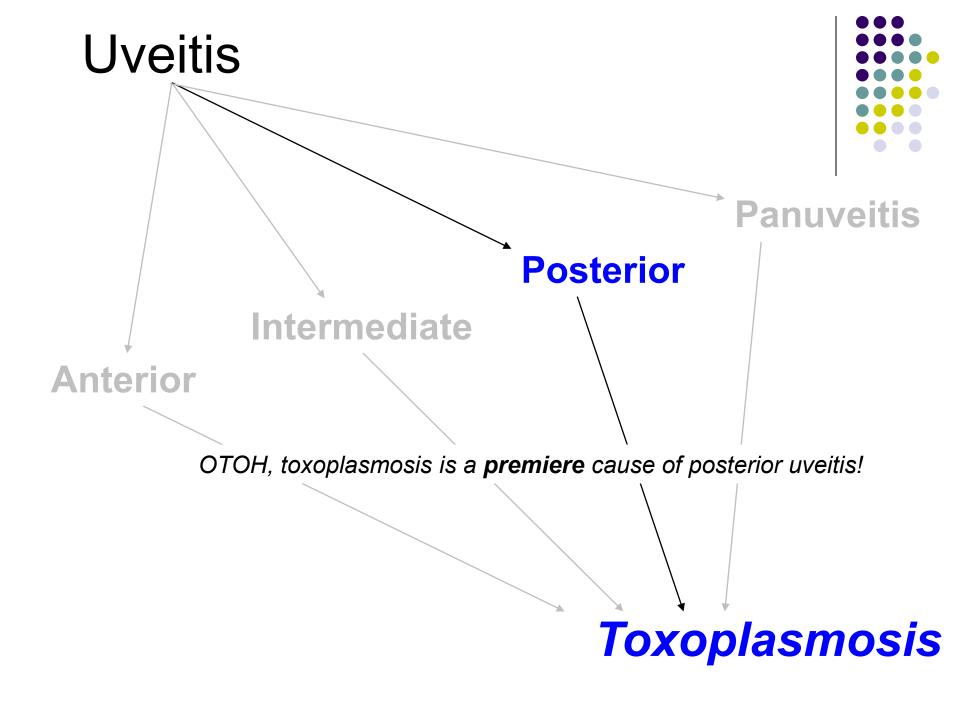


Now that the taxonomy is laid out: *Where does toxoplasmosis reside?* Among the granulomatous uveitides



The BCSC Uveitis book does not list toxoplasmosis in the DDx for intermediate uveitis

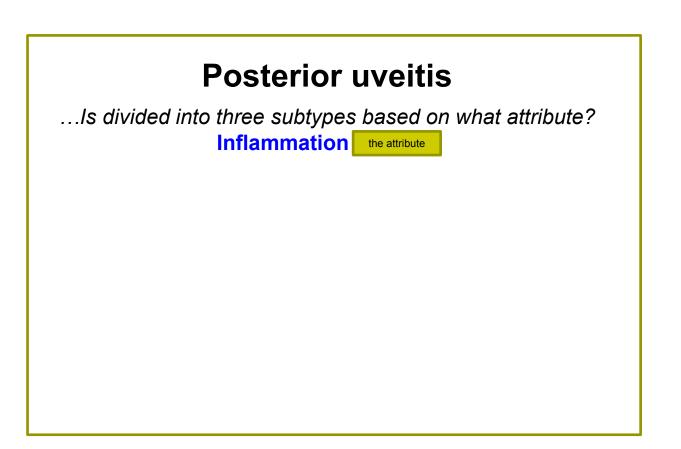
Toxoplasmosis





Posterior uveitis

... Is divided into three subtypes based on what attribute?

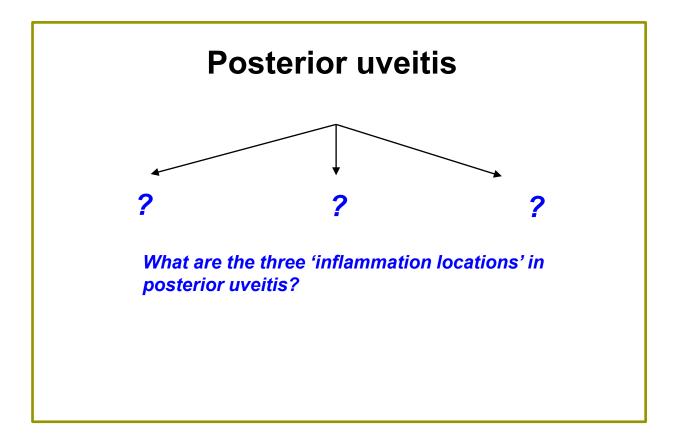




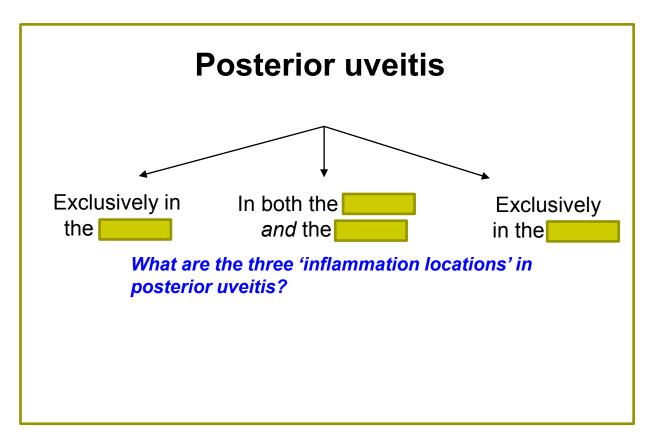


Posterior uveitis

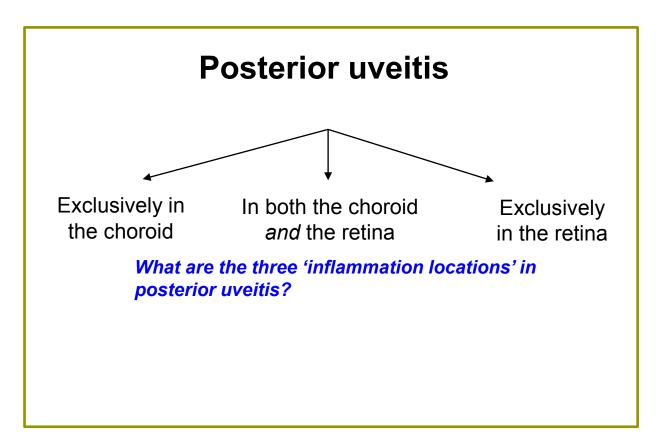
...Is divided into three subtypes based on what attribute? Inflammation location



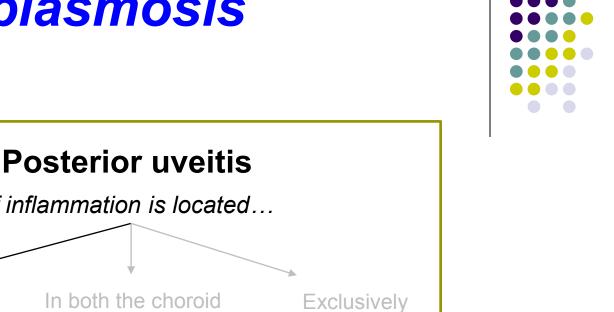


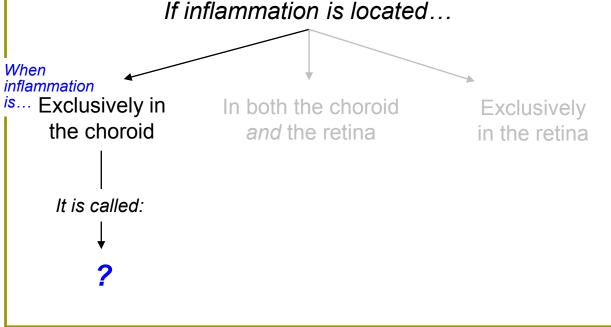


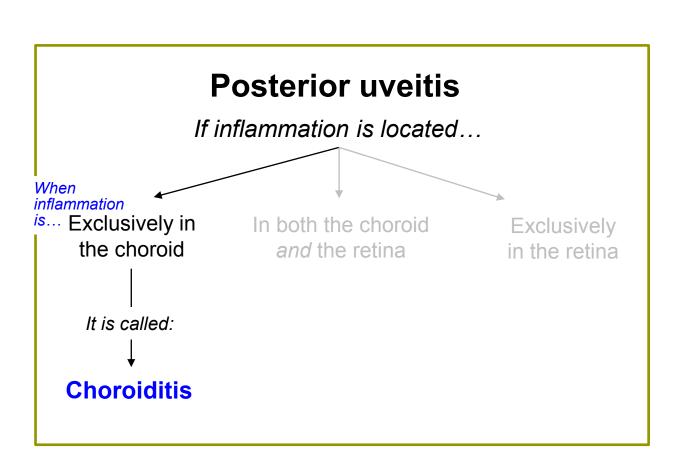






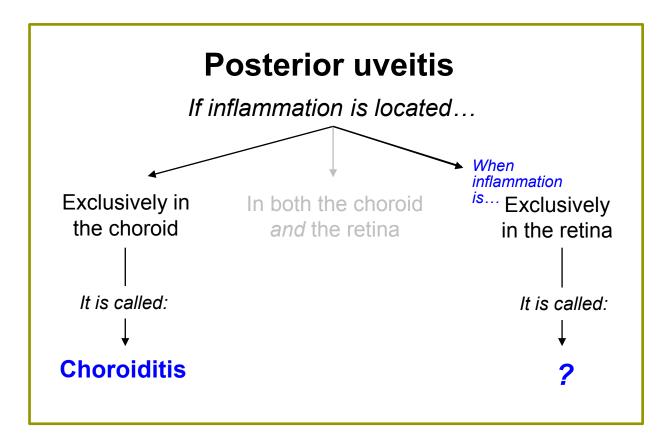




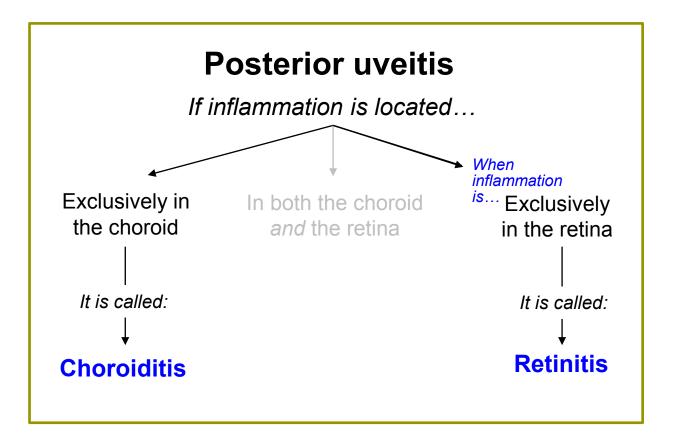




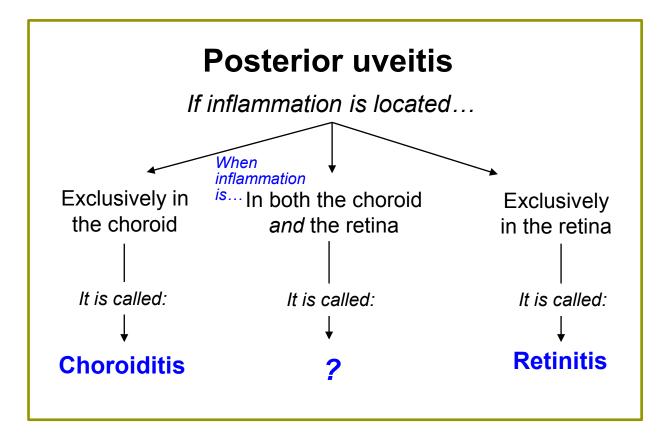




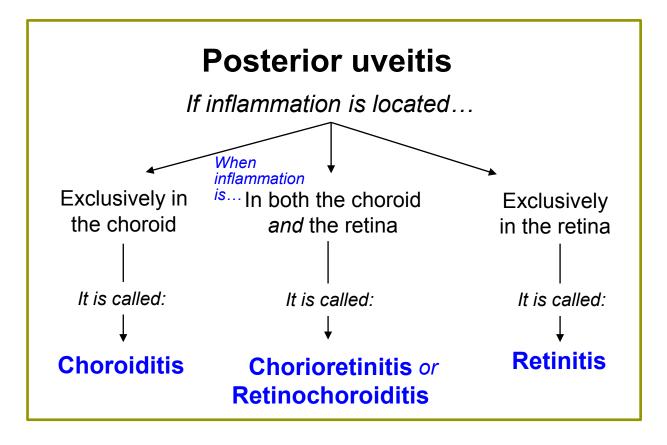


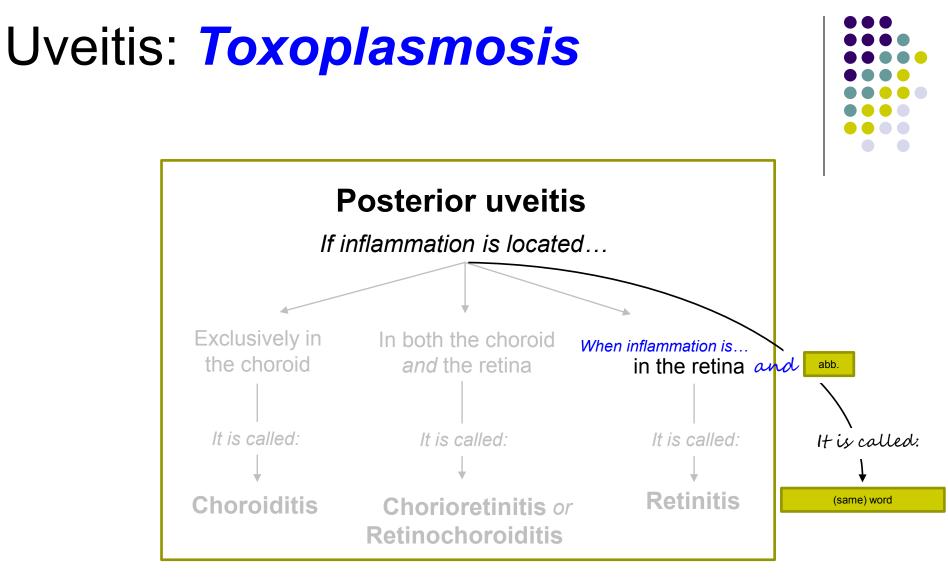




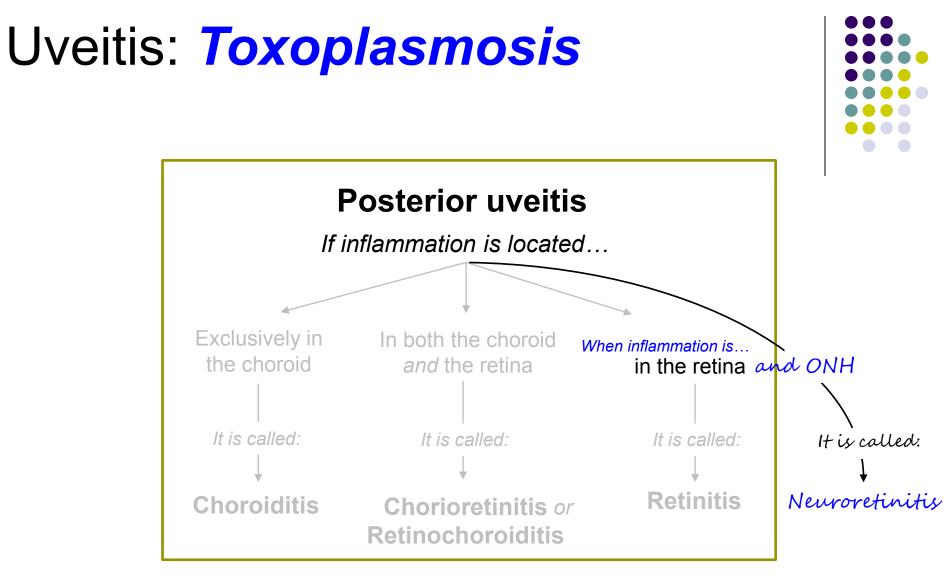






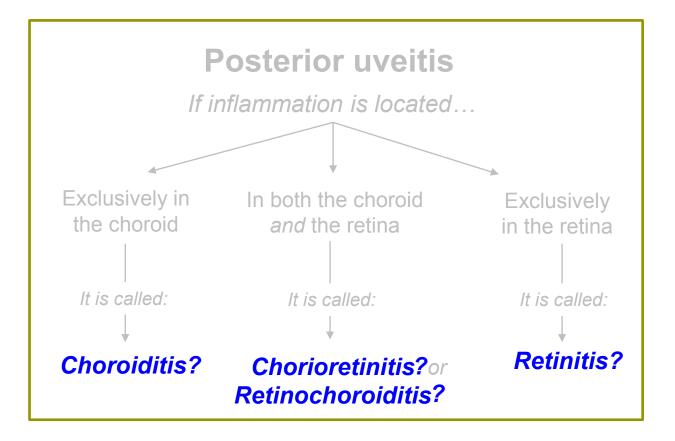


Note: If you took exception to the suggestion that there are only 3 posterior uveitis locations by pointing out the existence of ______, you're not wrong



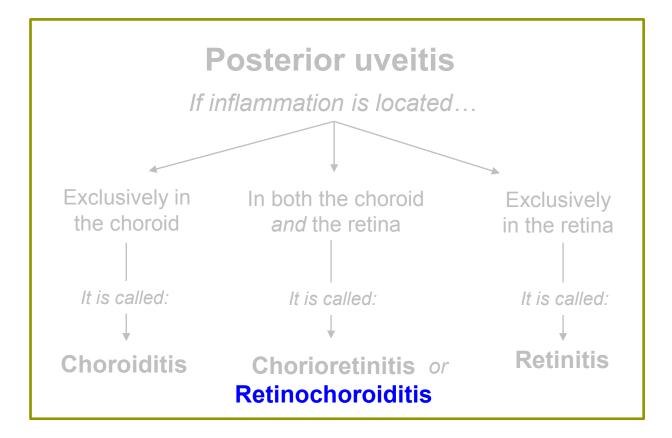
Note: If you took exception to the suggestion that there are only 3 posterior uveitis locations by pointing out the existence of *neuroretinitis*, you're not wrong





What is the classic posterior manifestation of toxoplasmosis?





What is the classic posterior manifestation of toxoplasmosis? **Retinochoroiditis**

Toxoplasmosis: Retinochoroiditis

What is the classic appearance of an *inactive* toxoplasmosis lesion?



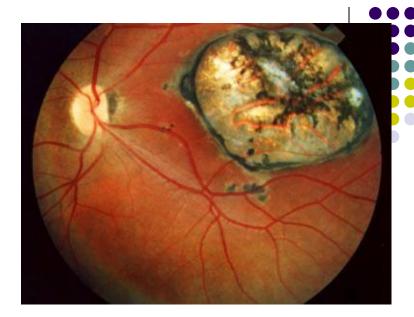
Toxoplasmosis: Retinochoroiditis

What is the classic appearance of an inactive toxoplasmosis lesion? A pigmented chorioretinal scar











Toxoplasmosis: Inactive scar

Toxoplasmosis: Retinochoroiditis

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What is the classic appearance of an active toxoplasmosis lesion?



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Toxoplasmosis: Retinochoroiditis

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What is the classic appearance of an **active** toxoplasmosis lesion? A white lesion



Toxoplasmosis: Retinochoroiditis

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What is the classic appearance of an active toxoplasmosis lesion?

A white lesion adjacent to a structure



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Toxoplasmosis: Retinochoroiditis

What is the classic appearance of an inactive toxoplasmosis lesion? A pigmented chorioretinal scar

What is the classic appearance of an **active** toxoplasmosis lesion? A white lesion adjacent to a scar, with overlying two words



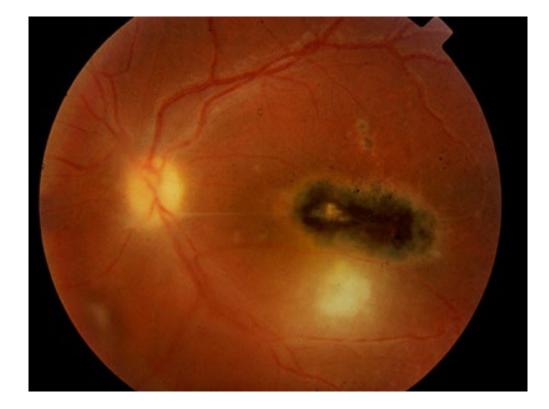
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Recurrent ocular toxoplasmosis. Note the active retinal lesion associated with an old inactive scar

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The fact that active lesions are usually adjacent to an old scar indicates what about their origin?



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Newly acquired toxo retinitis (note the absence of an adjacent scar)



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Active toxoplasmosis: Headlight in the fog



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In terms of the Headlight in the fog appearance... The headlight = ? The fog =



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Diagnosis

How is the diagnosis of ocular toxoplasmosis made?



Diagnosis

How is the diagnosis of ocular toxoplasmosis made? In most cases, clinically



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Is serology testing helpful?



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Is serology testing helpful? It can be. As with most infectious conditions, toxo acquisition produces a transient (<1 yr) ^{19?} response, and a lifelong ^{19?} response. Thus, a positive ^{19?} result confirms a recent infection.

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Treatment

What are the indications for treating active ocular toxoplasmosis?



Treatment

---? --? --? --?

What are the indications for treating active ocular toxoplasmosis?

This is controversial. Some physicians tx all lesions, whereas other elect to watch small peripheral ones. That said, the following clinical scenarios are considered relative indications to tx:

Treatment

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exam finding (two words)



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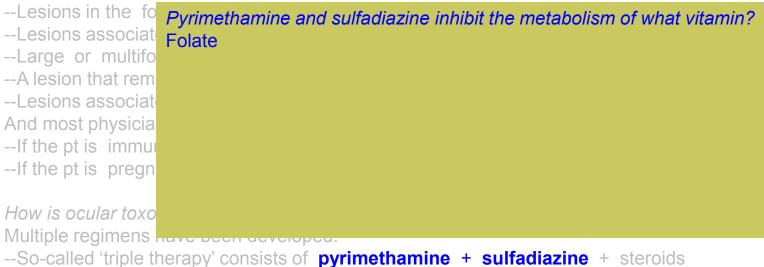
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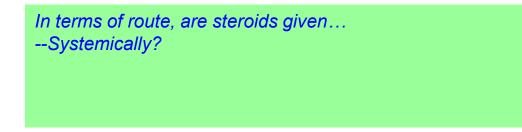
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+/- pyrimethamine is effective as well



Treatment

What are the indications for treating active ocular toxoplasmosis?

This is controversial. Some physicians tx all lesions, whereas other elect to watch small peripheral ones.

That said, the following clinical scenarios are considered relative indications to tx:

- --Lesions in the fovea or threatening the ONH
- --Lesions associated with decreased VA
- --Large or multifocal lesions
- --A lesion that remains active for >1 month
- --Lesions associated with significant vitreous inflammation
- And most physicians consider the following clinical scenarios **absolute** indications to tx:
- -- If the pt is immunocompromised
- --If the pt is pregnant , and has newly-acquired dz

How is ocular toxo treated?

Multiple regimens have been developed:

- --So-called 'triple therapy' consists of pyrimethamine + sulfadiazine + steroids
- --'Quadruple therapy' is triple therapy + clindamycin
- --TMP-SMX + steroids is preferred by many for its simplicity, availability and low price
- --Azythromycin +/- pyrimethamine is effective as well



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Which is the most effective tx regimen for ocular toxoplasmosis?



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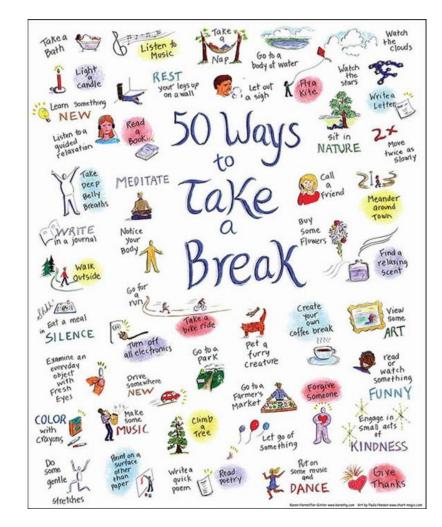
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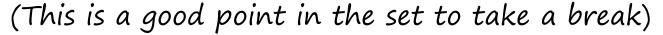
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Which is the most effective tx regimen for ocular toxoplasmosis?

None of the above. That is, while all are employed, <u>none has been shown via clinical trial to be</u> <u>definitively the best</u>.









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How might a pregnant woman become primarily infected? By any of the pathways described earlier—by consuming foodstuffs containing viable tissue cysts, consuming oocysts, or (very rarely) by blood-to-blood transmission of tachyzoites



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1) Don't consume two words or three words
2) ?



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What is the classic ocular manifestation of congenital toxoplasmosis?



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Toxoplasmosis: Congenital



Where in the retina are congenital lesions usually found?



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Where in the retina are congenital lesions usually found? The macula



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Does congenital toxo present unilaterally, or bilaterally?



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Does congenital toxo present unilaterally, or bilaterally? Bilaterally in the majority of cases



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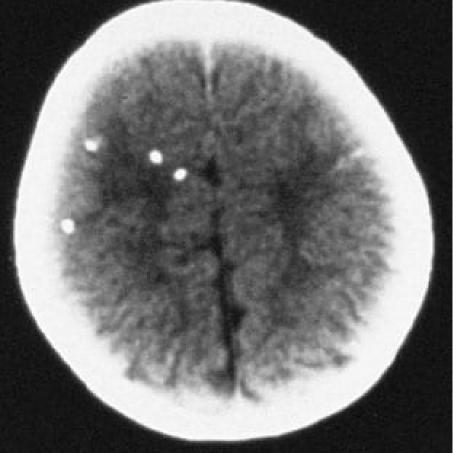
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Hydrocephalus

Intracranial calcifications

Congenital toxoplasmosis

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Unfortunately not. Almost all of these 'unaffected' infants will experience a retinochoroiditis event in one or both eyes at some point in childhood, and up to 25% will be blinded by it.



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Note: This is per the *Uveitis* book; the *Peds* book states "Ocular toxo does not require treatment unless it threatens vision." Caveat emptor.

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Toxoplasmosis is not the only infectious agent that presents in this manner when acquired in utero. What is the well-known mnemonic for the infectious agents?

- --
- ___
- __
- -
- -
- --
- --



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- --Toxoplasmosis (duh)
- --Other
- --Rubella
- --CMV
- --Herpesviruses, including
- --EBV
- --Syphilis



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The *Peds* book uses the mnemonic **TORCH**:

- --TOxoplasmosis
- --Rubella
- $--\mathbf{C}MV$
- --Herpeseviruses



In HIV+ pts

Is toxoplasmosis a common opportunistic infection in HIV/AIDS pts?



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In what key way might the presentation of toxo retinitis in an HIV+ pt deviate from the classic **headlight in the fog** *description?*



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In what key way might the presentation of toxo retinitis in an HIV+ pt deviate from the classic headlight in the fog description? By the absence or attenuation of the 'fog' aspect—a pt who is profoundly immunocompromised might not be able to generate a significant vitritis





Toxoplasmosis in AIDS. Note how clear the pic is, ie, there's no 'fog.' This is due to an absence of the normal vitritis.



In HIV+ pts



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In what key way might the presentation of toxo retinitis in an HIV+ pt deviate from the classic headlight in the fog description? By the absence or attenuation of the 'fog' aspect—a pt who is profoundly immunocompromised might not be able to generate a significant vitritis

Toxo retinochoroiditis presentation in HIV/AIDS pts differs in several other respects as well. What are they?

--? --?

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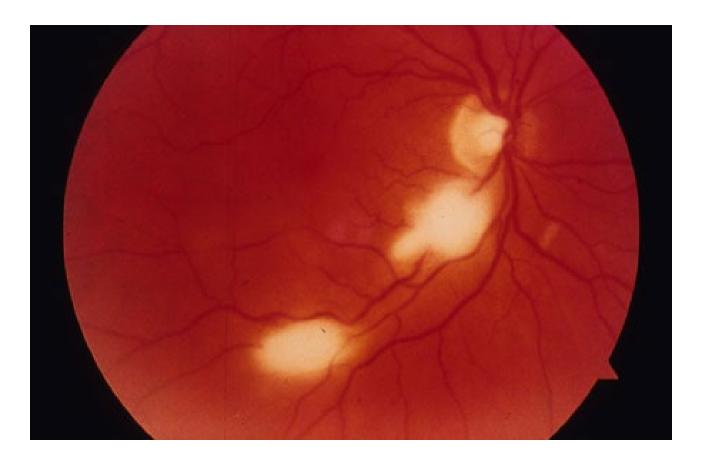
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- --The lesion(s) tend to be s/w larger than in immunocompetent pts
- --The lesion is much more likely to be de novo, ie, to not be associated with an old CR scar





Same pic of toxoplasmosis in AIDS. This time, note 1) the multifocality, and 2) the absence of a CR scar.



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Why must all toxo retinochoroiditis be treated in HIV/AIDS pts?



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Why must all toxo retinochoroiditis be treated in HIV/AIDS pts? Because unlike in immunocompetent pts, dz in HIV/AIDS pts does **not** resolve spontaneously—instead, it is relentlessly progressive



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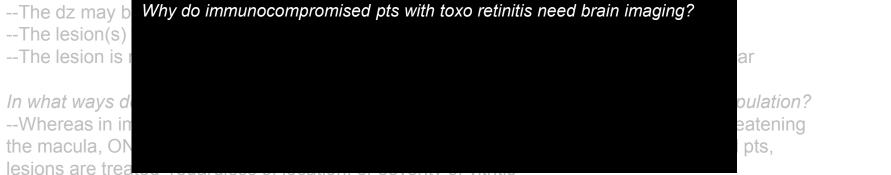
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In HIV+ pts

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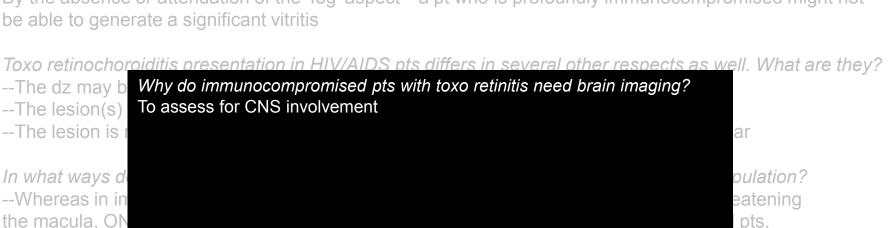
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Yes—up to %

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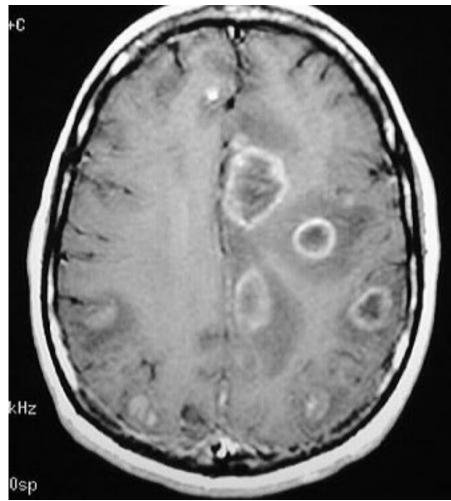
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CNS toxo: Ring-enhancing lesions



The following slides comprised a previous version of the toxo review slide-set. This previous version is, frankly, not as good as the one you just completed, and there's no new info of significance in it. That being said, it might be worth your time to go through it. (Or not—your call.)













In the US, what is the prevalence for toxoplasmosis infection?





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Is ocular toxoplasmosis known to present with anterior segment signs?





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Which four uveitides are associated with stellate KP? --? --? --? --Toxoplasmosis





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- Which four uveitides are associated with stellate KP?
- --HSV
- --VZV
- --Toxoplasmosis





- Where does toxoplasmosis rank as a cause of posterior segment infection? #1
- Classic description of the posterior pole exam in toxoplasmosis:





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In terms of the Headlight in the fog *appearance*... The headlight = The fog =





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In terms of the Headlight in the fog *appearance*... The headlight = the white toxo lesion The fog = the **dense overlying vitritis**





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- Classic description of the posterior pole exam in toxoplasmosis: 'Headlight in the fog'
- The toxo organism is an

obligate vs facultative





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intra- vs extracellular





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bug-host relationship





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class of bug





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What is the full name of the organism?





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What animal is the definitive host? The cat





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It had long been thought that virtually all cases of ocular toxo represented reactivation of congenital disease. However, recent research indicates a sizeable proportion of cases are acquired post-natally, with many experts now convinced that the **majority** of cases are acquired in this fashion.



exam

- Where does toxoplasmosis rank as a cause of posterwhat is the typical DFE finding in congenital toxoplasmosis?
- Classic in toxor
- The tox parasiti
- Can represent re-activation of congenital disease, or newly-acquired infection





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- -- two words ; or in
- -- three words ; or in
- -- three words





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- --Undercooked meat; or in
- --contaminated fruits/veggies; or in

milk

--unpasteurized





314

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 - fraction of AIDS patients with ocular toxo will have
 Inside Inside





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- 1/2 of AIDS patients with ocular toxo will have CNS lesions--they must undergo MRI (classic finding—ring-enhancing lesions)



- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...

•	structure/area 1
•	structure/area 2
•	structure/area 3





- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...
 - macula
 - ONH
 - major retinal vessels





- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...
 - macula
 - ONH
 - major retinal vessels
 - ...or in cases of severe





- Treatment of ocular toxoplasmosis:
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- Treatment of ocular toxoplasmosis:
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What is the natural course of untreated ocular toxoplasmosis?

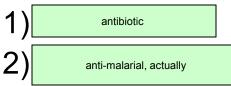


- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...
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 - major retinal vessels
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What is the natural course of **untreated** ocular toxoplasmosis? It is a self-limited disease in the immunocompetent--lesions will begin healing within a month or two



- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...
 - macula
 - ONH
 - major retinal vessels
 - ...or in cases of severe vitritis
 - Treat with



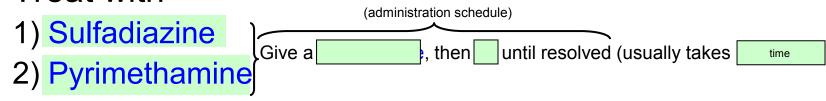


- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...
 - macula
 - ONH
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 - ...or in cases of severe vitritis
 - Treat with
 - 1) Sulfadiazine
 - 2) Pyrimethamine





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1) Sulfadiazine

2) Pyrimethamine

(administration schedule)

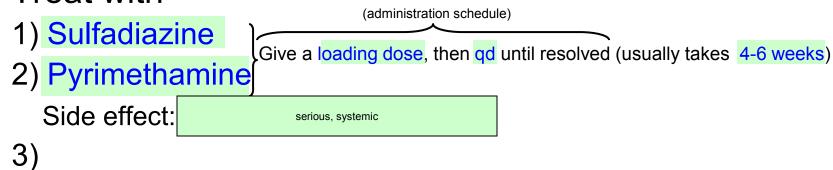
Give a loading dose, then qd until resolved (usually takes 4-6 weeks)







- Treat active infection if threatening the...
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 - ONH
 - major retinal vessels
- ...or in cases of severe vitritis
- Treat with





- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...
 - macula
 - ONH
 - major retinal vessels
 - ... or in cases of severe vitritis
 - Treat with
 - 1) Sulfadiazine
 - Give a loading dose, then qd until resolved (usually takes 4-6 weeks) 2) Pyrimethamine

(administration schedule)

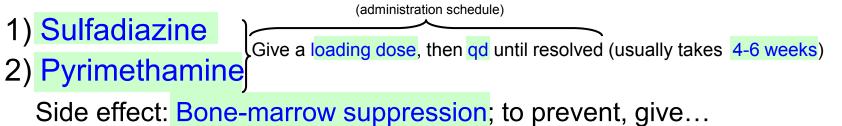
Side effect: Bone-marrow suppression

3)





- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...
 - macula
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3) two words

- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...
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 - Treat with
 - 1) Sulfadiazine
 - 2) Pyrimethamine

Give a loading dose, then qd until resolved (usually takes 4-6 weeks)

Side effect: Bone-marrow suppression; to prevent, give...

(administration schedule)

3) Folinic acid



- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...
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3)

Not a typo!

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- 2) Pyrimethamine

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With respect to treating ocular toxoplasmosis, this combo of meds is known as the classic...what?

(administration schedule)



- Treatment of ocular toxoplasmosis:
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 - 1) Sulfadiazine
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Side effect: Bone-ma

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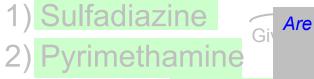
With respect to treating ocular toxoplasmosis, this combo of meds is known as the classic...what? 'Triple therapy'

(administration schedule)



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(administration schedule)



Side effect: Bone-m

3) Folinic acid

3) Prednisone

Are alternative therapies available?

A/Q

- Treatment of ocular toxoplasmosis:
 - Treat active infection if threatening the...

--?

- macula
- ONH
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(administration schedule) Are alternative therapies available?

1) Sulfadiazine Gi 2) Pyrimethamine

> --? Side effect: Bone-ma --?

3) Folinic acid 3) Prednisone

Yes, the following have been found to be efficacious alternatives:



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Side effect: Bone-ma

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(administration schedule)

- Are alternative therapies available?
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- --Trimethoprim-sulfamethoxazole
- --Azithromycin --Clindamycin



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Q



Does treatment eradicate the infection? No

Why not? Is the toxo bug not susceptible?



Sulfadiazine
 Pyrimethamine

Side effect: Bone-ma

3) Folinic acid3) Prednisone

(administration schedule)

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--Clindamycin

Giv





Why not? Is the toxo bug not susceptible?

The active bug (the *tachyzoite*) is quite susceptible. However, the bradyzoite form (aka *tissue cyst*) is impervious to the anti-infectives; thus, the infection is incurable.

Treat with

Sulfadiazine
 Pyrimethamine

Side effect: Bone-ma

3) Folinic acid3) Prednisone

(administration schedule)

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Does treatment prevent recurrences?

Treat with

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Side effect: Bone-ma

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(administration schedule)

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What is the purpose of anti-infective treatment, then?

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Side effect: Bone-ma

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What is the purpose of anti-infective treatment, then?

Practically speaking, the purpose of treatment is to allow for the administration of steroids (to reduce inflammation) without fear of exacerbating the infection itself

Treat with

- (administration schedule)
- Are alternative therapies available?
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Side effect: Bone-m

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(administration schedule)

Are alternative theranies available?

The triple therapy includes prednisone. Is it appropriate to use peri- or intraocular steroids instead?

Side effect: Bone-m

- 3) Folinic acid
- 3) Prednisone

--Clindamycin

Do HIV/AIDS pts require long-term suppressive therapy? Yes es:





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• Treat with

(administration schedule)

Are alternative theranies available?

The triple therapy includes prednisone. Is it appropriate to use peri- or intraocular steroids instead? No! Peri/intraocular steroids can result in inflammation so severe as to cause loss of the eye!

Side effect: Bone-mail: --Azithromycin 3) Folinic acid 3) Prednisone --Clindamycin Do HIV/AIDS pts require long-term suppressive therapy? Yes es:





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The active bug (the *tachyzoite*) is quite susceptible. However, the bradyzoite form (aka *tissue cyst*) is impervious to the anti-infectives; thus, the infection is incurable.

Does treatment preven recurrences No

When recurrence occurs, what relationship usually holds between the new lesion and the original one?

Practically speaking, the purpose of treatment is to allow for the administration of steroids (to reduce inflammation) without fear of exacerbating the infection itself

Treat with

1) Sulfadiazine

3) Folinic acid

3) Prednisone

2) Pyrimethamine

(administration schedule)

- Are alternative therapies available?
- Giv Yes, the following have been found to be efficacious alternatives: --Trimethoprim-sulfamethoxazole
- --Azithromycin Side effect: Bone-m
 - --Clindamycin
 - Do HIV/AIDS pts require long-term suppressive therapy? Yes





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Does treatment preven recurrences No

When recurrence occurs, what relationship usually holds between the new lesion and the original one? The recurrence will occur at the border of the original lesion (which is now a scar)

Practically speaking, the purpose of treatment is to allow for the administration of steroids (to reduce inflammation) without fear of exacerbating the infection itself

Treat with

1) Sulfadiazine

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Side effect: Bone-m

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