All of the following are associated with follicular conjunctivitis except (or are they all associated with it?):

- *Molluscum* lesions of the eyelid (not conj)
- Adult inclusion conjunctivitis
- Trachoma
- Parinaud’s oculoglandular syndrome
- Toxic response to topical meds
- *Moraxella* conjunctivitis
- Epidemic keratoconjunctivitis (EKC)
- Pharyngoconjunctival fever (PCF)
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause--any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.
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If a pt presents with chronic (>3 weeks) follicular conjunctivitis, consider these three causes, in this order:
1. Molluscum—inspect the lids carefully for lesions. If none are found…
2. Toxins—press the pt to recall any history of topical drop use. If no drops…
3. It’s Chlamydia until proven otherwise.
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Q/A
All are associated with it. Follicular conjunctivitis should make you think of 3 things: **viruses**, **Chlamydia** (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

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**Parinaud’s oculoglandular syndrome**, secondary to **Bartonella** infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is **Moraxella** conjunctivitis, aka **Axenfeld conjunctivitis**—another bacteria associated with follicles.

What non-ocular exam finding is common to all four of these as causes of conjunctivitis?
All are associated with **pre-auricular lymphadenopathy**.
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**Where does trachoma rank as a cause of blindness?**

It is the #1 cause of preventable/infectious blindness worldwide. Patients from the Middle East are most likely to be affected. Native Americans is the ethnic group in the US most likely to be affected. Does trachoma result from a single infectious episode? No, recurrent infections are required. When limbal follicles scar, what exam finding results? Herbert's pits. When upper-lid tarsal follicles scar, what exam finding results? Arlt's line. What corneal finding is associated with trachoma? Superior pannus.
Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Chronic follicular conjunctivitis can be caused by molluscum lid lesions. Extensive molluscum disease is associated with HIV infection; consider testing. EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A, B, C — trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D–K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis — another bacteria associated with follicles.

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Where does trachoma rank as a cause of blindness?
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Patients from what part of the world are most likely to be affected?

Patients from the **Middle East** are most likely to be affected.

Native Americans are the **most likely to be affected** in the US.

Does trachoma result from a single infectious episode?
No, recurrent infections are required.

When limbal follicles scar, what exam finding results?
Herbert’s pits

When upper-lid tarsal follicles scar, what exam finding results?
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What corneal finding is associated with trachoma?
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Arlt’s line

**What corneal finding is associated with trachoma?**
Superior pannus
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Where does trachoma rank as a cause of blindness?
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What other, arguably more important exam finding results from scarring of the upper-lid follicles?

When upper-lid tarsal follicles scar, what exam finding results?
Arlt’s line and ?

What corneal finding is associated with trachoma?
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What other, arguably more important exam finding results from scarring of the upper-lid follicles?
Cicatricial entropion

When upper-lid tarsal follicles scar, what exam finding results?
Arlt’s line and cicatricial entropion

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Where does trachoma rank as a cause of blindness? It is the #1 cause of preventable/infectious blindness worldwide.

What other, arguably more important exam finding results from scarring of the upper-lid follicles? Cicatricial entropion.

Why is cicatricial entropion a more important finding that Arlt’s line?

When upper-lid tarsal follicles scar, what exam finding results? Arlt’s line and cicatricial entropion.

What corneal finding is associated with trachoma? Superior pannus.
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Where does trachoma rank as a cause of blindness? It is the #1 cause of preventable/infectious blindness worldwide.

What other, arguably more important exam finding results from scarring of the upper-lid follicles?
Cicatrical entropion

Why is cicatrical entropion a more important finding than Arlt’s line?
Because while an important diagnostic clue, Arlt’s line is otherwise of no clinical significance. In contrast, cicatrical entropion is the initial event in the process that leads to blindness.

When upper-lid tarsal follicles scar, what exam finding results?
Arlt’s line and cicatrical entropion

What corneal finding is associated with trachoma?
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Why is cicatricial entropion a more important finding than Arlt’s line? Because while an important diagnostic clue, Arlt’s line is otherwise of no clinical significance. In contrast, cicatricial entropion is the initial event in the process that leads to blindness.

What process is that?

When upper-lid tarsal follicles scar, what exam finding results? Arlt’s line and cicatricial entropion

What corneal finding is associated with trachoma? Superior pannus

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**What corneal finding is associated with trachoma?**
Superior pannus
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause--any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?) and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud's oculoglandular syndrome, secondary to Bartonella infection, is an exception to the 'bacteria don't cause follicles' rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis —another bacteria associated with follicles.
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How does the follicular reaction of trachoma differ from that of adult inclusion conjunctivitis? The follicles of trachoma are found predominantly on the superior conjunctiva (especially the tarsal conj), whereas in adult inclusion disease the follicles are usually confined to the inferior palpebral conj.
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How are trachoma and adult inclusion conjunctivitis treated?

It's important to remember that, whereas trachoma is an ocular condition, adult inclusion conjunctivitis is a systemic disease, and must be treated as such. (That said, trachoma is often treated with both systemic and topical antibiotics.)

What treatment regimens are used?

Azithromycin 1 gm PO x 1 dose is the most convenient. Regimens employing erythromycin, doxycycline or tetracycline are also used. In addition to the systemic abx, trachoma is treated with topical tetracycline.
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What must you consider if a child presents with adult inclusion conjunctivitis?
That the child is a victim of sexual abuse
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**Briefly, how would you describe POS?**
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Q/A

**Briefly, how would you describe POS?**

It is a nonocular finding in the **laterality** and **regions** NOT ‘follicular’ conjunctivitis associated with nonocular finding.
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Take note of this. The BCSC books describe the conjunctivitis in POS as granulomatous, not ‘follicular.’ (The POS entry on the Academy website EyeWiki calls POS a “granulomatous follicular conjunctivitis.”) Briefly, how would you describe POS? It is a unilateral granulomatous conjunctivitis associated with lymphadenopathy in the preauricular and submandibular regions, and patients should be asked about urogenital symptoms (concurrent GC is common).
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Is Bartonella the only bug that causes a POS?
No, a handful of others can as well:
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--Chlamydia trachomatis
--Francisella tularensis
--Syphilis
--TB
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F **tularensis** is the causative organism for what disease?

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F tularensis is the causative organism for what disease?
Tularemia

Is Bartonella the only bug that causes a POS?
No, a handful of others can as well:
--Chlamydia trachomatis
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All are associated with it. Follicular conjunctivitis should make you think of 3 things: **viruses**, **Chlamydia** (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause--any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for **trachoma** (serotypes A,B,C—*trachoma is as simple as ABC*) and **adult inclusion conjunctivitis** (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to **Bartonella** infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular manifestations of *B. henselae* infection include a follicular conjunctivitis, blepharoconjunctivitis, episcleritis, episcleritis, and adenitis of the eyelid. Parinaud’s oculoglandular syndrome is caused by *Bartonella henselae*.

F **tularensis** *is the causative organism for what disease?*

Tularemia

What event would clue you in that a pt might have tularemia?

Is Bartonella *the only bug that causes a POS*?

No, a handful of others can as well:

-- **Chlamydia trachomatis**

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F _tularensis_ is the causative organism for what disease?
**Tularemia**

What event would clue you in that a pt might have tularemia?
Direct contact with wild animals (eg, rabbits; raccoons)

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**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**

--?

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--?

**Hints forthcoming…**
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**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**

- ? (a near-ubiquitous component of all drops)
- ? (a class of med used in virtually all clinic visits, and most surgeries)
- ? (a class of antibiotic)
- ? (a class of med used in most clinic visits, and most surgeries)
- ? (a class of anti-infective)
- ? (a class of glaucoma med)
- ? (another class of glaucoma med)
- ? (a class of med used adjunctively in some surgical procedures)
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**

- **Preservatives**
  (a near-ubiquitous component of all drops)
- **Anesthetics**
  (a class of med used in virtually all clinic visits, and most surgeries)
- **Aminoglycosides**
  (a class of antibiotic)
- **Cycloplegics**
  (a class of med used in most clinic visits, and most surgeries)
- **Antivirals**
  (a class of anti-infective)
- **Miotics**
  (a class of glaucoma med)
- **α-agonists**
  (another class of glaucoma med)
- **Antineoplastic**
  (a class of med used adjunctively in some surgical procedures)
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A, B, C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—a bacteria associated with follicles.

Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:

---Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
--Miotics
--α-agonists
--Antineoplastic

Which preservative is most commonly implicated in toxic keratoconjunctivitis?
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for *trachoma* (serotypes A,B,C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**

---**Preservatives**

---Anesthetics
---Aminoglycosides
---Cycloplegics
---Antivirals
---Miotics
---α-agonists
---Antineoplastic

Which preservative is most commonly implicated in toxic keratoconjunctivitis?

BAK
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and *toxins*. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for *trachoma* (serotypes A, B, C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D–K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis* —another bacteria associated with follicles.

*Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:*

--Preservatives
--Anesthetics
--Aminoglycosides
--Cyclopentolate
--Antivirals
--Miotics
--α-agonists
--Antineoplastic

Which preservative is most commonly implicated in toxic keratoconjunctivitis?

BAK

What does BAK stand for?

Benzalkonium (chloride)
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**

--- Preservatives
--- Anesthetics
--- Aminoglycosides
--- Cycloplegics
--- Antivirals
--- Miotics
--- α-agonists
--- Antineoplastic

*Which preservative is most commonly implicated in toxic keratoconjunctivitis?*

BAK

*What does BAK stand for?*

Benzalkonium (chloride)
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.

Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:

--Preservatives
--Anesthetics
--Aminoglycosides
--C
--Antivirals
--Miotics
--α-agonists
--Antineoplastic

Which topical anesthetic is most commonly implicated in toxic keratoconjunctivitis?
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for *trachoma* (serotypes A,B,C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (*can you think of a sex-related word that starts with D and ends with K?*), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

*Identify the classic causes of toxic (but **not** necessarily follicular!) keratoconjunctivitis:*

--Preservatives

--**Anesthetics**

--Aminoglycosides

--C

--Antiviral

--Miotics

--α-agonists

--Antineoplastic

*Which topical anesthetic is most commonly implicated in toxic keratoconjunctivitis?*

**Proparacaine**
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A, B, C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—a another bacteria associated with follicles.

Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:
--Preservatives
--Anesthetics
--Aminoglycosides

Name three topical aminoglycosides notorious for inducing toxic keratoconjunctivitis:
--
--
--

--Antineoplastic
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for *trachoma* (serotypes A, B, C—trachoma is as simple as ABC), and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**
- Preservatives
- Anesthetics
- **Aminoglycosides**

**Name three topical aminoglycosides notorious for inducing toxic keratoconjunctivitis:**
- Gentamycin
- Neomycin
- Tobramycin
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and *toxins*. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. *Chlamydia* is the agent responsible for *trachoma* (serotypes A,B,C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

*Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:*
--Preservatives
--Anesthetics
--Aminoglycosides
--**Cycloplegics**
--Antivirals
--Miotics
--α-agonists
--Antineoplastic

*Which cycloplegic is most likely to result in a toxic keratoconjunctivitis?*
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and *toxins*. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for *trachoma* (serotypes A,B,C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**

--Preservatives  
--Anesthetics  
--Aminoglycosides  
**--Cycloplegics**  
--Antivirals  
--Miotics  
--α-agonists  
--Antineoplastic

Which cycloplegic is most likely to result in a toxic keratoconjunctivitis?  
Atropine
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. *Chlamydia* is the agent responsible for *trachoma* (serotypes A,B,C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

Identify the classic causes of toxic (but **not** necessarily follicular!) keratoconjunctivitis:

--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--**Antivirals**
--Miotics
--α-agonists
--Antineoplasts

Which topical antiviral is most commonly implicated in toxic keratoconjunctivitis?
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.

Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:
--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
**--Antivirals**
--Miotics
--α-agonists
--Antineoplastics

Which topical antiviral is most commonly implicated in toxic keratoconjunctivitis?  
Trifluorothymidine
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D–K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**
--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
---Antivirals
----Miotics
------α-agonists
-------Antineoplastics

*Which topical antiviral is most commonly implicated in toxic keratoconjunctivitis?* 
Trifluorothymidine

*What is the brand name for trifluorothymidine in the US?*
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.

### Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:

- Preservatives
- Anesthetics
- Aminoglycosides
- Cycloplegics
- **Antivirals**
- Miotics
- \(\alpha\)-agonists
- Antineoplastics

**Which topical antiviral is most commonly implicated in toxic keratoconjunctivitis?**

Trifluorothymidine

**What is the brand name for trifluorothymidine in the US?**

Viroptic
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for *trachoma* (serotypes A, B, C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis* —another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**
--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
--Miotics
--α-agonists
--Antineoplastic

Which miotic glaucoma med is known to cause toxic keratoconjunctivitis?
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

**Eyelid molluscum lesions** are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) **EKC and PCF** are adenoviral syndromes with brisk follicular responses. **Chlamydia** is the agent responsible for **trachoma** (serotypes A,B,C—trachoma is as simple as ABC) and **adult inclusion conjunctivitis** (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). **Parinaud’s oculoglandular syndrome**, secondary to **Bartonella** infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is **Moraxella** conjunctivitis, aka **Axenfeld conjunctivitis**—another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**
--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
**--Miotics**
 Which miotic glaucoma med is known to cause toxic keratoconjunctivitis?
--α-agonists
Pilocarpine
--Antineoplastic
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

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**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**
--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
--Miotics
**--α-agonists**
--Antineoplastic

Which alpha-agonist is most commonly implicated in toxic keratoconjunctivitis?
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.

Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:
--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
--Miotics
--α-agonists
--Antineoplastics

Which alpha-agonist is most commonly implicated in toxic keratoconjunctivitis?
Brimonidine
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.

Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:
--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
--Miotics
--α-agonists
--Antineoplastic

Which alpha-agonist is most commonly implicated in toxic keratoconjunctivitis?
Brimonidine

Wait—what about apraclonidine? I thought it was more toxic.
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

**Eyelid molluscum lesions** are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. *Chlamydia* is the agent responsible for *trachoma* (serotypes A,B,C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). *Parinaud’s oculoglandular syndrome*, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

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**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**

--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
--Miotics
--α-agonists
--Antineoplastics

**Which alpha-agonist is most commonly implicated in toxic keratoconjunctivitis?**

Brimonidine

Wait—*what about apraclonidine? I thought it was more toxic.*

It probably is, but as it is rarely used on a chronic basis, the overall incidence of toxicity is lower.
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and *toxins*. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. *Chlamydia* is the agent responsible for *trachoma* (serotypes A,B,C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**

--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
--Miotics
--α-agonists

**Antineoplastic**

Which antineoplastic is referred to here?
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and **toxins**. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below).

Eyelid *molluscum* lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. *Chlamydia* is the agent responsible for *trachoma* (serotypes A, B, C—*trachoma is as simple as ABC*) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?) and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis* —another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**
--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
--Miotics
--α-agonists

**--Antineoplastic**

---Which antineoplastic is referred to here?
Mitomycin C
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, Chlamydia (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. Chlamydia is the agent responsible for trachoma (serotypes A,B,C—trachoma is as simple as ABC) and adult inclusion conjunctivitis (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to Bartonella infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.

Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:
--Preservatives?
--Anesthetics?
--Aminoglycosides?
--Cycloplegics?
--Antivirals?
--Miotics?
--α-agonists?
--Antineoplastic?

Circling back to something mentioned previously: While all of the meds discussed are capable of producing a toxic keratoconjunctivitis, not all cause a toxic follicular conjunctivitis. Of the listed classes, which are known to result in a toxic follicular conjunctivitis?
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, *Chlamydia* (which, as an obligate intracellular parasite, is very virus-like), and toxins. Bacteria generally cause a papillary, not follicular, response (two exceptions are discussed below). Eyelid molluscum lesions are a classic cause—any chronic follicular conjunctivitis should elicit a thorough search for molluscum lid lesions. (Extensive molluscum disease is associated with HIV infection; consider testing.) EKC and PCF are adenoviral syndromes with brisk follicular responses. *Chlamydia* is the agent responsible for *trachoma* (serotypes A,B,C—trachoma is as simple as ABC) and *adult inclusion conjunctivitis* (serotypes D-K). Adult inclusion disease is sexually transmitted (can you think of a sex-related word that starts with D and ends with K?), and patients should be asked about urogenital symptoms (concurrent GC is common). Parinaud’s oculoglandular syndrome, secondary to *Bartonella* infection, is an exception to the ‘bacteria don’t cause follicles’ rule. Ocular meds are notorious for producing a follicular conjunctivitis; atropine, dipivefrin, miotics and Viroptic are classic culprits. The zebra in the question is *Moraxella* conjunctivitis, aka *Axenfeld conjunctivitis*—another bacteria associated with follicles.

**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**

--Preservatives
--Anesthetics
--Aminoglycosides
--**Cycloplegics**
--Antivirals
--**Miotics**
--α-agonists
--Antineoplastic

Circling back to something mentioned previously: While all of the meds discussed are capable of producing a toxic keratoconjunctivitis, not all cause a toxic **follicular** conjunctivitis. Of the listed classes, which are known to result in a toxic follicular conjunctivitis?

**Cycloplegics, miotics and alpha-agonists**