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*All are associated with it!*
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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).
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.

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A single *Molluscum* lid lesion, and follicular conjunctivitis
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Molluscum contagiosum in HIV/AIDS
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If a pt presents with chronic (>3 weeks) follicular conjunctivitis, consider these three causes, in this order:

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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

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3. It’s Chlamydia until proven otherwise.
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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*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…
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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…
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\textbf{If a pt presents with chronic (>3 weeks) follicular conjunctivitis, consider these three causes, in this order:}
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\item \textit{Molluscum}—inspect the lids carefully for lesions. If none are found…
\item Toxins—press the pt to recall any history of topical drop use. If no drops…
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\end{enumerate}
Follicular conjunctivitis should make you think of three 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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What non-ocular exam finding is common to all four of these as causes of conjunctivitis?

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

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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.

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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Preauricular lymphadenopathy in pt with epidemic keratoconjunctivitis
Where does trachoma rank as a cause of blindness?

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. *Trachoma* is the #1 cause of preventable/infectious blindness worldwide. Patients from which three parts of the world are most likely to be affected? The Middle East, South Asia, Africa. Which ethnic group in the US is most likely to be affected? Native Americans. Does trachoma result from a single infectious episode? No, recurrent infections are required. What corneal finding is associated with trachoma? Superior pannus, and eventually opacification. In what two locations do trachoma pts develop follicles? The superior palpebral conjunctiva and the superior limbus. 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?
It is the #1 cause of preventable/infectious blindness worldwide.
Where does *trachoma* rank as a cause of blindness? 
It is the **#1 cause** of preventable/infectious blindness worldwide

**Patients from which three parts of the world are most likely to be affected?**

- The Middle East
- South Asia
- Africa

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Patients from which three parts of the world are most likely to be affected?
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Which ethnic group in the US is most likely to be affected?
**trachoma**

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**All are associated with it.** Follicular conjunctivitis should make you think of 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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**Q**

- 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
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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 which three parts of the world are most likely to be affected?**
The Middle East, South Asia, Africa.

**Which ethnic group in the US is most likely to be affected?**
Native Americans.

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Does trachoma result from a single infectious episode? No, recurrent infections are required.
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**What corneal finding is associated with trachoma?**
Superior pannus, and eventually opacification

**In what two locations do trachoma pts develop follicles?**
The superior palpebral conj, and the superior limbus
All of the following are associated with follicular conjunctivitis except (or are they all associated with it?):
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In what two locations do trachoma pts develop follicles?

- Parinaud’s oculoglandular syndrome
- Bartonella infection (Parinaud’s oculoglandular syndrome is an exception to the ‘bacteria don’t cause follicles’ rule.)
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When limbal follicles scar, what eponymous exam finding results?
Herbert's pits

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- Toxins

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Does trachoma result from a single infectious episode?
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What corneal finding is associated with trachoma?
Superior pannus, and eventually opacification

In what two locations do trachoma pts develop follicles?
The superior palpebral conj, and the superior limbus

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

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Trachoma: Herbert’s pits
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Does trachoma result from a single infectious episode?
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When upper-lid tarsal follicles scar, what eponymous exam finding results?
Arlt's line

In what two locations do trachoma pts develop follicles?
The superior palpebral conj, and the superior limbus

The zebra in the question is Axenfeld conjunctivitis, aka Axenfeld conjunctivitis—another bacteria associated with follicles.
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Does trachoma result from a single infectious episode? No, recurrent infections are required. In what two locations do trachoma pts develop follicles? The superior palpebral conj, and the superior limbus. Where does trachoma pts develop follicles? Superior palpebral and limbal.

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Trachoma: Arlt’s line
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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
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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

**In what two locations do trachoma pts develop follicles?**

The *superior palpebral conj*, and the *superior limbus*
Trachoma: Cicatricial entropion
All of the following are associated with follicular conjunctivitis except (or are they all associated with it?):

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

In what two locations do trachoma pts develop follicles?
- Superior palpebral conj
- Superior limbus

The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis — another bacteria associated with follicles.

What does trachoma result from and does it require recurrent infections?
- Recurrent infections are required.

What corneal finding is associated with trachoma?
Superior pannus, and eventually opacification

In what two locations do trachoma pts develop follicles?
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Why is cicatricial entropion a more important finding than Arlt’s line?
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What other, arguably more important exam finding results from scarring of the upper-lid follicles?
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What corneal finding is associated with trachoma?
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What process is that?
The constant irritation of the cornea by the inturned lashes of the entropic upper lid leads to its ulceration, scarring and ultimately opacification.

Arlt’s line and...cicatricial entropion

In what two locations do trachoma pts develop follicles? The superior palpebral conj, and the superior limbus.

The zebra in the question is Moraxella conjunctivitis, aka Axenfeld conjunctivitis — another bacteria associated with follicles.
Trachoma: Cicatricial entropion/trichiasis with corneal opacification
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.

How does the follicular reaction of trachoma differ from that of adult inclusion 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.

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.
Follicular conjunctivitis should make you think of 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 conjunctivitis is a systemic disease, and must be treated as such. (That said, trachoma is often treated with both systemic and topical antibiotics.)

**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.

**How are trachoma and adult inclusion conjunctivitis treated?**

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.
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 a systemic disease, and must be treated as such. (That said, trachoma is often treated with both systemic and topical antibiotics.)

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.

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.)
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 conjunctivitis **is** a systemic disease, and must be treated as such. (That said, trachoma is **often** treated with both systemic and topical antibiotics.)

**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?**
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.

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.

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.)

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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.

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.
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?). 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.

What must you consider if a child presents with adult inclusion 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?). 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 must you consider if a child presents with adult inclusion conjunctivitis?
That the child is a victim of sexual abuse
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.

**Q/A**

*Briefly, how would you describe Parinaud oculoglandular syndrome?*

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
- Toxin 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, 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.

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)
- Pharyngocconjunctival fever (PCF)

**Briefly, how would you describe Parinaud oculoglandular syndrome?**

It is a laterality, **NOT ‘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.

**Briefly, how would you describe Parinaud oculoglandular syndrome?**

It is a unilateral granulomatous 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.

**Q/A**

Briefly, how would you describe Parinaud oculoglandular syndrome?

It is a unilateral granulomatous conjunctivitis associated with nonocular finding.
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.

**Q/A**

**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)

**Briefly, how would you describe Parinaud oculoglandular syndrome?**
*It is a unilateral granulomatous conjunctivitis associated with lymphadenopathy.*
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.

**Q/A**

- Briefly, how would you describe Parinaud oculoglandular syndrome?
  - It is a unilateral granulomatous conjunctivitis associated with lymphadenopathy in the [ ] and [ ] regions.

- 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 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)

Briefly, how would you describe Parinaud oculoglandular syndrome? It is a unilateral granulomatous conjunctivitis associated with lymphadenopathy in the preauricular and submandibular regions.

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.
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.

Briefly, how would you describe Parinaud oculoglandular syndrome? It is a unilateral granulomatous conjunctivitis associated with lymphadenopathy in the preauricular and submandibular regions. 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.

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.”)
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.

A number of other bugs can cause Parinaud oculoglandular syndrome. Which four did I choose to list below?

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

Parinaud's oculoglandular syndrome
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.
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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**F. tularensis** is the causative organism for what disease?

- Tularemia
- *Francisella tularensis* is the causative organism for *Tularemia*.
- *Francisella tularensis* is the causative organism for Tularemia.
- *Francisella tularensis* is the causative organism for Tularemia.
- *Francisella tularensis* is the causative organism for Tularemia.
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- *Francisella tularensis* is the causative organism for Tularemia.

**Parinaud’s oculoglandular syndrome**
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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**F. tularensis is the causative organism for what disease?**
Tularemia

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

**Parinaud’s oculoglandular syndrome**
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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:**

--- Preservatives (a near-ubiquitous component of all drops)

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

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

---?
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**Q**

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

---Preservatives

---?

---?

---?

---?

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---?

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

(a class of med used in virtually all clinic visits, and most surgeries)
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Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:

--Preservatives
--Anesthetics
--Aminoglycosides (a class of antibiotic)
--?
--?
--?
--?
--?

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).

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- Anesthetics
- Aminoglycosides
- ?
- ?
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**Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:**
--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics (a class of med used in most clinic visits, and most surgeries)
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--?
--?
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--Preservatives
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--Aminoglycosides
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--Antivirals
--Miotics
--α-agonists
--? (a class of med used adjunctively in some surgical procedures)
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--Cycloplegics  
--Antivirals  
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--α-agonists  
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\textbf{Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:}

\textbf{--Preservatives}

\begin{itemize}
  \item Anesthetics
  \item Aminoglycosides
  \item Cycloplegics
  \item Antivirals
  \item Miotics
  \item \(\alpha\)-agonists
  \item Antineoplastic
\end{itemize}

Which preservative is most commonly implicated in toxic keratoconjunctivitis?

\begin{itemize}
  \item BAK
\end{itemize}
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---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.

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

--- **Preservatives**

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

Which preservative is most commonly implicated in toxic keratoconjunctivitis? BAK

What does BAK stand for?

**BAK**

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
- Cycloplegia
- Antivirals
- Miotics
- \(\alpha\)-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.
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Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:

---Preservatives
---Anesthetics
---Aminoglycosides
---Corticosteroids
---Antivirus
---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*—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 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).

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**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  

--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
- **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.

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

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

Which cycloplegic is most likely to result in a toxic keratoconjunctivitis?

Atropine

---

A

- 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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*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?*
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—a *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 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**

Which miotic glaucoma med is known to cause toxic keratoconjunctivitis?

--α-agonists
--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.

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

---

Which miotic glaucoma med is known to cause toxic keratoconjunctivitis?

- Pilocarpine
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**Q**

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?

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)
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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 α-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).

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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 α-agonist is most commonly implicated in toxic keratoconjunctivitis? Brimonidine

Wait—what about apraclonidine? I thought it was more toxic. What’s the deal?
All are associated with it. Follicular conjunctivitis should make you think of 3 things: viruses, \textit{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. \textit{Chlamydia} is the agent responsible for \textit{trachoma} (serotypes \textit{A,B,C}—trachoma is as simple as \textit{ABC}) and \textit{adult inclusion conjunctivitis} (serotypes \textit{D-K}). \textit{Adult inclusion disease} is sexually transmitted (can you think of a sex-related word that starts with \textit{D} and ends with \textit{K}?) and patients should be asked about urogenital symptoms (concurrent \textit{GC} is common). Parinaud’s oculoglandular syndrome, secondary to \textit{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 \textit{Moraxella} conjunctivitis, aka \textit{Axenfeld conjunctivitis}—another bacteria associated with follicles.

\textbf{Identify the classic causes of toxic (but not necessarily follicular!) keratoconjunctivitis:}

--Preservatives
--Anesthetics
--Aminoglycosides
--Cycloplegics
--Antivirals
--Miotics
\textbf{--\textit{\alpha}-agonists}

\textbf{Which alpha-agonist is most commonly implicated in toxic keratoconjunctivitis?}
Brimonidine

\textbf{Wait—what about apraclonidine? I thought it was more toxic. What’s the deal?}
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. Ocularmeds 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

**Which antineoplastic is referred to here?**
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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--Preservatives
--Anesthetics
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--Antivirals
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**Which antineoplastic is referred to here?**

Mitomycin C
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**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*

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)