



Infectious Bovine Keratoconjunctivitis (IBK): Etiopathogenesis, Clinical Staging, Therapeutics, and Integrated Control in Cattle

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Infectious Bovine Keratoconjunctivitis (IBK), or "pinkeye," is the most prevalent ocular disease of cattle worldwide. Driven primarily by the Gram-negative diplobacillus *Moraxella bovis*—and frequently co-isolated with *Moraxella bovoculi* and *Mycoplasma* spp.—IBK causes acute conjunctivitis, photophobia, epiphora, and progressive corneal ulceration culminating in stromal rupture and permanent blindness. Virulence depends on Type IV fimbriae (pili) mediating corneal epithelial attachment and a calcium dependent repeats in toxin (RTX) cytotoxin (MbxA) that lyses epithelial cells and neutrophils. Outbreaks reflect an epidemiological triad of host susceptibility (calves and unpigmented breeds), environmental micro-trauma (ultraviolet radiation and pasture seed awns), and mechanical vector transmission by the face fly (*Musca autumnalis*). Beyond welfare compromise, IBK reduces weaning weights, depresses milk yield, and increases treatment costs. This review synthesizes IBK molecular pathogenesis, clinical staging, diagnostics, evidence-based antimicrobial and surgical interventions, and integrated immunoprophylaxis.

Keywords: Corneal ulceration, Infectious bovine keratoconjunctivitis, *Moraxella bovis*, *Moraxella bovoculi*, *Musca autumnalis*

Introduction and Economic Significance

Infectious Bovine Keratoconjunctivitis (IBK) is a highly contagious ocular syndrome affecting beef and dairy cattle globally (Angelos, 2015; Constable et al., 2017). Acute ocular pain and visual loss markedly depress grazing and feed intake. Beef calves with unilateral IBK weigh 7 to 9 kg (15–20 lb) less at 205-day weaning than healthy peers, while bilateral infection causes deficits of 14 to 18 kg (30–40 lb) or more (Snowder et al., 2005; Alexander, 2010). In dairy herds, outbreaks precipitate sharp drops in milk yield and body condition. Coupled with antibiotic costs, chute labor, and market discounts for corneal scarring (leukoma) or globe atrophy (phthisis bulbi), IBK severely impacts farm profitability (Cullen et al., 2016).

Etiology and Molecular Pathogenesis

The primary pathogen is *Moraxella bovis*, an aerobic, Gram-negative, oxidase-positive, non-motile diplobacillus colonizing the conjunctival sac and nasolacrimal duct (Brown et al., 1998). A second hemolytic species, *Moraxella bovoculi* (Angelos et al., 2007), is frequently isolated at higher prevalence during acute field outbreaks, acting as a key co-pathogen

(Angelos, 2015). *Mycoplasma bovoculi* and *Mycoplasma bovis* impair mucociliary clearance and facilitate *Moraxella* colonization, whereas *Listeria monocytogenes* causes distinct silage-associated keratoconjunctivitis and uveitis (Constable et al., 2017).

Non-pathogenic *M. bovis* strains overwinter in carrier nasal and conjunctival mucosa. Clinical pathogenicity requires two virulence factors:

- A. **Type IV Fimbriae (Pili):** Surface filaments bind corneal epithelial microplicae against tear flushing. *Moraxella bovis* alternates between a piliated, infective I-phase and a non-infective Q-phase across seven pilus serogroups (A–G). Chromosomal inversion of pilin genes enables *in vivo* serogroup switching and immune evasion (Postma et al., 2008).
- B. **RTX Cytotoxin (MbxA / Hemolysin):** Encoded by the *mbxCABD* operon, this calcium-dependent pore-forming toxin lyses bovine erythrocytes, corneal epithelial cells, and polymorphonuclear neutrophils (PMNs) (Angelos, 2015).

Corneal stromal liquefaction ("melting") is driven by bacterial fibrinolysin and phospholipase B alongside host immunopathology: MbxA-induced neutrophil lysis releases endogenous matrix metalloproteinases (MMPs) and collagenases that digest stromal collagen (Postma et al., 2008).

Epidemiological Triad and Predisposing Factors

Summer and autumn outbreaks occur when carrier shedding converges with environmental insults and arthropod vectors (Alexander, 2010):

- **Vectors:** The face fly (*Musca autumnalis*) abrades the conjunctiva with sharp prestomal teeth to stimulate lacrimation. *Moraxella bovis* survives on fly mouthparts and in regurgitated crop contents for up to 72 hours, driving rapid herd transmission (Brown et al., 1998).
- **Solar UV Radiation:** UVB light (280–320 nm) induces photokeratitis and epithelial desquamation. Cattle lacking periocular melanin (Hereford, Charolais, Simmental, and white-faced crossbreds) suffer significantly higher morbidity than dark-pigmented *Bos taurus* (Angus) or *Bos indicus* breeds (Snowder et al., 2005).
- **Mechanical Trauma & Host Immunity:** Coarse pasture seed awns, overhead hay racks, and dust create micro-abrasions required for pilus attachment. Calves exhibit lower tear secretory IgA than adults. Concurrent Bovine Herpesvirus-1 (IBR) or Bovine Viral Diarrhea Virus (BVDV) infections, alongside Vitamin A, copper, zinc, and selenium deficiencies, further impair mucosal defense (Constable et al., 2017).

Clinical Staging and Diagnosis

Following a 2–3-day incubation period, untreated IBK progresses through four stages, potentially perforating the cornea within 7-14 days (Alexander, 2010).

Table 1: Clinical Staging, Gross Lesions, and Prognosis of IBK

Stage (Timeline)	Gross Pathological Lesions	Clinical Signs	Prognosis (Treated)
Stage I (Days 1–3)	Conjunctival hyperemia, chemosis, 1–2 mm central corneal opacity/erosion.	Blepharospasm, photophobia, profuse serous epiphora.	Excellent: Resolves without scarring.
Stage II (Days 4–7)	Stromal edema (blue-white haze), shallow ulcer, peripheral neovascularization (salmon-pink ring).	Mucopurulent discharge, head-tilting, shade-seeking, hyporexia.	Good: Heals in 2–3 wk; minor nebula possible.
Stage III (Days 8–14)	Deep stromal ulcer, yellow-white leukocytic infiltrate, hypopyon, anterior uveitis (miosis).	Severe pain, anorexia, milk drop, functional blindness.	Guarded: Heals in 4–8 wk; dense central leukoma.
Stage IV (Day 15+)	Descemetocoele, corneal rupture, iris prolapse, synechiae, keratoconus, or phthisis bulbi.	Aqueous humor leakage, chronic granulation, permanent blindness.	Poor/Grave: Sight lost; enucleation if needed.

Diagnostics: Fluorescein sodium staining confirms ulceration by binding exposed hydrophilic stroma, fluorescing green under cobalt-blue light. Pre-treatment swabs transported in cool Amies medium and plated on 5% blood agar yield small (1–3 mm), friable, grey-white beta-hemolytic *M. bovis* colonies that pit the agar (Angelos, 2015). Multiplex PCR or MALDI-TOF MS differentiates *M. bovis* from *M. bovoculi* (Angelos et al., 2007).

Evidence Based Therapeutic Management

Antimicrobial therapy eliminates *Moraxella* from tears, halts MbxA synthesis, and stops carrier shedding. Systemic lipophilic antibiotics achieving tear Minimum Inhibitory Concentrations (MICs) are preferred (Dueger et al., 1999; Cullen et al., 2016).

Table 2: Recommended Antimicrobial Regimens for IBK

Agent	Route	Dosage Regimen	Clinical & Pharmacokinetic Notes
Oxytetracycline (LA, 200 mg/mL)	IM / SC	20 mg/kg BW; repeat at 48–72 h	First-line systemic standard; high tear levels; clears nasal carriers (George, 1990).
Tulathromycin	SC	2.5 mg/kg BW (single dose)	Prolonged tissue half-life; high field cure rates (Cullen et al., 2016).
Florfenicol	IM / SC	20 mg/kg IM q48h or 40 mg/kg SC once	Rapidly halts ulcer progression and blepharospasm (Dueger et al., 1999).
Penicillin G Procaine	Subconjunctival	1–2 mL (300,000 IU/mL), 22–25G needle	High local tear levels; requires strict restraint to avoid globe trauma (Alexander, 2010).
Cloxacillin (Benzathine)	Topical	Conjunctival sac q24–48h	Non-irritating localized ointment for tractable dairy cattle (Constable et al., 2017).

Adjunctive, Surgical, and Contraindicated Care:

- **Analgesia & Cycloplegia:** Secondary anterior uveitis causes painful ciliary spasm. Systemic NSAIDs like flunixin meglumine (1.1–2.2 mg/kg IV) or meloxicam (0.5 mg/kg SC/PO)—relieve pain. In Stage II–III ulcers with miosis, topical 1% atropine sulfate (q12–24h) induces mydriasis, relieves spasm, and prevents synechiae; treated cattle require shade or patching as the pupil cannot constrict (Constable et al., 2017).
- **Patching & Surgery:** Gluing a breathable cloth patch over the orbit blocks UV light and flies; the ventral edge must remain unglued for exudate drainage. Deep Stage III ulcers or descemetocoeles require a surgical third-eyelid flap or temporary lateral tarsorrhaphy to prevent rupture (Angelos, 2015).
- **Contraindications:** Ocular corticosteroids (dexamethasone) are contraindicated in deep/melting ulcers as they inhibit fibroblasts and activate collagenases, triggering perforation. Ethnoveterinary irritants (dry salt, sugar, turmeric, ground glass, kerosene) destroy regenerating epithelium and must be avoided (Constable et al., 2017).

Integrated Prevention

Sustainable control combines biosecurity and immunoprophylaxis prior to summer transmission:

1. **Vector & Pasture Control:** Combine insecticide ear tags (rotating pyrethroids, organophosphates, and macrocyclic lactones) with back-rubbers, dust bags, and oral larvicides (diflubenzuron, methoprene, tetrachlorvinphos) (Alexander, 2010). Top coarse pastures before seeding, use ground bunks over overhead racks, disinfect headgates/tongs with chlorhexidine, and wear gloves when examining eyes.
2. **Vaccination:** Commercial *M. bovis*/*M. bovoculi* bacterins must be completed 4 to 6 weeks before fly season to establish tear IgA and IgG titers (Angelos, 2015). Where pilus

serogroup diversity or *M. bovoculi* limits efficacy (O'Connor et al., 2011), autogenous bacterins containing local isolates and MbxA toxoid provide targeted protection. Combining early Stage I–II treatment, NSAID analgesia, ocular patching, vector suppression, and pre-seasonal vaccination effectively safeguards herd vision and productivity.

Conclusion

Infectious Bovine Keratoconjunctivitis remains a complex, multifactorial challenge driven by bacterial virulence, environmental micro-trauma, and arthropod vector ecology. Mitigating its severe welfare and economic toll requires shifting from reactive ulcer management to early Stage I–II clinical detection, targeted antimicrobial and analgesic therapy, and protective ocular shielding. Ultimately, integrating multimodal fly suppression, pasture hygiene, strict chute biosecurity, and pre-seasonal vaccination provides the most sustainable strategy for preserving herd vision and productivity.

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