Picornaviridae

FMD , Duck Viral Hepatitis

Pico = minute /small and rna = genome

Classification of Virus

Group IV: ssRNA positive-strand viruses

The virion RNA is infectious and serves as both the genome and viral messenger RNA

Family: Picornaviridae

Genus

Apthovirus	Foot & Mouth Disease, Equine Rhinovirus
Enterovirus	Swine Vesicular Disease, Avian encephalomyelitis, Duck Hepatitis, Polio
Cardiovirus	
Rhinovirus	Human & Bovine Rhinovirus
Hepatovirus	
Parechovirus	

FOOT & MOUTH DISEASE

Foot and mouth disease (FMD) is the most contagious disease of cloven footed animals and has a great potential for causing severe economic losses.

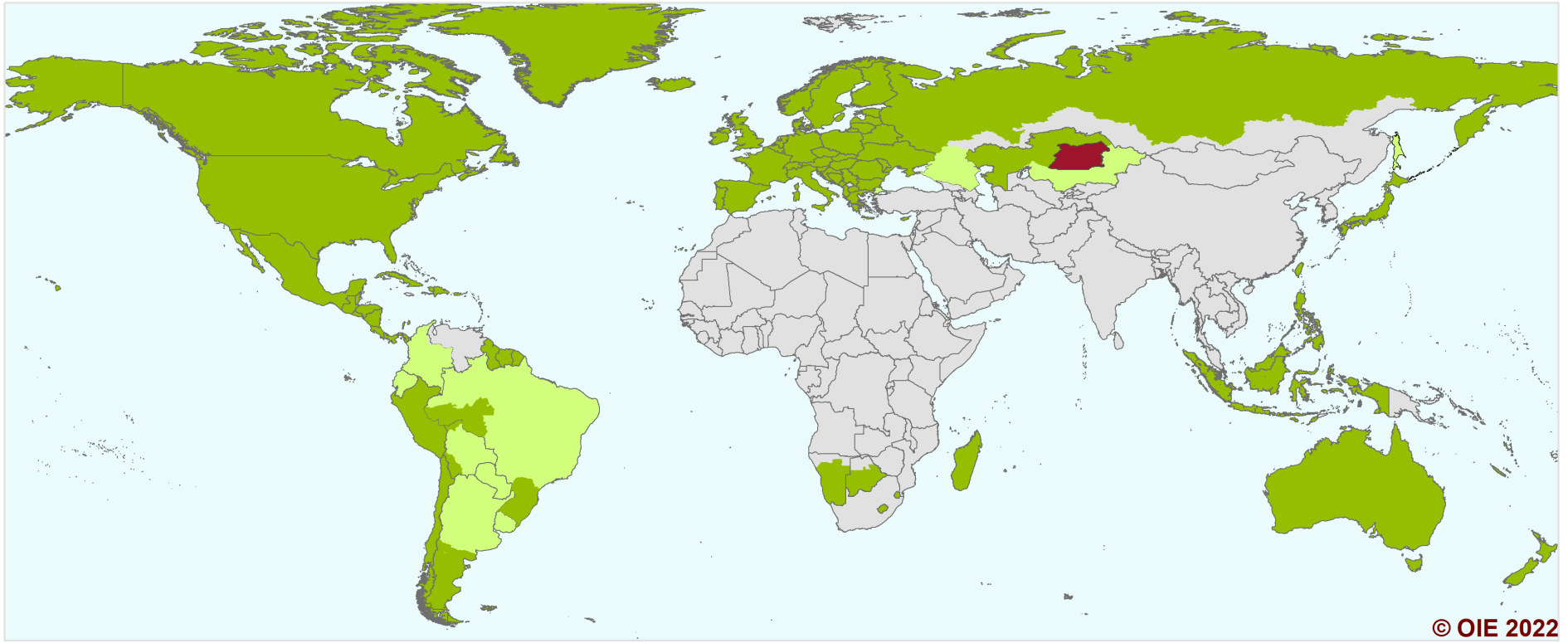
Caused by one of the
seven serotypes of FMD virus, namely, O, A, C, SAT 1, SAT 2, SAT 3 and Asia 1 , characterised by a vesicular condition of the feet, buccal mucosa and, in females, the mammary glands.

NATURAL HOSTS Mostly cloven-hooved animals

TROPISM
Primary site of infection: epithelial cells of the soft palate and pharynx, followed by the lung.
Secondary site of infection: epithelial cells of the feet and mouth.

OIE Members' official FMD status map

Last update January 2022



FOOT & MOUTH DISEASE

Direct losses:

Due to death in young animals.
Loss of milk and meat.
Decrease in productive performance.
Cost occurred for control and eradication of FMD is quite high.
Direct losses in India are estimated to the tune of Rs.6o million per year.(Venkatramanan,*et.al.*2005)

Indirect losses:

Imposition of trade restrictions.

History

Loeffler and Frosch (1897) demonstrated that a filterable agent (virus) caused FMD. This was the **first demonstration that a disease of animals was caused by a filterable agent** and ushered the era of **Veterinary virology**.

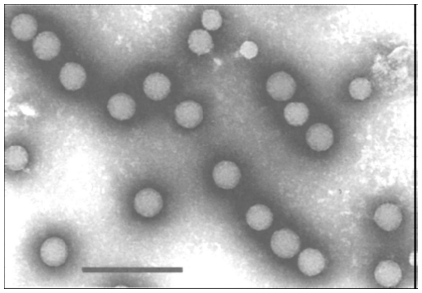
A century later the same virus was among the **first animal viruses to have its structure revolved at atomic level by X-ray crystallography**.

Wladmann and Pape - susceptibility of Guinea pig in 1920.
Skinner -suckling mouse in 1951 (Brown 2003).

Foot and Mouth Disease

F. A. J. Loeffler (left) and P. Frosch, in 1898, working with Robert Koch (right), discovered the **first virus of vertebrates**, foot-and mouth disease virus.

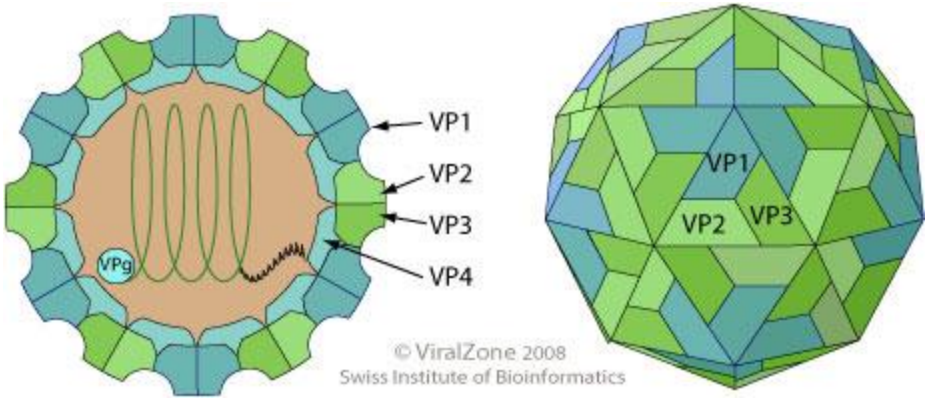
Properties of FMD Virus

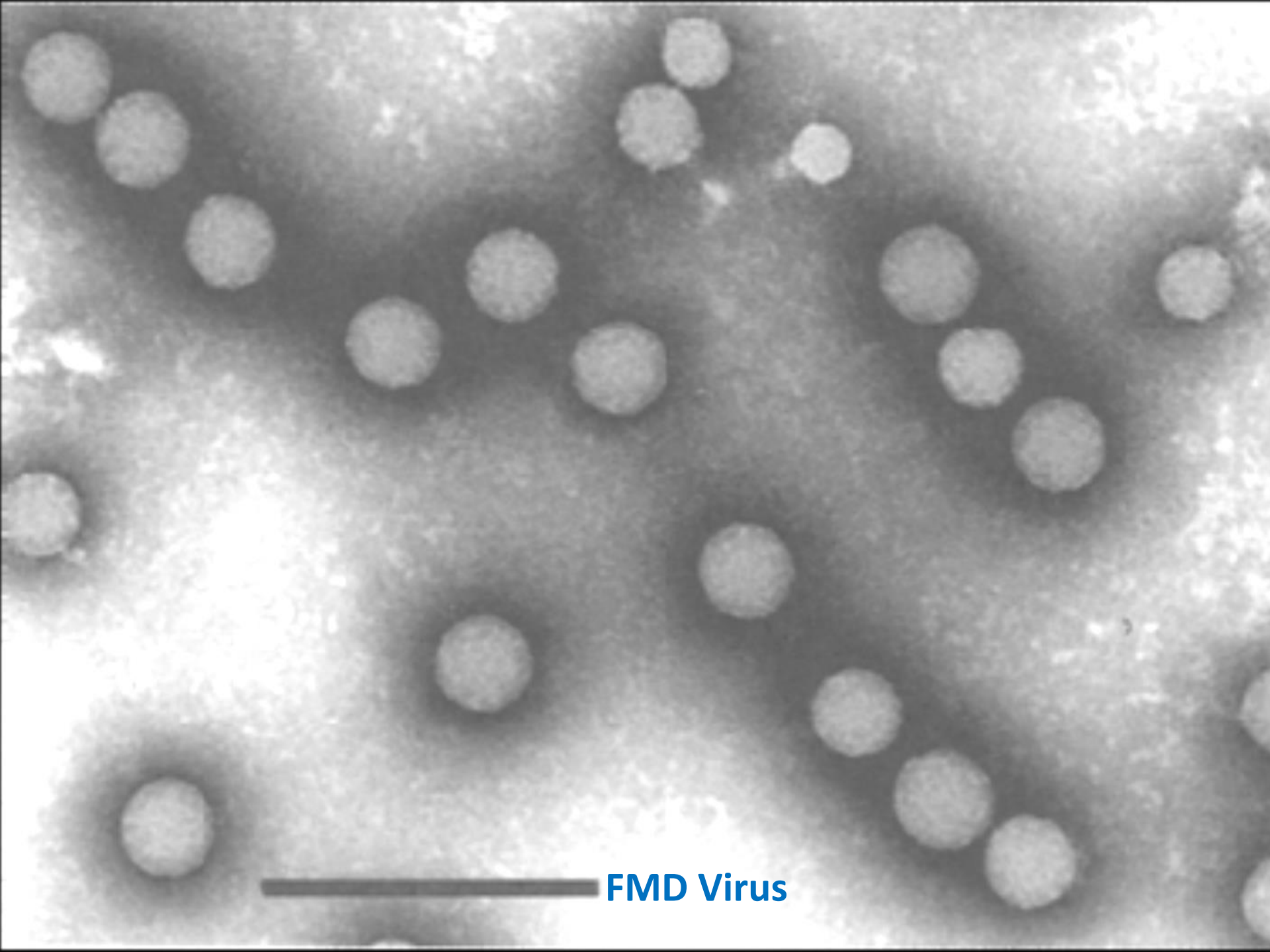


- Non-enveloped virus.
- Virus appears smooth and round outline,
- Size: 27 nm in diameter.
- **Icosahedral symmetry**.
- **Genome**:-Single molecule of linear **positive sense,SSRNA** measuring 7.2-8.4 Kb in size.
- **Genomic RNA is infectious.** Virion RNA acts as a mRNA translated to yield 11 individual proteins.

- Cytoplasmic replication.
- A virus of the family Picornaviridae, genus *Aphthovirus*.

- Seven immunologically distinct serotypes:
  A, O, C, SAT1, SAT2, SAT3, Asia1




FMD Virus

Physical and chemical properties – FMD Virus

- Viable for long period in frozen meat.
- Completely resistant to general chemical disinfection.
- HgCl2 1:1000, 3% Cresol (fail to kill in 6 hours),
- 1% phenol for 5 minutes, **1% Potassium permanganate**
- 70% alcohol for 2-3 days
- Ether and chloroform resistant.
- May survive 85OC for 4 hours in presence of protective colloids.(organic matter). Easily destroyed by alkalies, acid.

Geographical distribution of FMD serotypes

Seven immunologically distinct serotypes: A, O, C, SAT1, SAT2, SAT3, Asia1.

O-Oise Valley,France
A-Allemagne , France

Infection with one serotype doesnot confer the immunity against another.

Prevalent serotypes in India:
In last two decades: **O,A** and **Asia 1**

Geographical Area - Serotypes Prevalent
INDIA –O,A,Asia 1,
ASIA-O,A,C,Asia 1
S.America -O,A,C
Europe -O,A,C
Africa-O,A,C,SAT1,SAT2, SAT3.
N and Central America,Oceania & Caribean Virus free

Transmission of FMD Virus

Direct or indirect contact (droplets)
Animate vectors (humans, etc.)
Inanimate vectors (vehicles, implements)
Airborne, especially temperate zones (up to 60 km overland and 300 km by sea)

Source of virus

Incubating and clinically affected animals
Breath, saliva, faeces, and urine; milk and semen (up to 4 days before clinical signs)

Meat and by-products in which pH has remained above 6.0

Carriers: Virus persists in the oropharynx for up to 30 months in cattle or longer in buffalo, 9 months in sheep.

Hosts

Bovidae (cattle, zebus, domestic buffaloes, yaks), sheep, goats, swine, all wild ruminants and suidae.

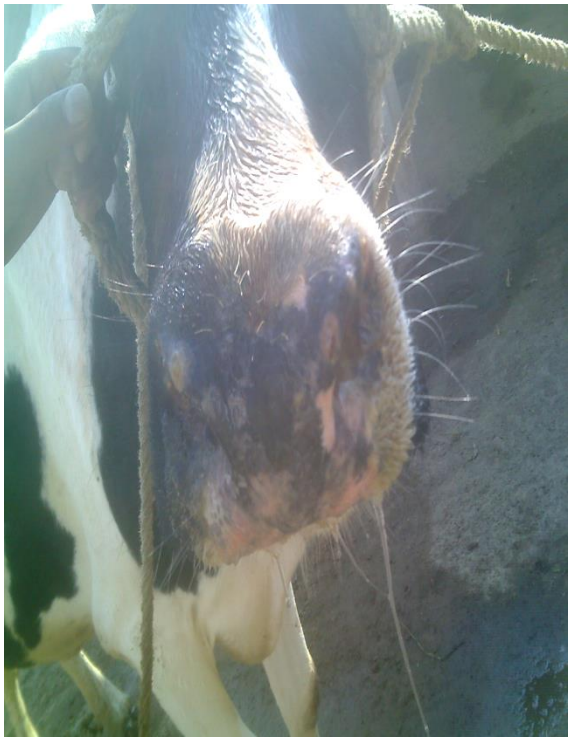
Camelidae (camels, dromedaries, llamas, vicunas) .

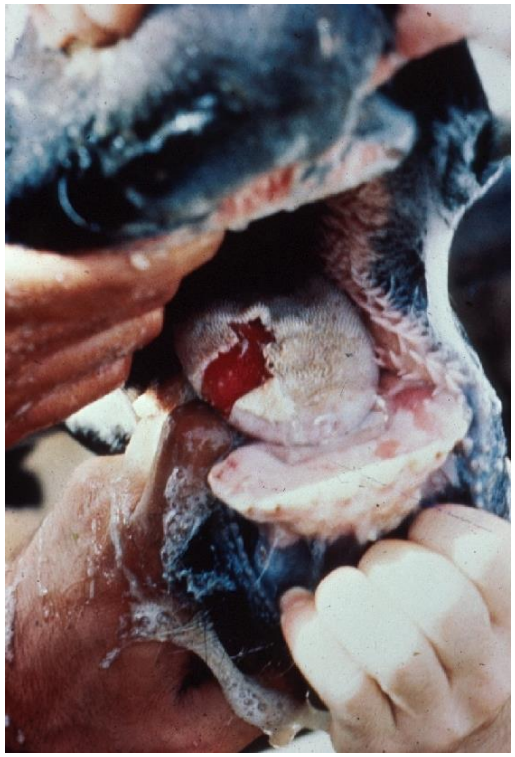
Clinical Features of FMD

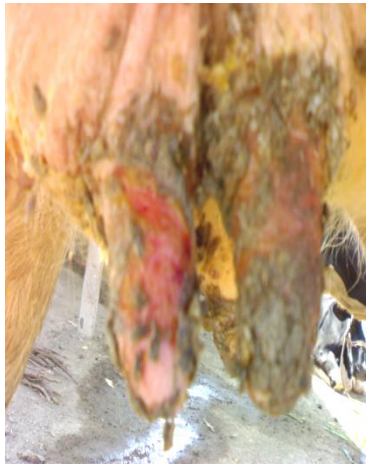
Cattle
Incubation period is 2-8 days
Pyrexia
anorexia
shivering
Marked drop in milk production for 2-3 days.
Animal may open and close its mouth with a characteristic 'smacking' of the lips, grinding of the teeth.
within 24 hours drooling of saliva commences, lameness,
stamping or kicking of the feet: caused by vesicles (aphthae) on buccal and nasal mucous membranes

After 24 hours: rupture of vesicles leaving erosions/denuded ulcerative lesions.
vesicles can also occur on the mammary glands.-leading to mastitis.

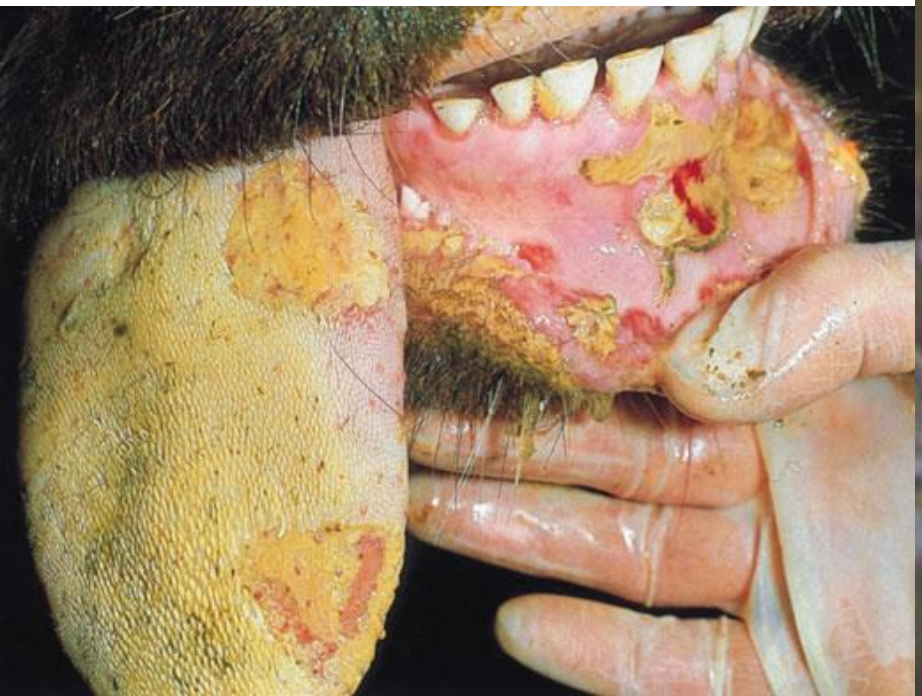
Foot and Mouth Disease



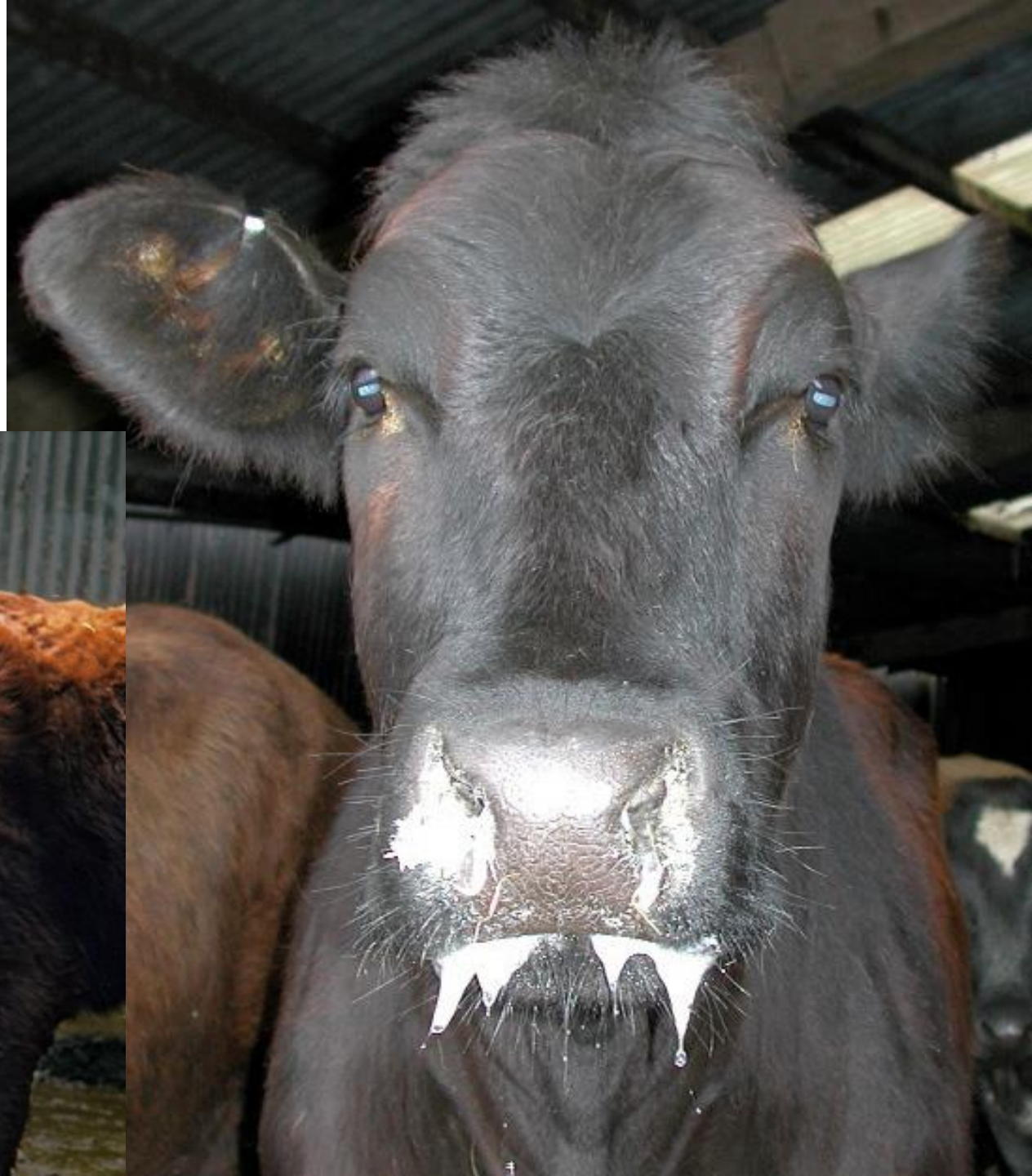




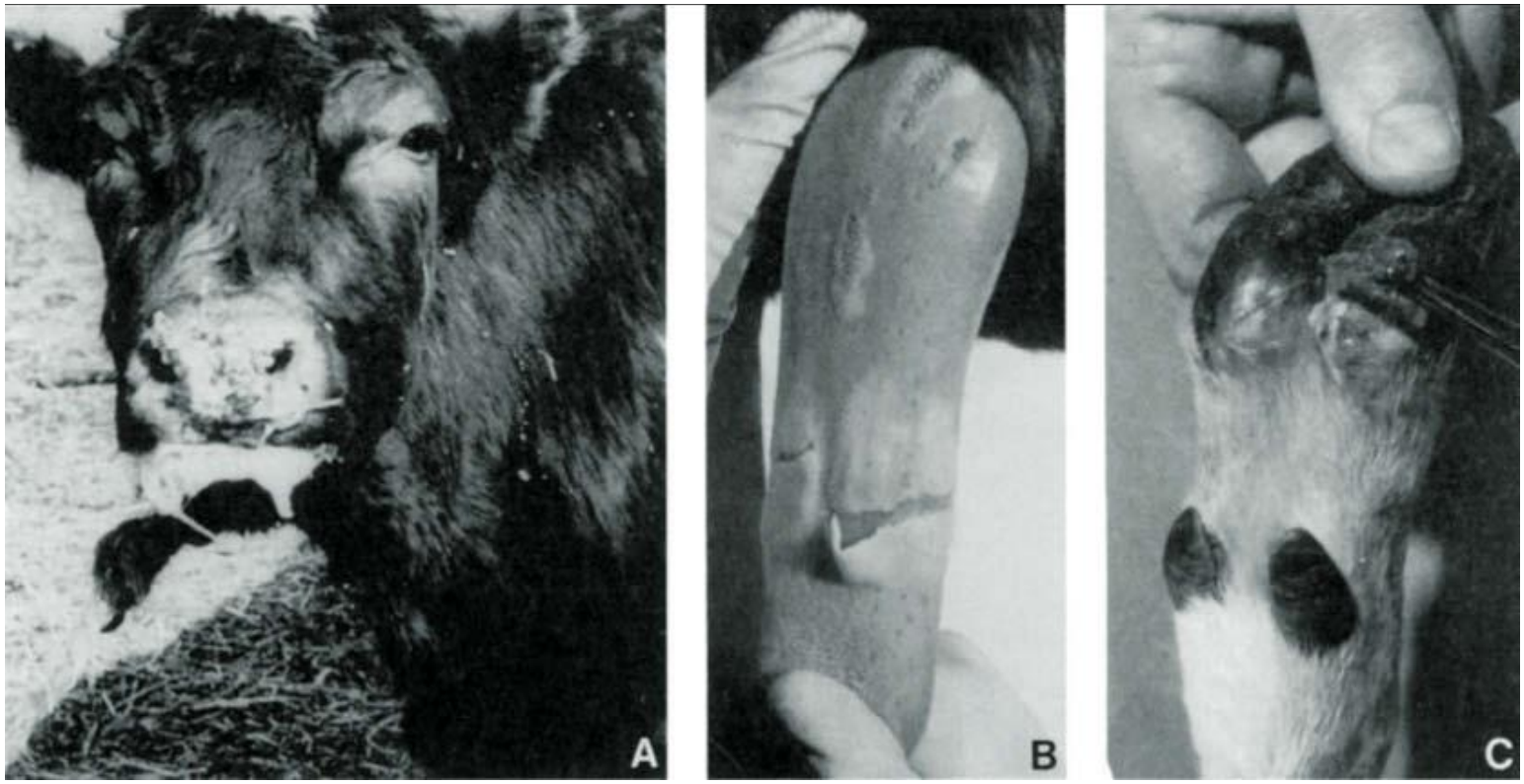
Foot and Mouth Disease



Foot and Mouth Disease

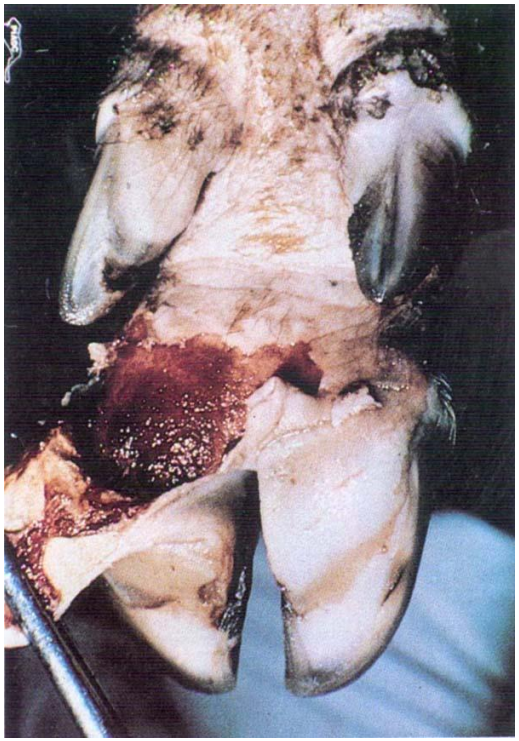


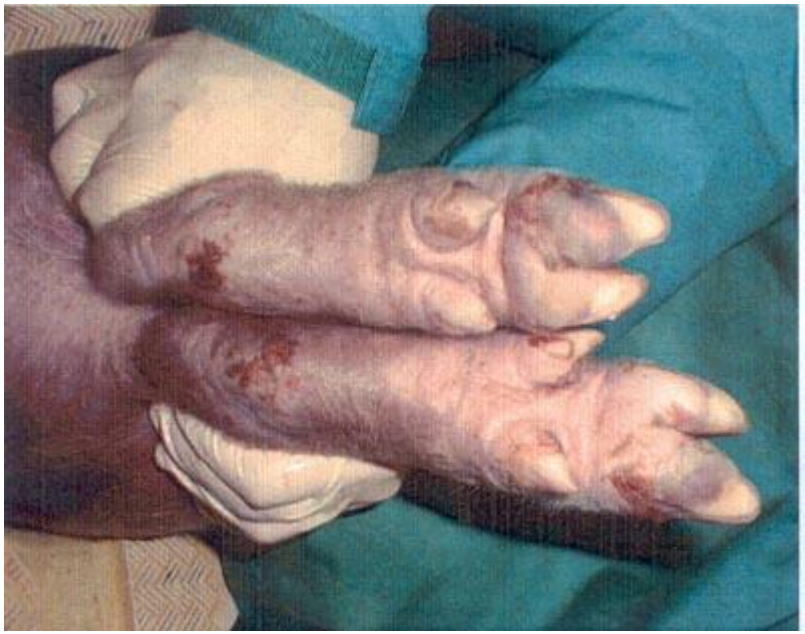
Foot and Mouth Disease



Foot-and-mouth disease. (A) Profuse salivation by a diseased cow. (B) Ruptured vesicles on the tongue of a steer. (C) Vesicular lesions on the foot of a deer.

Foot and Mouth Disease





FMD Detachment of epithelium on the pig's foot

Pigs Lameness is first sign. Denuded areas between the claws become infected with bacteria. May develop severe foot lesions particularly when housed on concrete. **Vesicles may be seen on snout. Vesicles in mouth are less prominent.**

High mortality in piglets a frequent occurrence.

Recovery generally occurs within 8-15 days

Complications: Tongue erosions, superinfection of lesions (secondary bacterial infection), hoof deformation, mastitis and permanent impairment of milk production, myocarditis, abortion, death of young animals, permanent loss of weight,

Panting syndrome - Loss of heat control ability.

Mortality in adult is low.
Morbidity is high.

In calves upto six months of age – May cause death due to myocarditis.

Sheep and goats Lesions are less pronounced.

Foot lesions may go unrecognised.

Lesions in dental pad of sheep. Agalactia in milking sheep and goats is a feature.

Death of young stock.

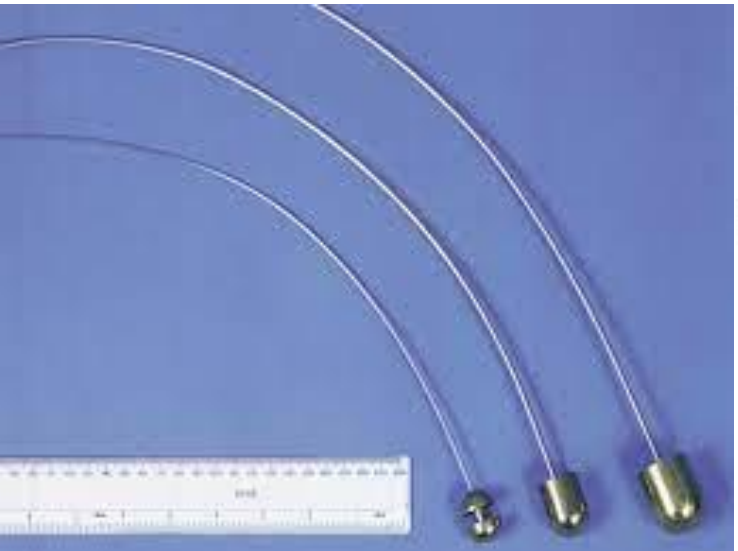
Lesions

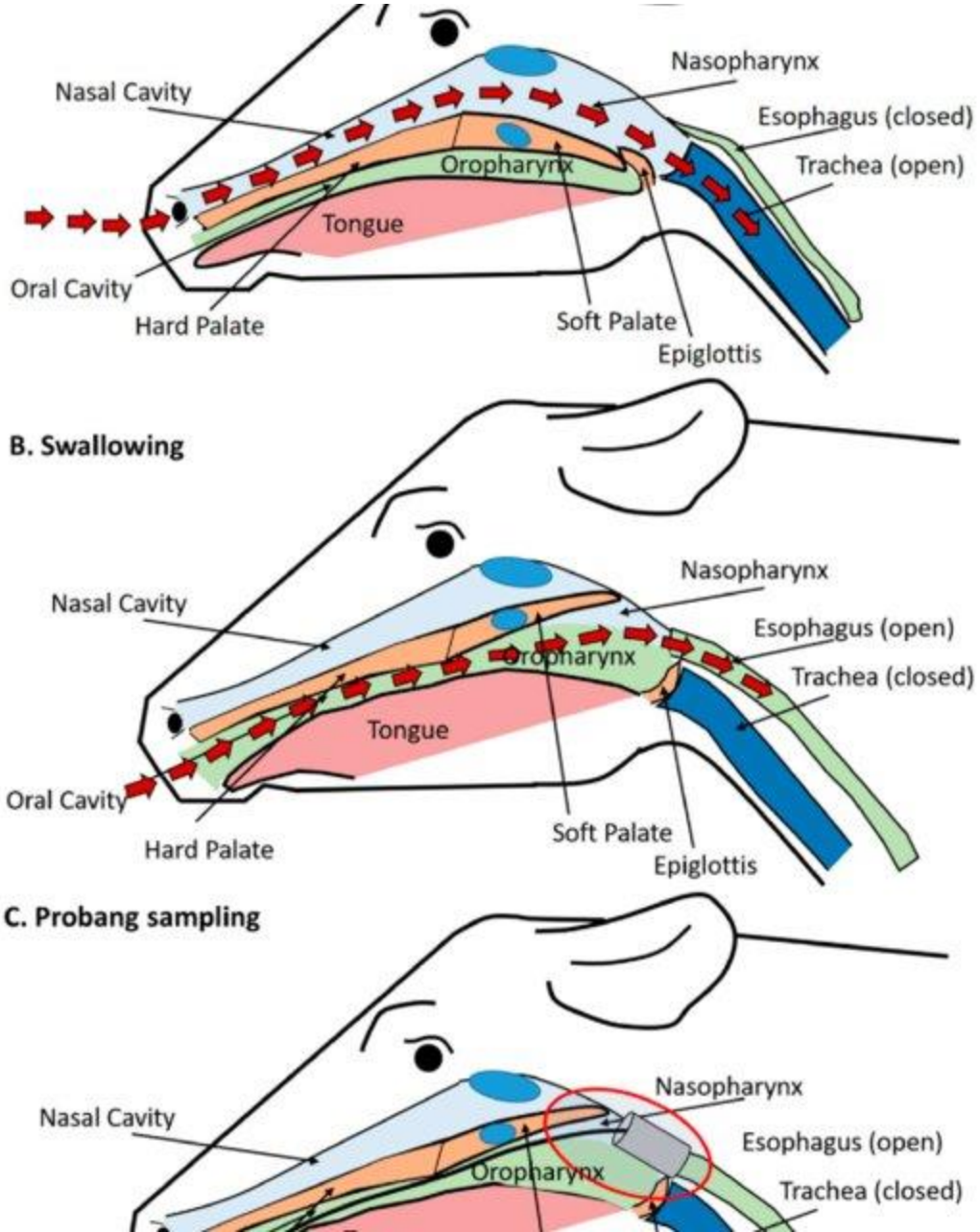
Vesicles or blisters on the tongue, dental pad, gums, cheek, hard and soft palate, lips, nostrils, muzzle, coronary bands, teats, udder, snout of pigs, corium of dewclaws and interdigital spaces

Treatment* for *FMD Animal Isolation, *washing* mouth with 1% *Potassium Permanganate* solution, Applying the mixture of boric acid and glycerine in the mouth.

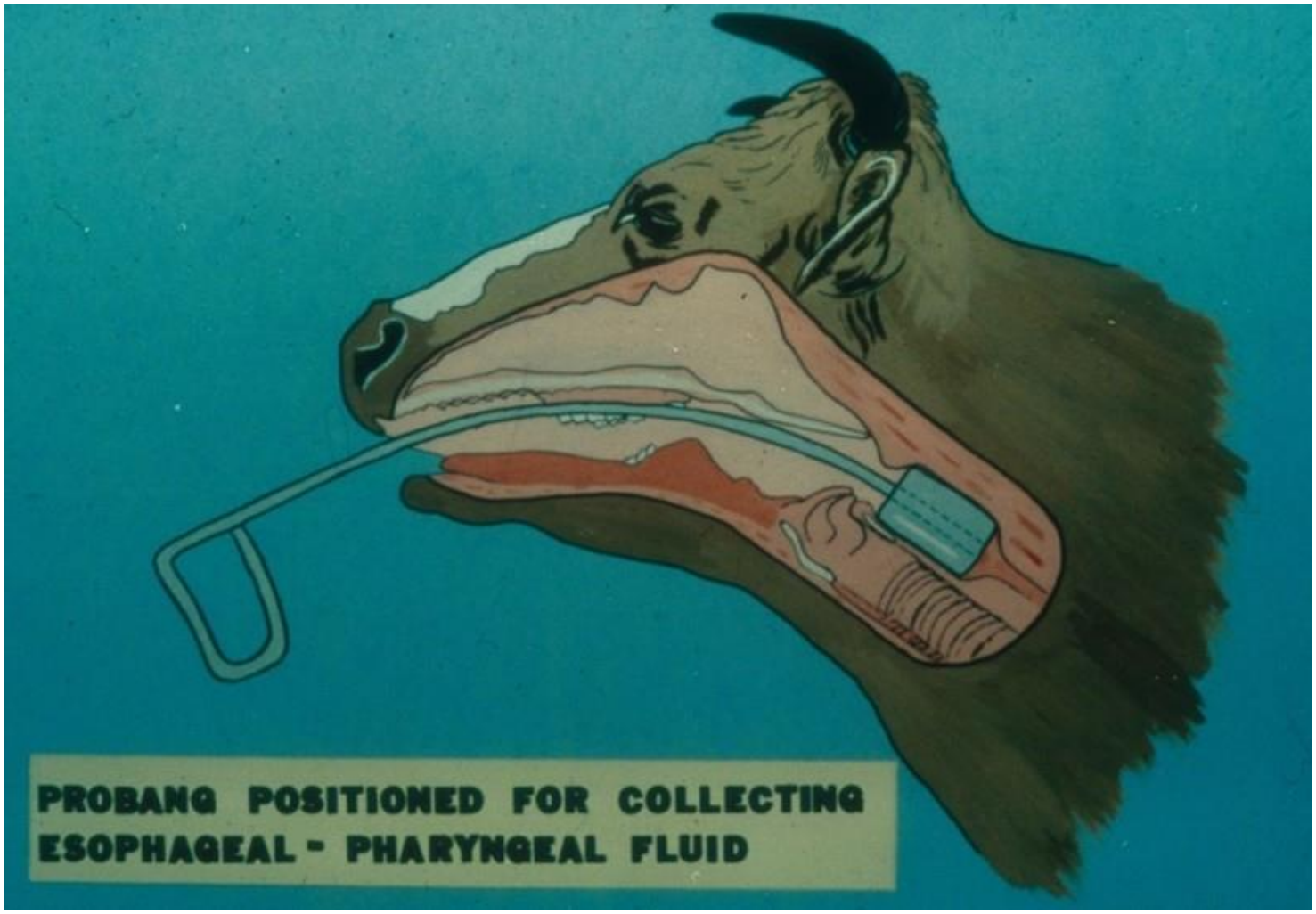
Material Collection

Probang cup





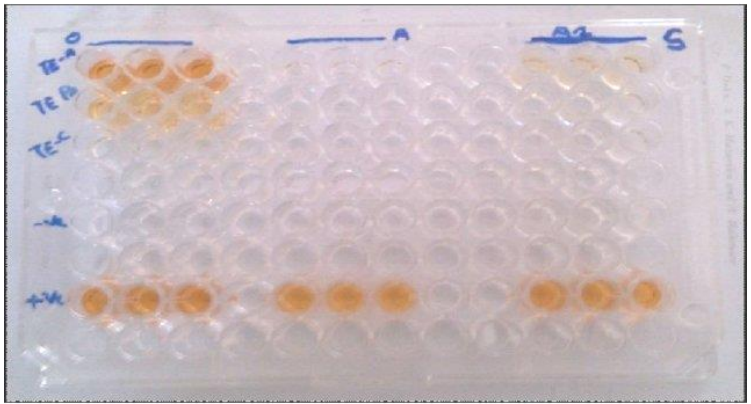
Material Collection



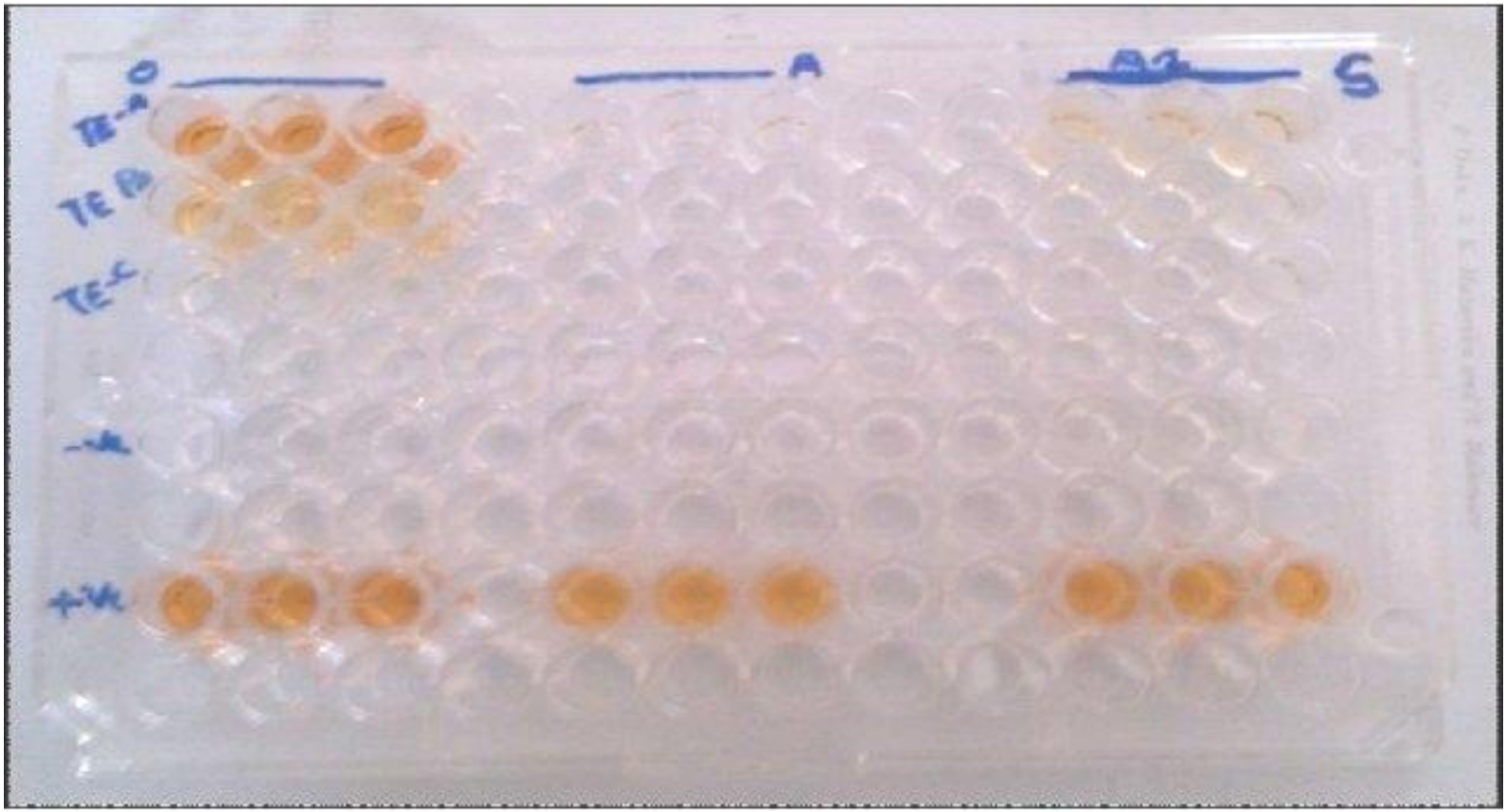
Diagnosis of FMD

1. **Identification of the agent**
   a) **Virus isolation**
      BHK-21, Guinea Pig – Foot pad Inoculation

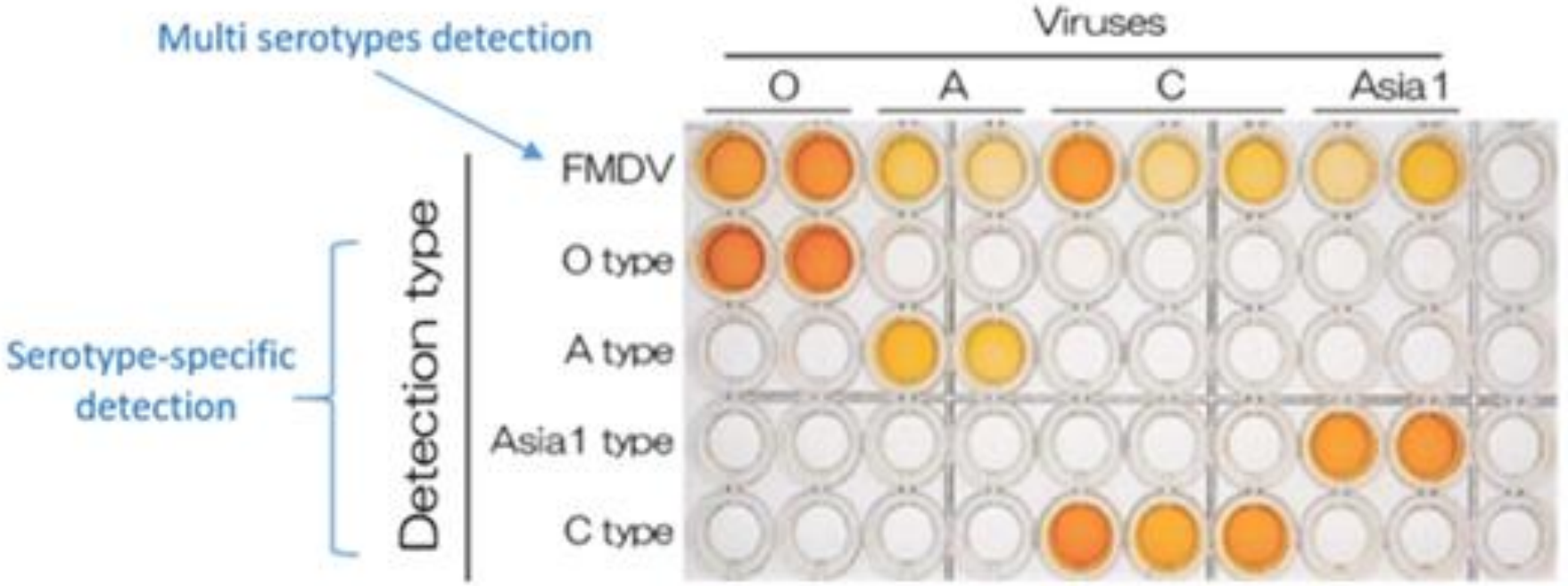
2. **Enzyme-linked immunosorbent assay, Liquid-phase blocking enzyme-linked immunosorbent assay (a prescribed test for international trade)**
3. **Complement fixation test**
4. **Nucleic acid recognition methods: RT-PCR - VP1 gene**
5. **Virus neutralisation**
6. **Nonstructural protein antibody tests.**



ELISA – Foot and Mouth Disease



Foot and Mouth Disease – Serotype Detection



Prevention & Control of FMD

1. Slaughter of infected, recovered, and FMD-susceptible contact animals.

2. Disinfection of premises and all infected material (implements, cars, clothes, etc.)

3. Destruction of cadavers, litter, and susceptible animal products in the infected area.

4. Quarantine measures &

5. Vaccination

Vaccination

Inactivated Foot and Mouth Disease vaccine with concentrated antigens adjuvanted with mineral oil**.**

Composition

The vaccine contains a mixture of Foot and Mouth Disease virus Serotype(s) O, A, and Asia 1, propagated in cell culture, inactivated by BEI, concentrated and emulsified in a mineral oil adjuvant.

Administration and Dosage (Intramuscular injection)

Cattle and Buffaloes - 2 ml

Sheep and Goats - 1 ml

Vaccination Schedule

Cattle, Buffalo- After 4 months of age-After 6 months of Primary vaccination-Yearly after Booster vaccination

Sheep & Goat-After 4 months of age-After 6 months of Primary vaccination-Yearly after Booster vaccination

Storage: Between 2 - 8°C. Do not freeze. Presentation :Vials of 10 ml (5 doses) and 30 ml (15 doses),Shelf Life:24 months

Duck Viral Hepatitis

1945 Long Island, New York
Prevalent in Duck raring parts in India – Kerala, West Bengal

Host: Ducklings during first few weeks.

Etiology – Duck Virus Hepatitits

DVH is caused by three different viruses.

The most severe and widely distributed virus, **Duck hepatitis A virus** (DHAV) 1 (formerly called DHV-1), belongs to the **Genus Enterovirus** and family *Picornaviridae*, and causes disease in ducklings before 6 weeks old.

The other two viruses are **Duck Astrovirus** (formerly known as DHV-2), which causes disease in **ducklings between 6 and 10 weeks** old and

DHV-3 caused by another virus unrelated to DHV-1 and DHV-2 which causes **milder disease**.

Transmission

The disease is very contagious and the **virus excreted in faeces** is transmitted by **direct contact between birds or through fomites** such as brooders, water, feed, equipment.

Recovered animals can shed the virus for up to 8 weeks.

Duck Virus Hepatitis

Clinical signs

- Incubation period lasts 1 to 2 days DHV-1 causes the most severe disease.
- Death occurs within an hour (sudden death)after the first clinical sign
- Affected **ducklings kicks spasmodically in lateral recumbency**.
- Retracts its head and neck and death occurs (opisthotonos).
- Other clinical signs include lethargy, anorexia.
- **Morbidity is often 100% and mortality reaches 80%.**
- Disease is less severe in ducks older than 7 weeks.

Post-mortem findings

- The **liver is enlarged with haemorrhagic lesions** (petechia, ecchymosis) , mottled and discolouration. The spleen and kidneys enlarged. (Severe Hepatic Necrosis)

Duck Viral Hepatitis

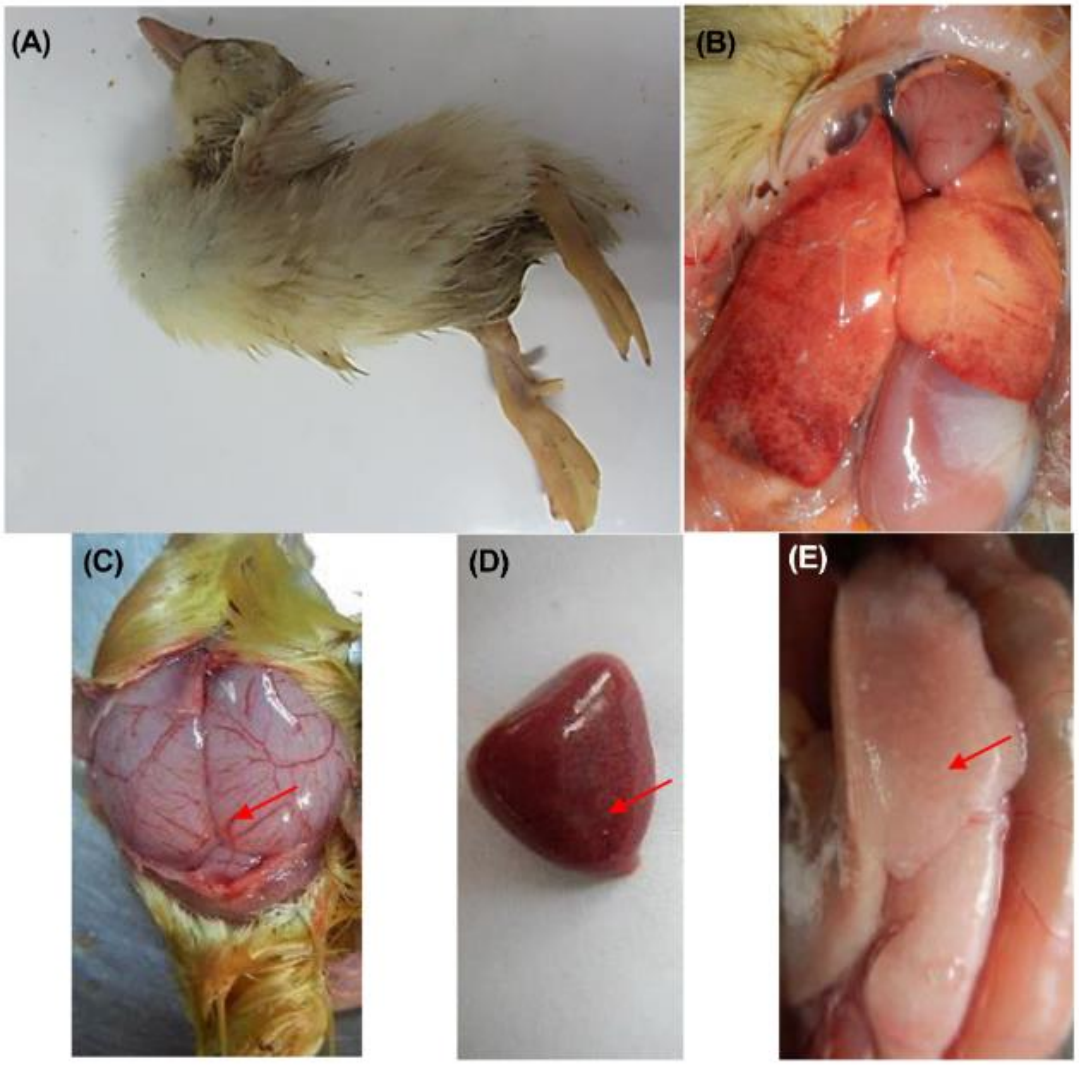
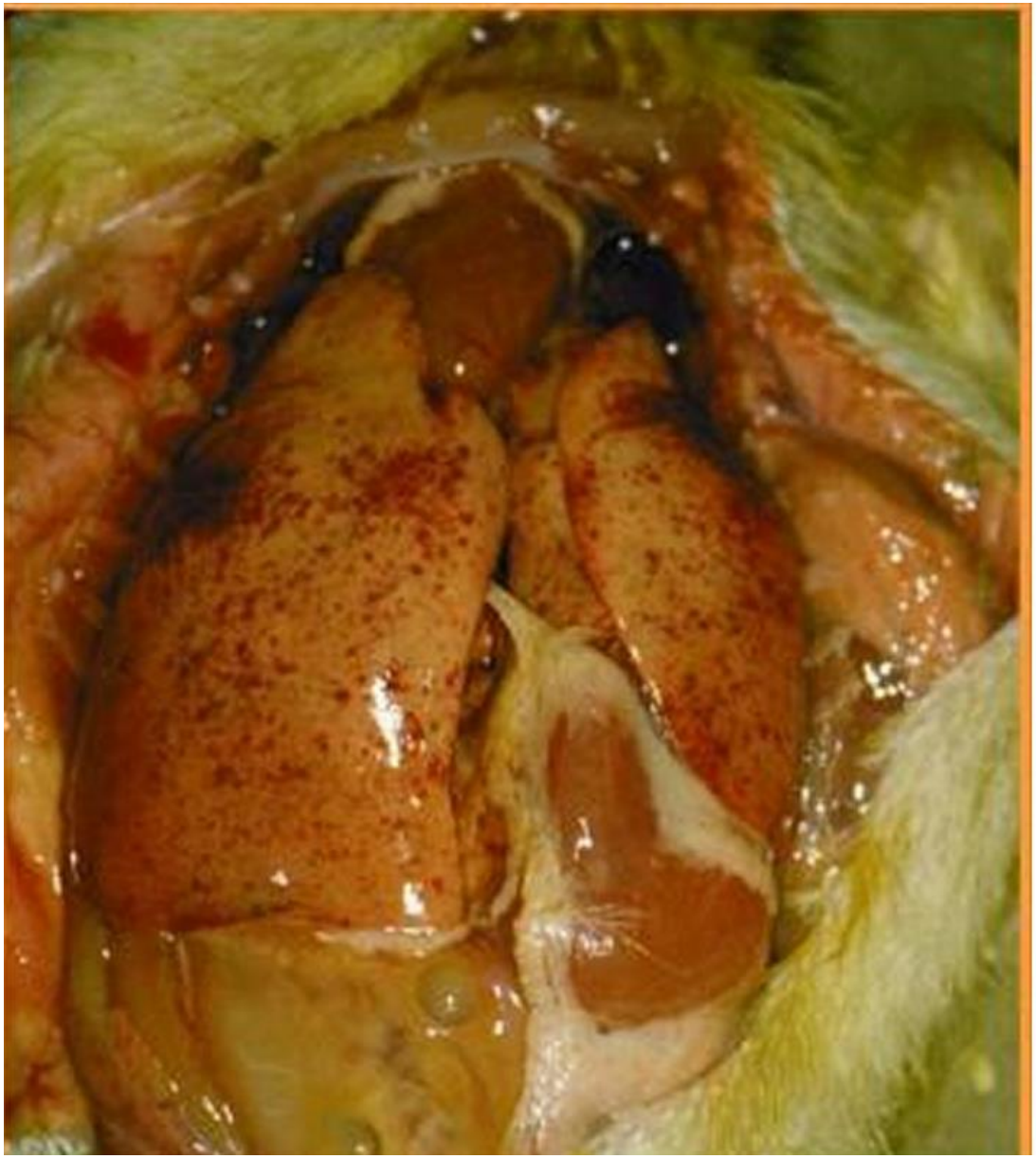


Figure 2. Gross lesions of ducklings infected with DHAV-1 and DHAV-3 at 48 h post-infection. (**A**) The dead ducklings neck back and opisthotonos. (**B**) The liver of dead ducklings is typically enlarged with petechial and ecchymosis hemorrhages throughout. (**C**) Severe meningeal hyperemia and hemorrhage was in some dead ducklings (red arrow). (**D**) The spleen presented reddish brown with congestion and gray-white with necrosis (red arrow). (**E**) The pancreas were covered with gray-white focal necrosis (red arrow).



Duck hepatitis A virus type 1, week-old duckling

Duck hepatitis A virus type 1 in a week-old duckling. Note hemorrhagic lesions on liver.

Courtesy of Dr. Peter R. Woolcock.

Laboratory Diagnosis Duck Hepatitis

1.Isolationof DHAV-1 :

Liver-material of choice.

ECE-Cam/Allantoic route Death of embryo after 4-5 days with haemorhages and edema.

Primary duckling embryo monolayer or duckling liver monolayer.

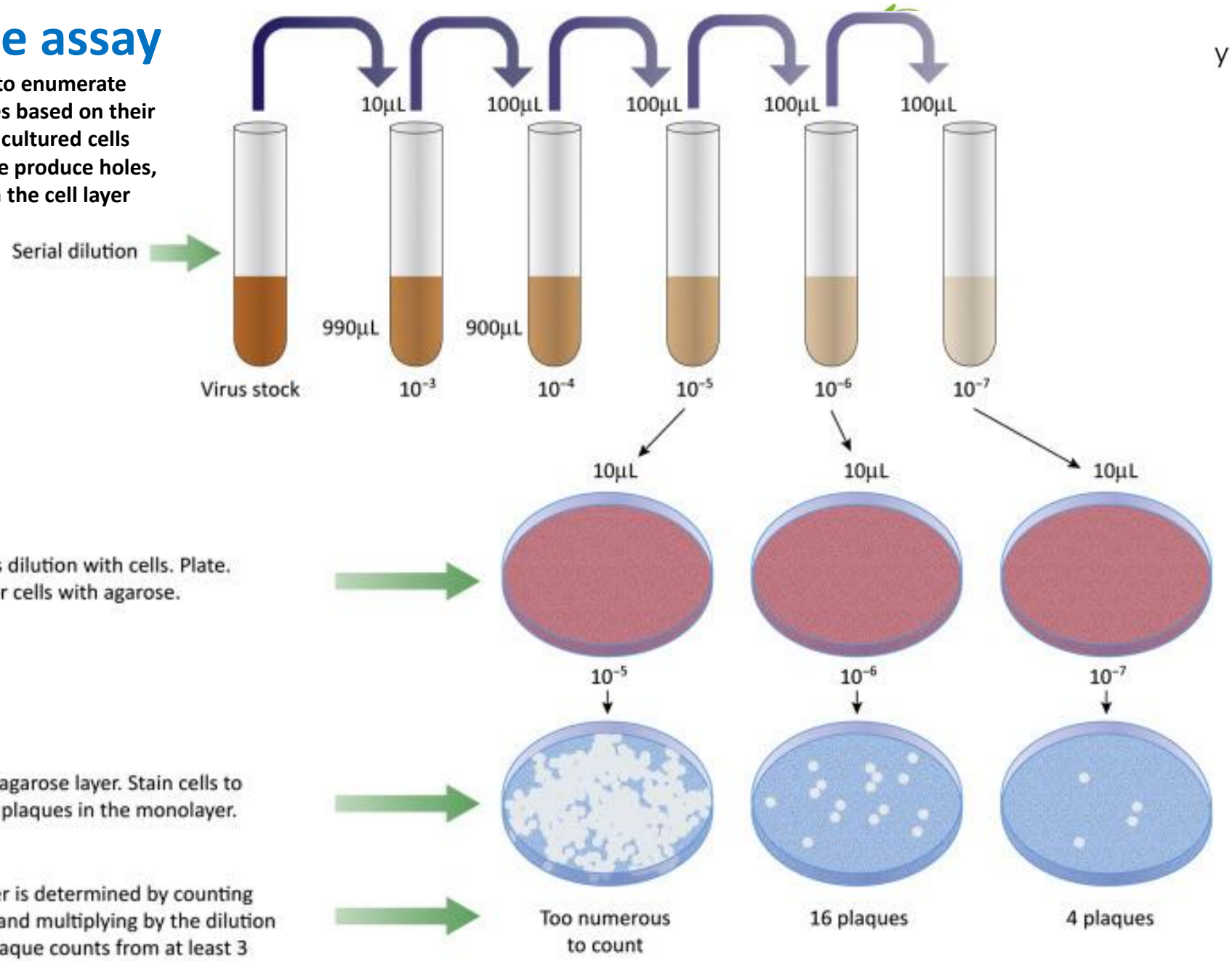
CPE- Hydropic degeneration, roundening.

DHAV can readily be recovered from liver tissue by homogenisation as a 20% (w/v) suspension in buffered saline. The suspension is clarified, and can then be treated further (if desired) with **5% chloroform (v/v) for 10–15 minutes** at ambient temperature. **DHAV is resistant to this treatment**.

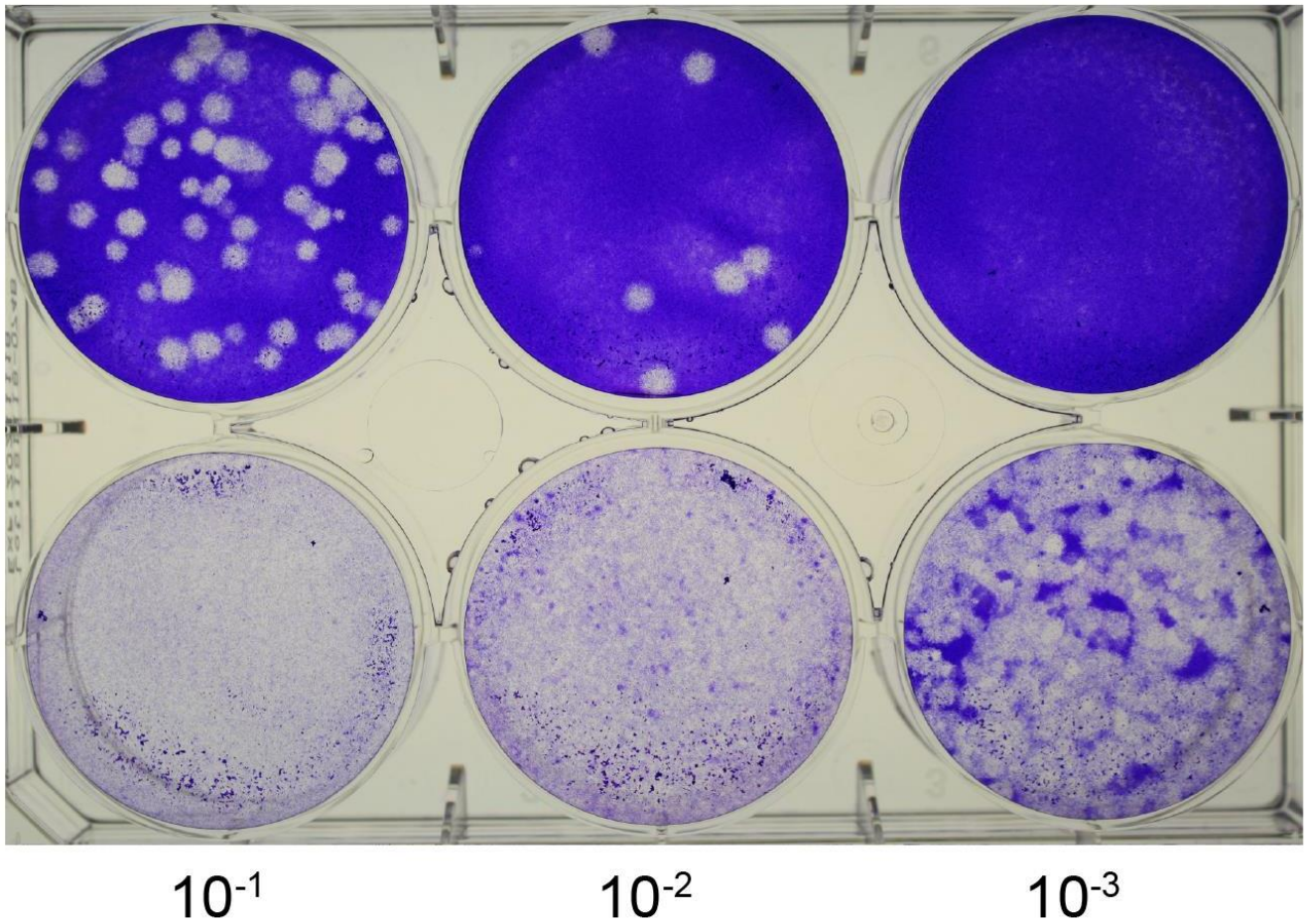
The presence of DHAV is usually confirmed by one or more of the procedures listed i) By inoculation of primary cultures of **duck embryo liver (DEL) cells**, which are particularly sensitive. Dilutions of the liver homogenate containing DHAV type 1 cause a cytopathic effect (CPE), which is characterised by **cell rounding and necrosis. When overlaid with a maintenance medium containing 1% agarose (w/v), the CPE gives rise to plaques approximately 1 mm in diameter**

Plaque assay

a technique to enumerate virus particles based on their ability to kill cultured cells and therefore produce holes, or plaques in the cell layer



Plaque assay



Laboratory Diagnosis Duck Viral Hepatitis

2.FAT

Detection of DHAV antigen by FAT

3.Differential diagnosis from Salmonellosis & Aflatoxicosis.

4. RT-PCR

Duck Hepatitis A

Vaccines

- Vaccination against DHAV-1 and DHV-3 is possible using live attenuated vaccines.
- A killed vaccine is also available against DHAV-1.

THANKS

AB's Veterinary Microbiology

www.veterinarymicrobiology.in