

## *Scrapie*

- Its non febrile, fetal chronic disease of sheep & goats characterize by long incubation period, pruritus abnormal gait
- It caused by prion protein (PrP<sup>sc</sup>) which is an abnormal isoform of host coded membrane glycoprotein which may accumulated in neurons or other tissues & highly resist to chemical & physical agents.

### Epidemiology...

- 1-Its enzootic disease with wide distribution, affected mature animals (2.5-4.5 years) with high mortalities
- 2-pre-clinically infected sheep is the main source of infection
- 3-infection can be demonstrated in placenta, fetal fluids, intestine & nasal mucosa of naturally occurring cases
- 4-ingestion of infected materials considered as a good role for transmission
- 5-Horizontal transmission mostly occur through contaminated placenta from dam to lamb or lamb to lamb
- 6-Vertical transmission may also occur (scrapie agent were demonstrated in ovaries & uterus but not in testes & semen)
- 7-hay mites may consider as reservoir & there is no evidence to transmission to human.

### Pathogenesis ...

- 1-After entrance to body, replication occur in lymphoreticular tissues (tonsils, retropharyngeal L.N, spleen & intestine)
- 2-The agent then reach CNS through uncertain way (may be sympathetic nerves or vagus )
- 3-In brain the agent cause non-inflammatory, vacuolar degeneration of gray matter

### Clinical findings

- 1- Incubation period varies from several months –several years
- 2-early signs manifested by

Transient nervous signs included sudden collapse, behavior change, rubbing & biting at the fleece (pruritus) with loss of body condition.

3-Advanced signs characterized by...

A-intense pruritus (persistence rubbing) (nibbling)

B-loss of wool, hematoma of ears and swelling of face due to severe scratching

C- Abnormality of gait (incomplete flexion of hocks, shortening of the step, weakness, lack of balance, ataxia)

D-general nervous signs included, tremor, hyperexcitability, jerking of head, nystagmus, rotation of the head, inability to swallow, blindness,

E-finally severe emaciation, sternal to lateral recumbency, & death.

Clinical path..

- No hematological changes
- Examination of skin to exclude other skin infection
- Detection of the prion by tonsillar biopsy

Control...

\*\*Eradication

### ***Bovine spongy form encephalopathy* ,,(*BSE*)**

- BSE is a non febrile neurological disorders of adult cattle may believed to belong to scrapie when cattle expose to feed (animal protein contaminated with scraipe agents). And may transmitted to human.
- Its caused by transmissible agent of a scraipe strain that's modified to infect cattle
- The disease described in Great Britain (at the first time)then developed as epizootics
- Adults cattle affected with long incubation period (2.5-8 years)or more
- The disease can also affected other types of animals & The main route of transmission is through ingestion of meat& bone meal
- Transmission to human is possible (Creutzfeldt-Jakob, kuru, )

#### Pathogenesis ...

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- 2-The agent then reach CNS through uncertain way (may be sympathetic nerves or vagus )
- 3-In brain the agent cause non-inflammatory, vacuolar degeneration of gray matter

#### Clinical findings....

- Predominates nervous signs (behaviors changes, hyperesthesia ,ataxia,)loss of body condition ,decrease milk production,

#### Clinical path...

- 1-Apolipo-protein E were detected in CSF
- 2-histo-pathologic change detected bilateral symmetric intracytoplasmic vacuolation of neurons

#### Control...

#### Eradication

