



Better Training for Safer Food BTFSF

**Importation of vector-borne infectious
diseases**

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Importation routes

Trade

- **Infected live animals (e.g. H5N1 avian influenza)**
- **Infected animal products (e.g. FMD) and by-products (e.g. anthrax)**

Movements of travellers and pets

- **Epidemic diseases (e.g. SARS)**
- **Endemic diseases (e.g. rabies)**

Movements of wildlife (e.g. raccoon dog and rabies, wild birds and H5N1 influenza)

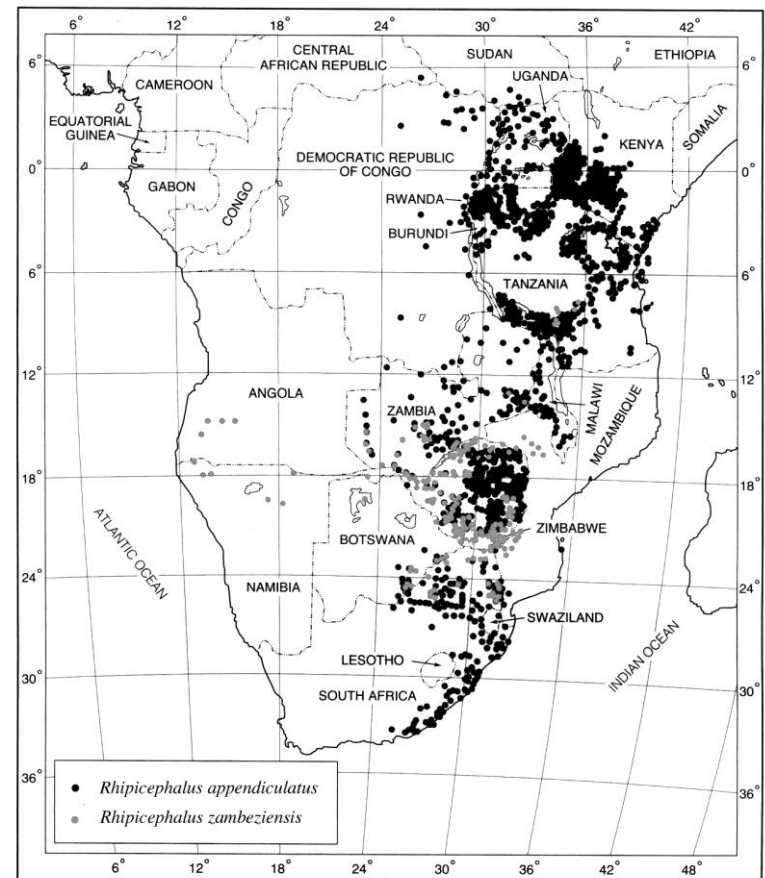
Movements of vectors (or invertebrate hosts)

Trade: the example of East Coast fever in Africa

*Tick-borne disease:
Theileria parva transmitted
by Rhipicephalus
appendiculatus*

*Endemic in East and
Central Africa*

- **Calves infected early in life**
- **Strong immunity in surviving animals**
- **Long term carrier state**
- **ECF went unnoticed in endemic areas**



East Coast fever in South-Africa

Rinderpest outbreak: 1897 – slaughter of large numbers of cattle

Anglo-Boer war: 1899-1902 – destruction of farms

Restocking of cattle through importation from the whole British empire

East Coast fever outbreaks in Durban – “atypical piroplasmosis” : > 90% mortality!



European
Commission





European



Main determinants of the first ECF outbreak in South Africa

Large population of susceptible animals (non immune)

Infected animals from Kenya bypassed geographical barriers (unsuitable for tick survival)

Healthy carriers

Local environment appropriate for

- **Vector survival and development**
- **Pathogen transmission**

Unknown epidemiology

- **Role of ticks**
- **Carrier animals highly appreciated!**

ECF eradication in South Africa

*Most of SA areas are inappropriate for
*R. appendiculatus**

Well organised and very strict veterinary services

- **Tick control**
- **Destruction of infected animals**
- **Prohibited chemotherapy**

Long and expensive exercise

Failure to eradicate the vector tick

*Failure to eradicate *T. parva* from wildlife*

*Failure to eradicate or even stop the progression
of ECF in most surrounding countries*



European
Com

ECF and wildlife

"Corridor disease"

Buffaloes are asymptomatic carriers

*Cattle are infected when
exposed to ticks that previously
fed on buffaloes*

Breaking out of the fences

Transfer of animals

Acute and lethal disease in cattle

Adaptation to cattle?



Traveller movements: the example of *Dirofilaria spp*

Nematod worms:

- *Dirofilaria repens* (subcutis)
- *Dirofilaria immitis* (heart)

FH: Domestic and wild carnivores (mostly dogs)

IH: Culicidae (mosquitoes)

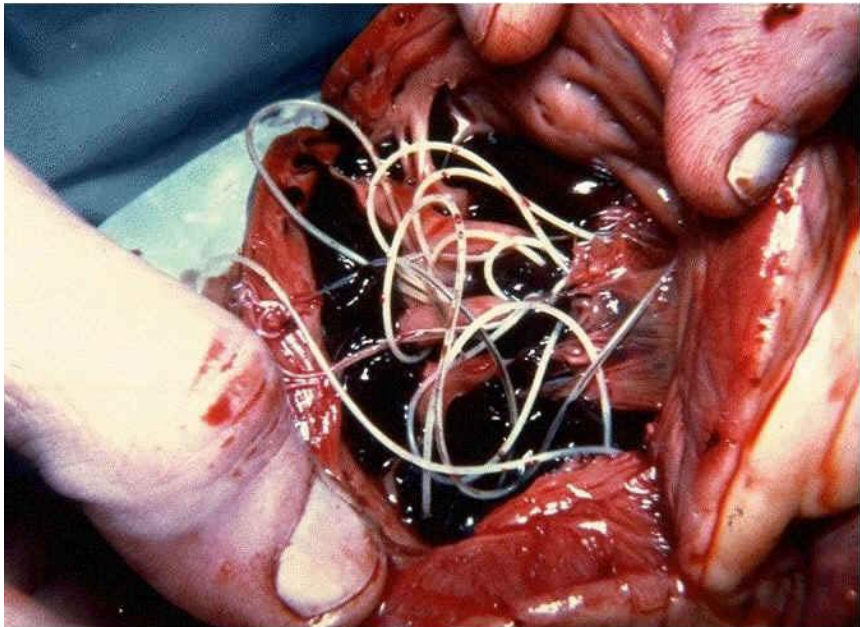
Humans: incidental host

Geographic distribution: Southern and Eastern Europe

High temperature required for the maturation of the larval stage in mosquitoes

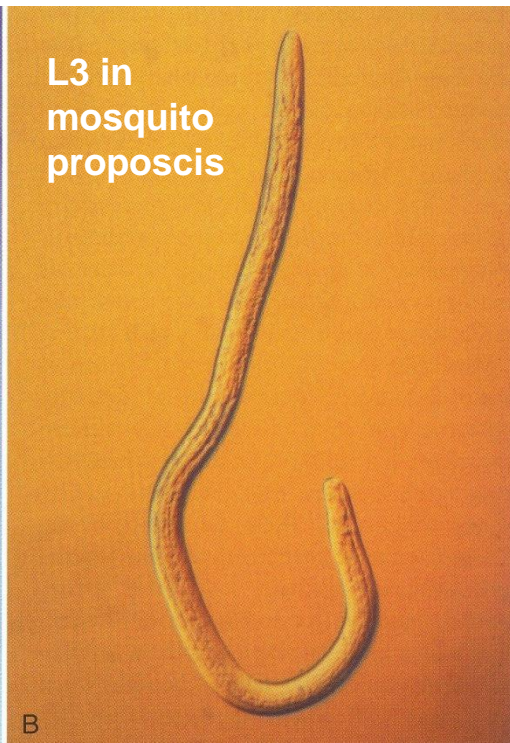


D. immitis



Micrifilaria
in blood

A



L3 in
mosquito
prothorax

B



Dirofilariosis in Northern Europe

Traveling pets to the South – Exposure to mosquitoes

Temporary transmission possible in summer

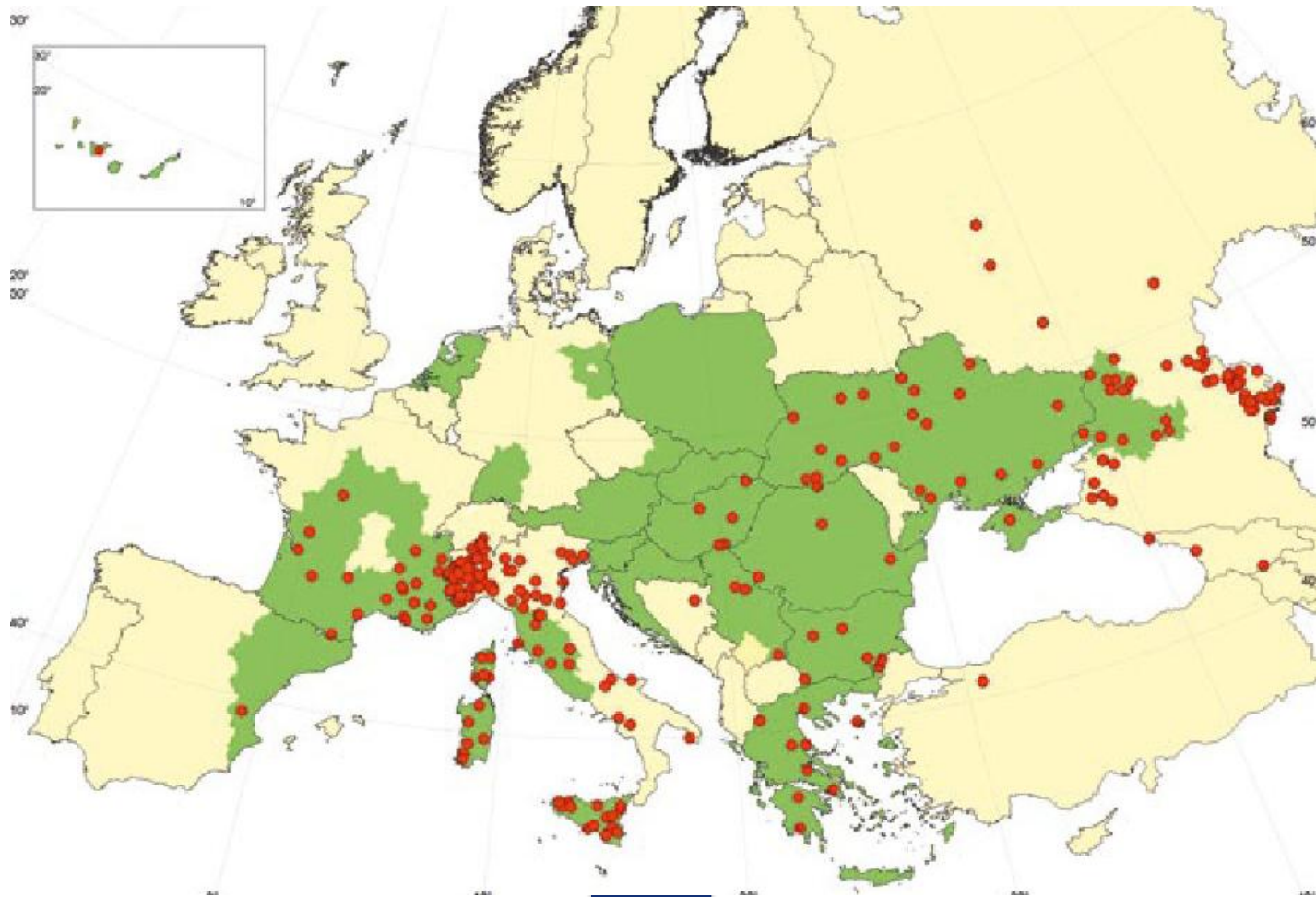
=> Endogenous cases

Often unnoticed infections:

- **in pets only adult *D. immitis* is pathogenic**
- **in humans: *larva migrans* (face and lungs)**

=> Poor passive surveillance

D. Repens in Europe: Pantchev et al (2011);
green = animal cases; red spots = human cases



Dirofilariosis: key epidemiological determinants

Presence of reservoir hosts (infected FH)

Presence of culicidae (low IH specificity)

Abundance of culicidae

Effect of temperature on the maturation of the larval stage

Effect of global warming (on parasite and IH)?

Effect of traveling?

Need for surveillance and prophylaxis?

Vector movements: the example of midges and bluetongue

Bluetongue: non-contagious viral disease of ruminants

Mostly asymptomatic infections

Clinical signs: oedema of lips, head and tongue

50/1500 midge species can transmit BTV

- **high abundance and feeding mostly on ruminants are required**
- ***C. imicola* group: Africa, Middle-East and Southern Europe – warm and dry areas**
- ***C. obsoletus* group: Northern Europe – cold and humid areas**



Effect of climate on disease transmission

Vector abundance

Extrinsic incubation period function of ambient temperature

Shorter feeding intervals with high temperatures

High temp on larvae -> increased competence of adults?

Overwintering

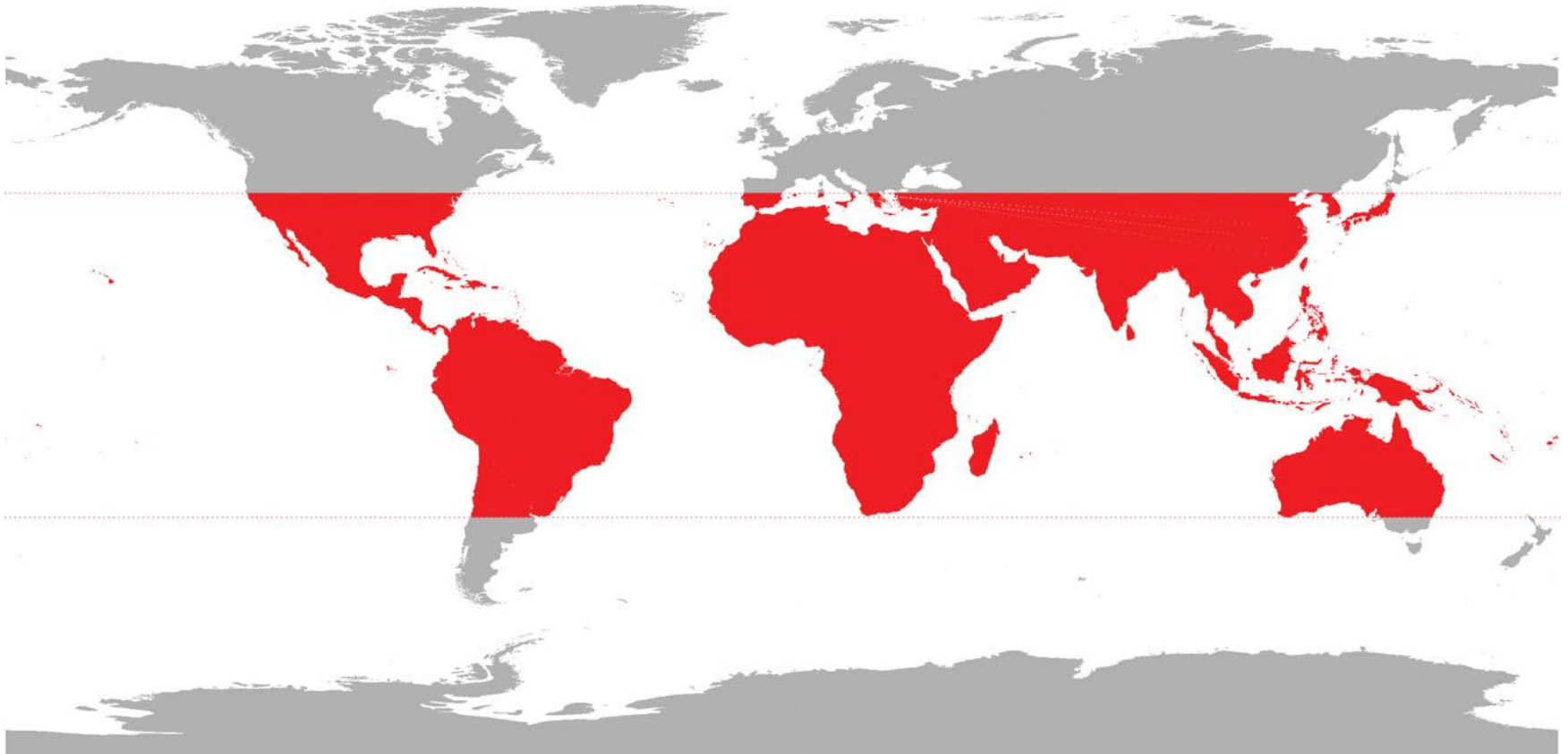
- **In midges?**
- **In ruminants?**
- **Other transmission routes during winter?**

BTV spread

Are recent BTV outbreaks caused caused by:

- **Movements of the vector?**
- **Movements of infected animals?**
- **Global warming?**
- **Combination of these?**

Bluetongue situation before 1998



Wilson and Mellor 2009

Transmission mechanisms

Movement of carrier animals

Active midge flights (1-2 km)

Transport of midges by wind

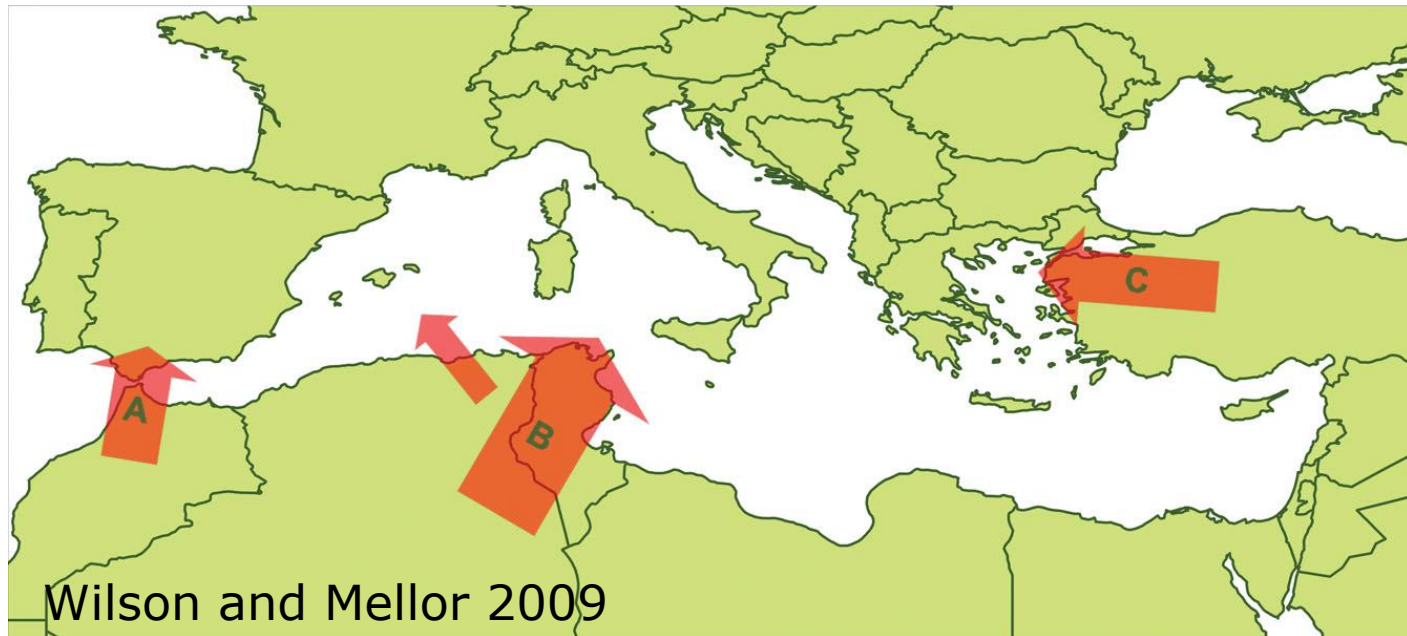
- **large distances (>100 km)**
- **crossing Mediterranean**
- **infection of islands**

Fully receptive populations -> outbreaks

Major introduction routes between 1998 and 2006

*Introduction of *C. imicola* through passive transport by wind*

Self-limited outbreaks restricted to Southern Europe



BT outbreak in Northern Europe in 2006

August 2006: outbreak in B, NL, D, L and Northern F

Exceptionally warm summer and autumn in 2006

Overwintering and further expansion in 2007: UK, D, Ch and Czech Republic

Vaccination using inactivated vaccine

Repeated introductions: BTV8 (2006), BTV6 (2008) and BTV11 (2009) from sub-Saharan Africa

Route still unknown but different from previously described routes

Bluetongue: conclusions

Important role of midges movements

Effect of temperature

Reservoir role of animals – herd immunity

Attenuated vaccine strains do circulate... Risk of recombination

Numerous questions remain unanswered

Passive and active surveillance of midges and BT

Other midge-borne viruses: horse sickness, Schmallenberg...

Adaptation pathogen-vector-host: the example of trypanosomosis

*Tsetse transmitted protozoan
Nagana in animals*

Sleeping sickness in humans

Different types of cycles:

- **Sylvatic**
- **Encroachment of domestic animals in wildlife areas**
- **Wildlife/domestic interface**
- **Domestic**



Transmissibility and virulence

Trypanosoma transmissibility to tsetse flies

- ***Trypanosoma virulence and parasitaemia***
- ***Fly receptivity (species, age, nutritional status...)***
- ***High trypanosoma abundance are detrimental to the flies***

Trypanosoma virulence

- **Wildlife trypanosomes are very pathogenic in domestic animals: high but short-lived parasitaemia**
- **Domestic trypanosomes are unlikely to develop in wildlife: too low parasitaemia**
- **Transmissibility seems equivalent in sylvatic and domestic trypanosomes (unlike virulence)**

Pathogen – vector – mammalian host: triangular relationship

Host resistance/tolerance against the pathogen

Host resistance/tolerance against the vector

Vector resistance against the pathogen

Burden of vectors on mammalian hosts

Pathogenicity of pathogens in mammalian hosts and vectors

=> *Selection for:*

- **most effective invasion or defence mechanisms**
- **maximised dissemination (balance between virulence and host survival)**

Importation of animal diseases

Conclusions in terms of risk

Infection reservoir:

Movement

- **Domestic animals**
- **Wild animals**
- **Vectors**

Climate

- **Wind – transport of pathogens or vectors**
- **Temperature and humidity**

Survival or development of the pathogen in the environment

Survival of its vector

Development of the pathogen within the vector

Importation of animal diseases

Conclusions in terms of surveillance

Early detection of first cases

Close monitoring of potential entry points

Passive surveillance:

- **Owners of livestock and companion animals**
- **Veterinary practitioners**
- **Abattoirs**

Wildlife reservoir

Monitoring of vectors



Thank you for your attention