

Colour Section



Plate 1 Non-human primates represent the origin of many important viral zoonoses, (See page 26).



Plate 2 Animal markets represent a risk factor for transmission of various viral zoonoses, e.g. SARS.
Source: Reuters/SCANPIX. (See page 32).

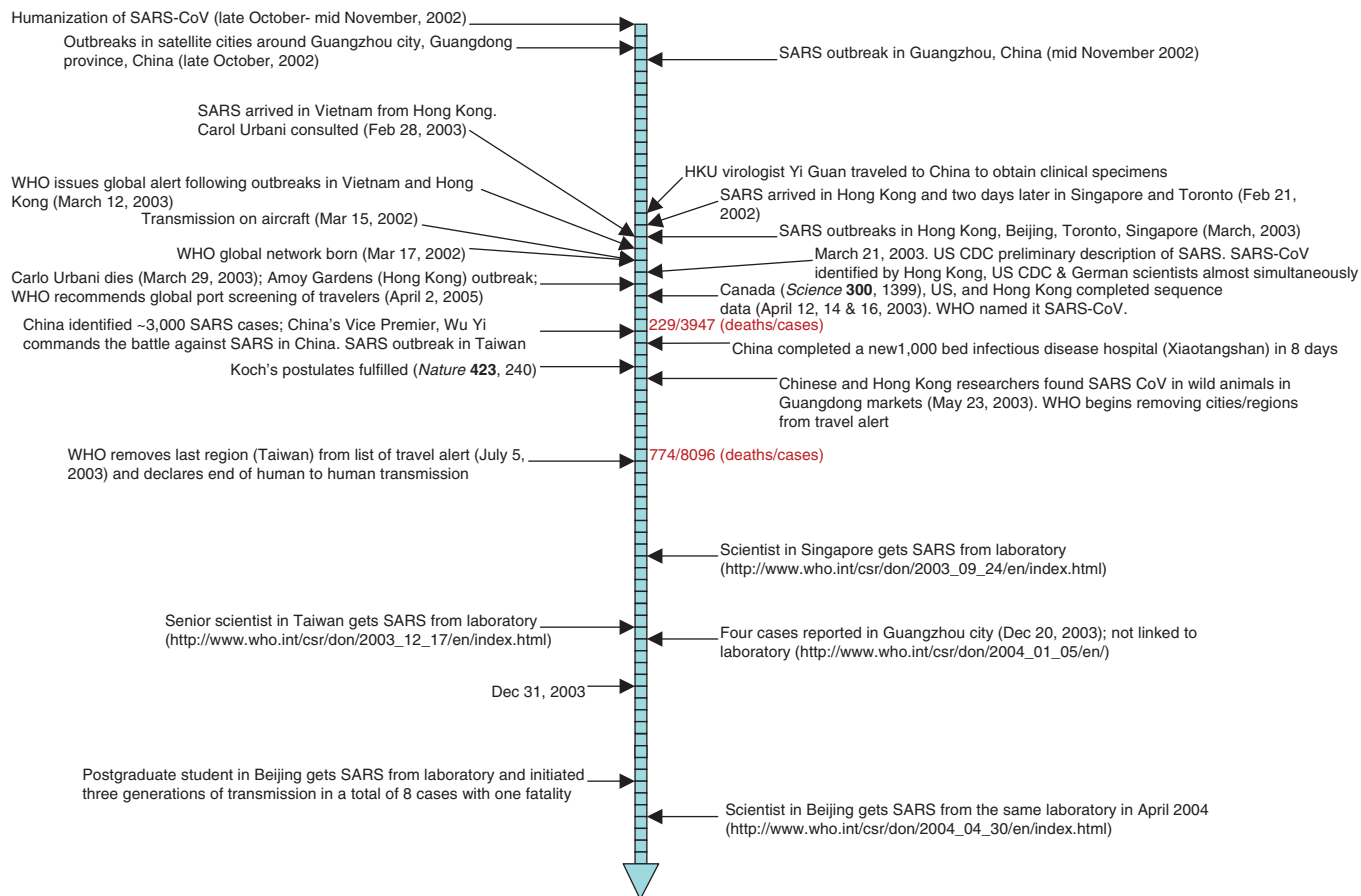


Plate 3 Timeline of the SARS epidemic. Major events are listed from top to bottom. Each interval in the arrow represents 1 week. For information on the first weeks of the epidemic in China, consult the book by Thomas Abraham, *Twenty-First Century Plague. The Story of SARS*. The Johns Hopkins University Press. Baltimore, Maryland, 2005. (See page 46).

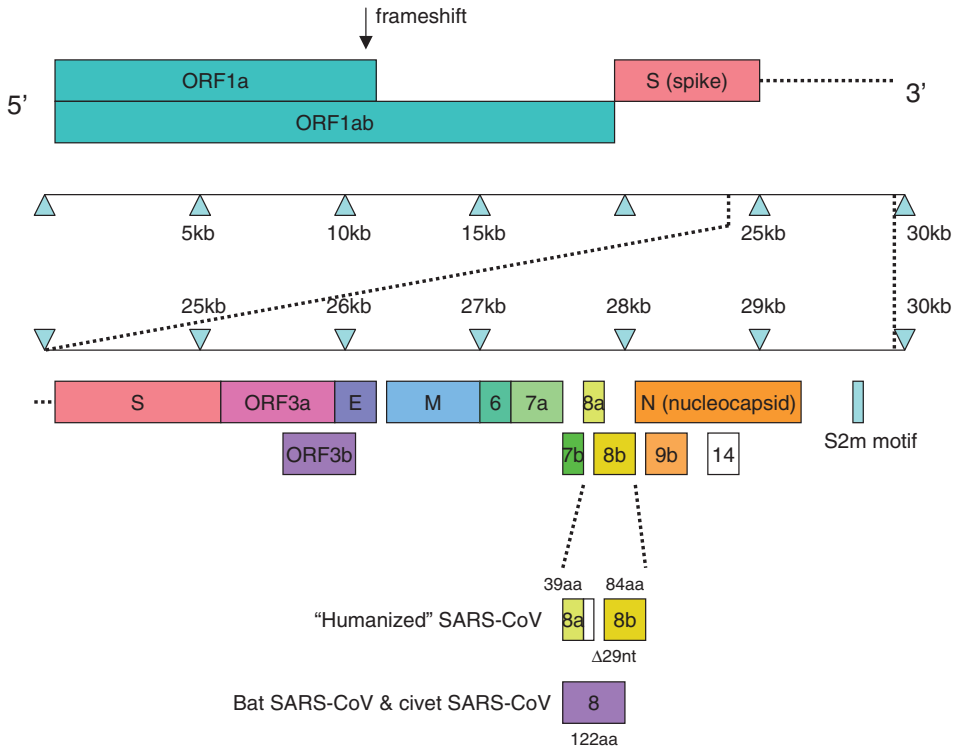


Plate 4 SARS-CoV genome organization. The genome organization is similar to other coronaviruses with respect to overall size, the relative positions of replicase, spike, envelope, membrane and nucleocapsid genes, and certain other features (see text for details). A 29-nucleotide stretch is deleted in the strain found in human isolates, as illustrated at the bottom. (See page 51).

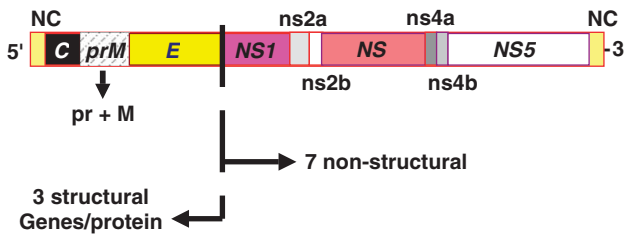


Plate 5 Genomic structure of WNV, showing 3 structural proteins, C-capsid, M-membrane, and E-envelope; and 7 nonstructural proteins (Petersen and Roehrig, 2001). (See page 134).

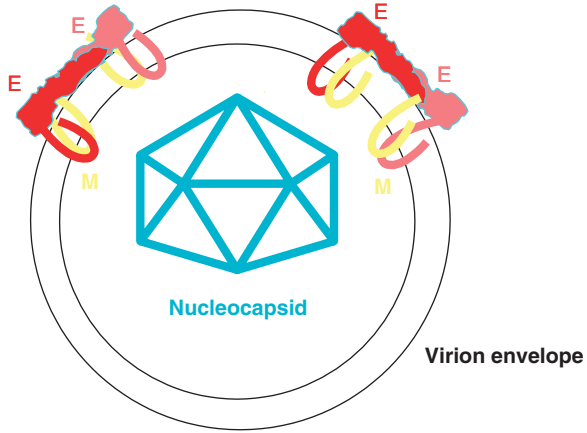


Plate 6 Flavivirus virion diagram. The single stranded RNA is enclosed in the nucleocapsid, which in turn is surrounded by an envelope containing E-glycoproteins (E) and integral membrane proteins (M) (Petersen and Roehrig, 2001). (See page 134).

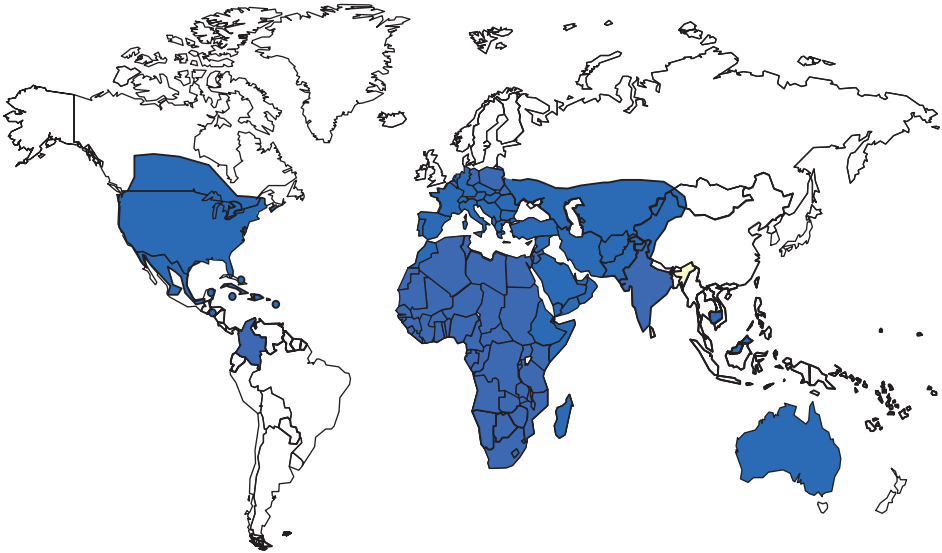
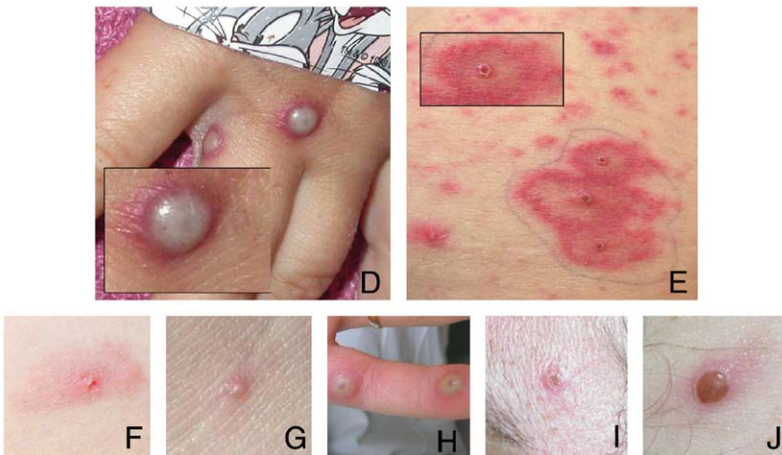


Plate 7 Approximate global distribution of West Nile virus. (See page 137).

Inoculation lesions



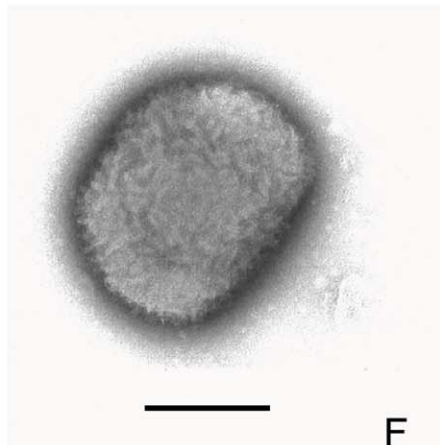
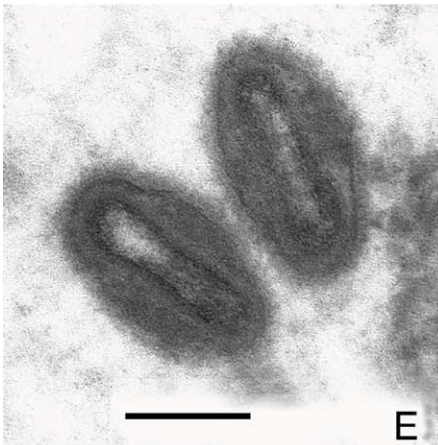
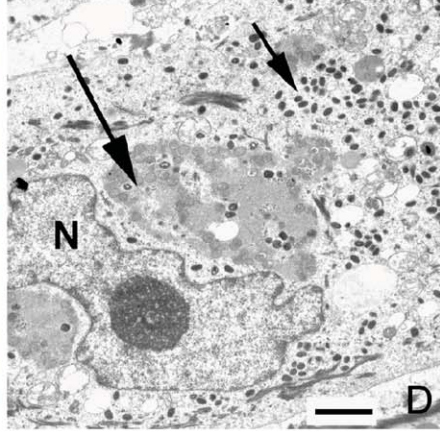
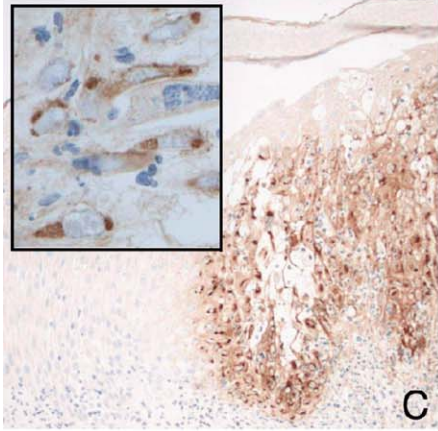
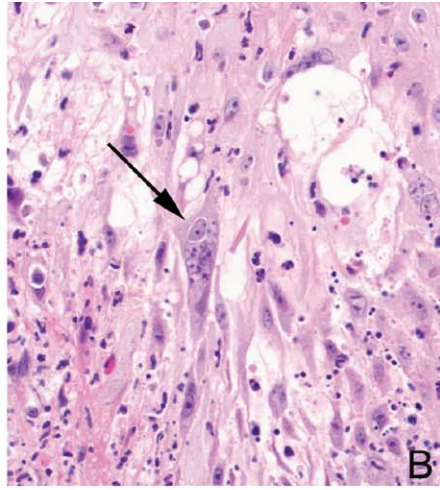
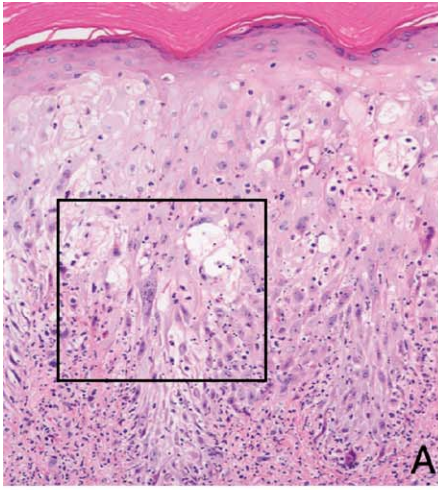
Dissemination lesions



Evolution of primary lesions



Plate 8 Cutaneous lesions of human monkeypox. The top panels show primary inoculation lesions at the site of a prairie dog bite (A) or scratch (B and C). The middle panels show the variation of the appearance of disseminated lesions of monkeypox ranging from smallpox-like (D) to varicella-like (E–J). The lower panels (K–M) document the progression of a primary lesion from the pustular stage through scarring. (See page 153).



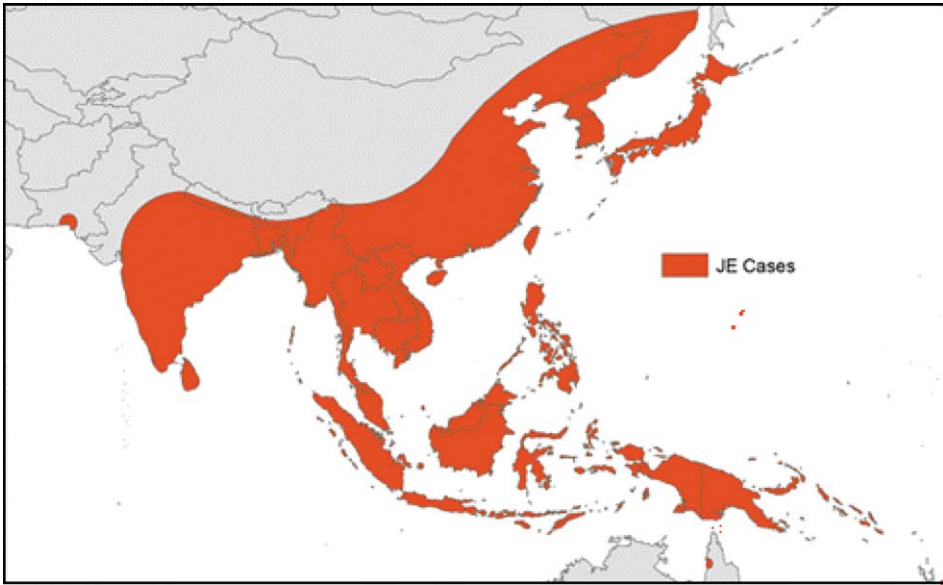


Plate 10 Map showing the distribution of JE cases (modified from CDC, Atlanta). (See page 215).

Plate 9 Histological, ultrastructural, and immunohistochemical appearance of MPV infection. Panel A: Scattered degenerating and necrotic keratinocytes are shown within the epidermis along with a moderate inflammatory cell infiltrate in the superficial dermis (hematoxylin and eosin). Panel B: Higher magnification of the boxed area shows multinucleated cells (long arrow) and eosinophilic viral inclusion bodies. Panel C: Strong immunoreactivity for orthopoxvirus antigen is present in the epidermis. Panel D: Transmission electron microscopy shows virions within the cytoplasm of a keratinocyte, including immature forms undergoing assembly (long arrow) and mature forms (short arrow). Panel E: High magnification shows the characteristic dumbbell-shaped inner core of poxviruses. Panel F: Negative staining of a virion from cell culture shows the brick-shaped particle with regularly spaced, threadlike ridges on the exposed surface. (See page 156).

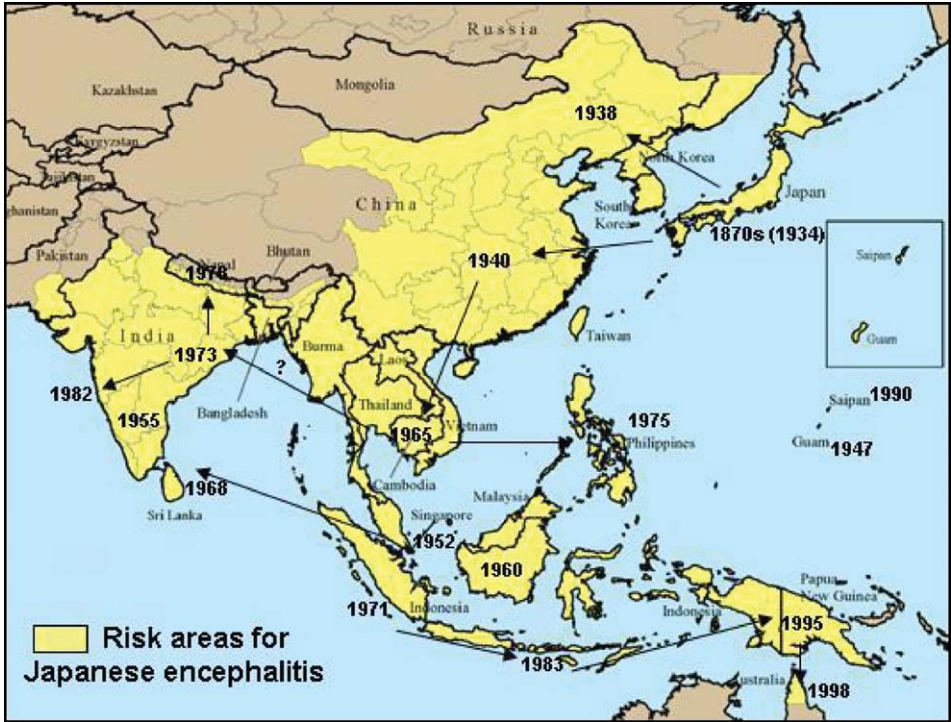


Plate 11 Map showing the temporal spread of JEV from the initial isolation in Japan. Modified from Solomon (2000). (See page 238).

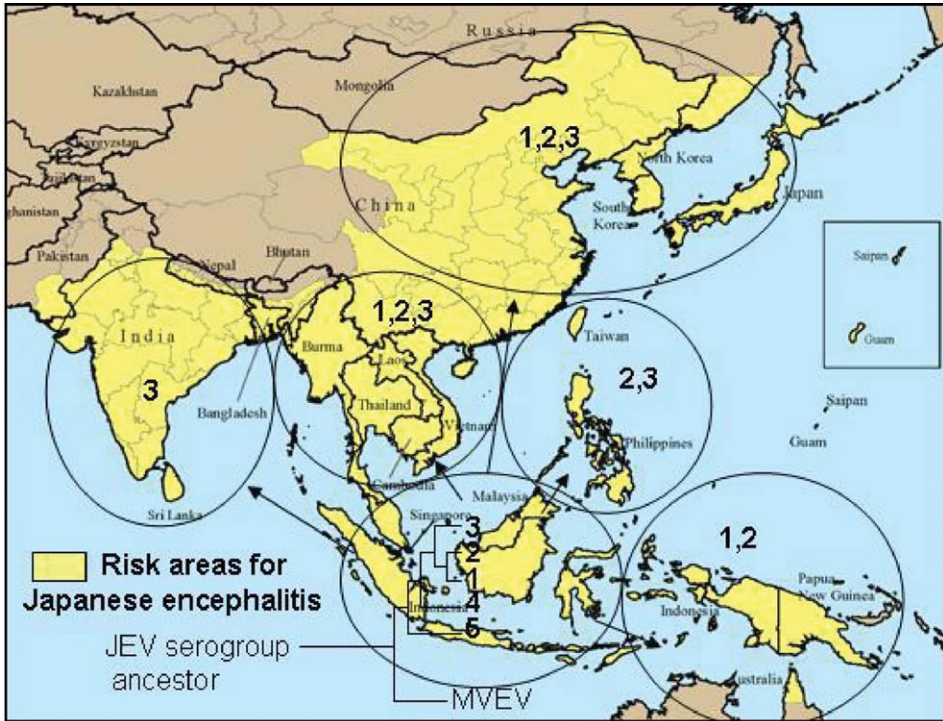


Plate 12 Map showing the possible origin and distribution of JEV, showing the geographic distribution of the genotypes. The ancestral virus is hypothesized to have given rise to genotype 4, and then to the other genotypes. A possible genotype 5, based on the Muar strain, would have been the earliest genotype if its sequence is confirmed. From Solomon and Winter (2004) and Solomon et al. (2003a). (See page 240).

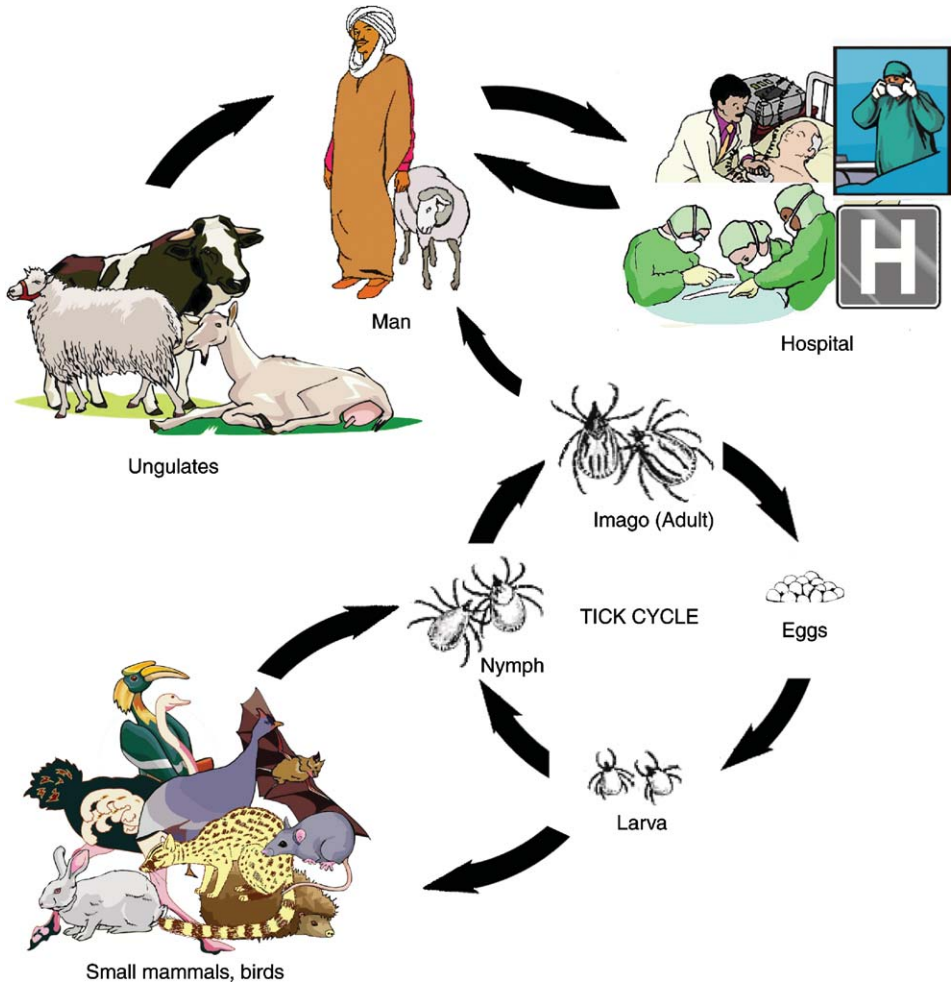


Plate 13 Example of CCHF virus circulation: transmission by the *Hyalomma marginatum rufipes* tick. (See page 307).

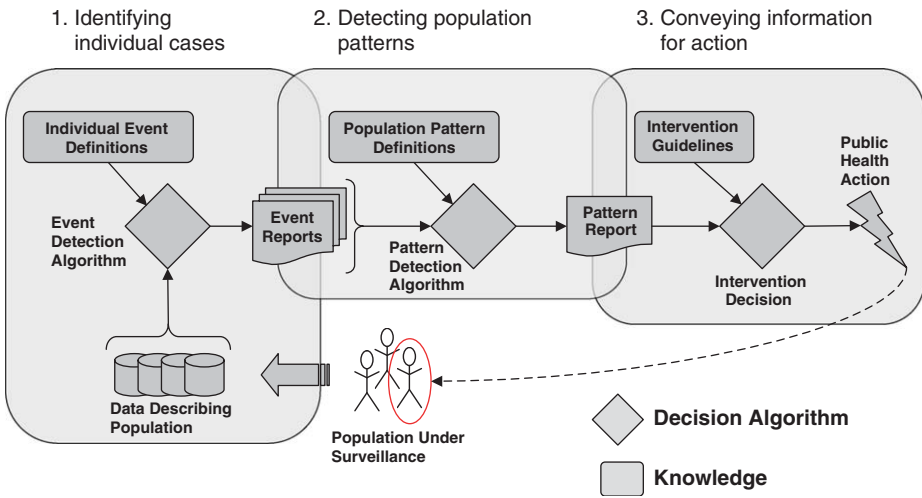


Plate 14 The process of surveillance. Critical points in this process include the detection of events in individuals (e.g., a diagnosis of measles), the identification of patterns in the population (e.g., a rapid rise in incidence in a geographic location), and the incorporation of information about identified patterns into decisions about interventions. (See page 330).