

Life-long shedding of Puumala hantavirus in wild bank voles

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Introduction

The knowledge of viral shedding patterns and viraemia in the reservoir host species is a key factor in assessing the human risk of zoonotic viruses. The shedding of hantaviruses (family *Bunyaviridae*) by their host rodents has widely been studied experimentally, but rarely in natural settings. Here we present the dynamics of Puumala (PUUV) hantavirus shedding and viraemia in naturally infected, wild bank voles (*Myodes glareolus*).

Materials & Methods

In a monthly capture-mark-recapture study, we analyzed 18 bank voles for the presence and relative quantity of PUUV RNA in the excreta and blood from 2 months before up to 8 months after the appearance of PUUV-specific antibodies (Figure 1).

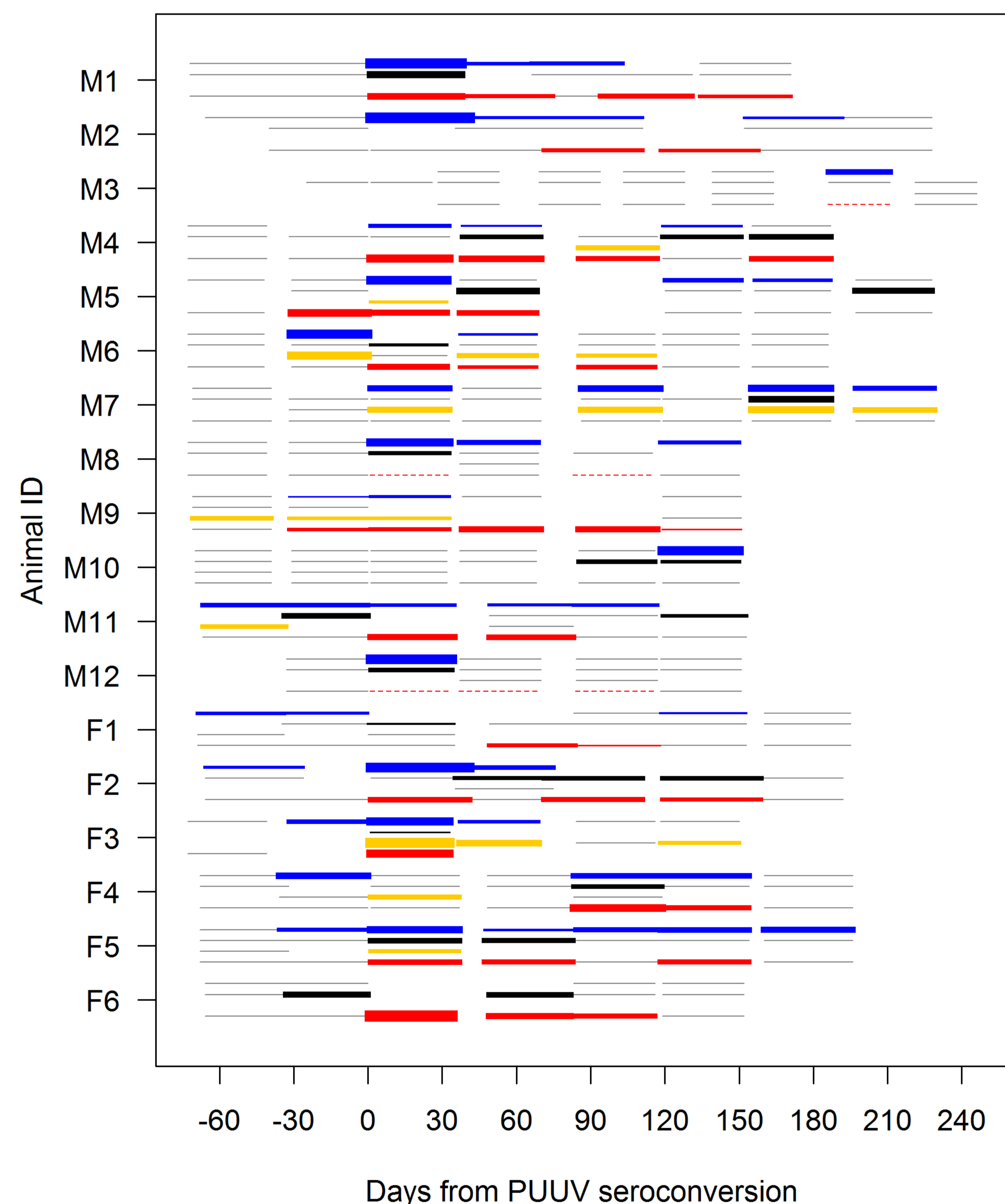


Figure 1. The presence and relative quantities of Puumala virus (PUUV) RNA in sequential samples of **saliva**, **faeces**, **urine**, and **blood** of individual bank voles in relation to the appearance of PUUV antibodies. The line width illustrates the log₁₀ relative quantity of PUUV RNA. **Gray** lines = PUUV RNA negative samples; **dashed** lines = RT-PCR positive samples without RNA quantitation; M=male; F=female.

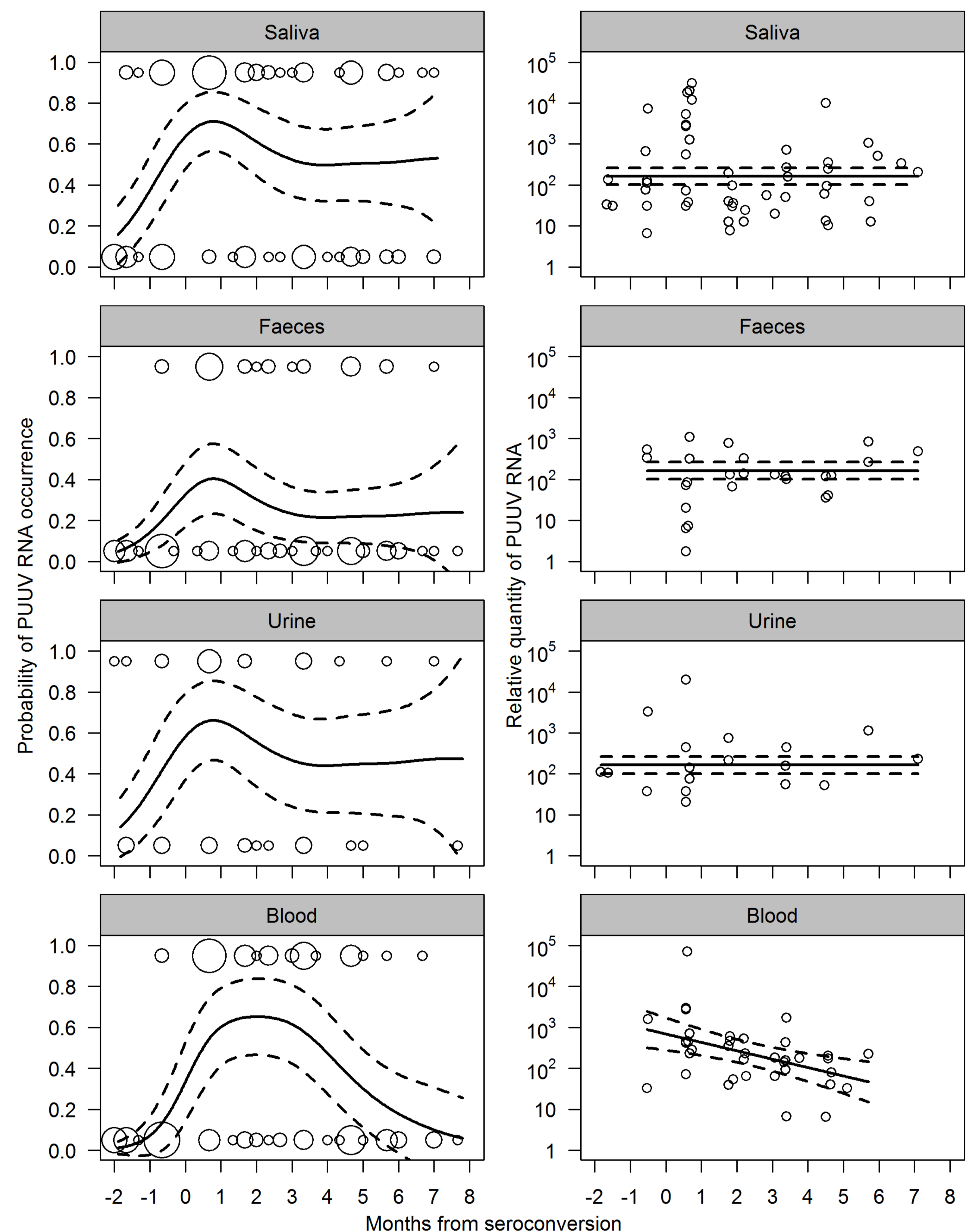


Figure 2. The probabilities of bank vole excreta and blood being Puumala virus (PUUV) RNA positive (left panel) and the relative PUUV RNA quantities (log₁₀-transformed) in RT-PCR positive samples (right panel) in relation to the time of seroconversion. The lines indicate predictions from best-supported statistical models with 95% confidence intervals. Circles denote the observed data. Circle sizes indicate the number of overlapping observations.

Results & Conclusions

The proportion of animals shedding PUUV RNA in saliva, urine, and faeces peaked during the first month after seroconversion, but continued for several months with only a slight decline (Figure 2). The quantity of shed PUUV in RT-PCR positive excreta was constant over time. In blood, PUUV RNA was present for up to 7 months but both the probability of viraemia and the virus load declined by time.

Our findings contradict the current view of a decline in virus shedding after the acute phase and a short viraemic period in hantavirus infection. We suggest the life-long shedding as a means for hantaviruses to survive over host population bottlenecks, and to disperse in fragmented habitats where local host and/or virus populations face temporary extinctions. Our results indicate that the kinetics of pathogens in wild hosts may considerably differ from those observed in laboratory settings.