7 List of figures

Figure 1	The life cycle of vaccinia virus
Figure 2	Proteins of IEV
Figure 3	An idealized scheme of in vitro actin polymerization
Figure 4	Actin polymerization in vivo
Figure 5	Life cycle of Listeria monocytogenes
Figure 6	Putative domain structure of the Listeria ActA, Shigella IcsA and rat N-WASP
Figure 7	Actin tail formation by Listeria and Shigella
Figure 8	Intracellular pathogens move on the tip of actin tails in infected cells
Figure 9	Localization of VASP in infected cells
Figure 10	Localization of Arp3, vinculin and N-WASP to actin tails of intracellular pathogens
Figure 11	Localization of phosphotyrosine epitopes in uninfected and infected HeLa cells
Figure 12	Micro-injection of anti-phosphotyrosine antibody into cells infected with vaccinia
	virus severly inhibits viral actin tail assembly
Figure 13	Role of IEV proteins in actin tail formation
Figure 14	Infection for 8 hours with vaccinia strains that do not make actin tails still induces
	disassembly of actin stress fibers
Figure 15	Vaccinia strains lacking the IEV specific proteins B5R and F13L are able to form
	actin tails at late stages of infection
Figure 16	Vaccinia virus strain A34R does not make IEVs
Figure 17	Vaccinia infection consistently induces tyrosine phosphorylation of three proteins
Figure 18	A36R is phosphorylated on tyrosine
Figure 19	Micro-injection of antibodies raised against IEV specific proteins reveals that A36R
	has a large cytoplasmic domain
Figure 20	A model of the topologies of IEV specific proteins
Figure 21	A schematic representation of the 'parental' construct used for the transfection assay
Figure 22	Transfection of A36R into cells infected with A36R rescues actin tail formation
Figure 23	Ectopic expression of A36R in A36R infected cells leads to overexpression of A36R
	and rescues phosphorylation of pTyr50
Figure 24	Western analysis of ectopically expressed A36R point mutants
Figure 25	Quantification of actin tail rescue by tyrosine to phenylalanine mutants of A36R
	reveals that only Y112 is required for efficient actin tail formation
Figure 26	Transfected A36R proteins localize to IEV
Figure 27	Alignement of the aminoacid sequence just C-terminal of A36R Y112 with the
	optimal peptide sequence required for binding to the SH2 domain of Nck
Figure 28	Localization of Nck to the tip of viral actin tails
Figure 29	Nck interacts directly with phosphorylated Y112 of A36R and the complex of these
	proteins recruits N-WASP from cell extracts

- Figure 30 Overexpression of the Nck-SH2 domain or an N-WASP construct lacking the WA domain inhibits actin tail formation in WR infected cells
- Figure 31 Alignment of the region around A36R Y112 and the optimal substrate sequence for phosphorylation of a peptide by c-Src
- Figure 32 The Src-family kinase inhibitor PP1 inhibits actin tail formation...
- Figure 33 PP1 does not inhibit viral morphogenesis
- Figure 34 Over-expression of "dead open" c-Src inhibits vaccinia actin tail formation
- Figure 35 Closer examination of infected cells expressing low levels of "dead open" c-Src reveals that the protein is recruited to viral particles that have induced actin tails
- Figure 36 PP1 and "dead open" c-Src reduce phosphorylation of A36R
- Figure 37 Cortactin is phosphorylated during vaccinia infection
- Figure 38 Localization of cortactin to actin tails of intracellular pathogen...
- Figure 39 Induction of tyrosine phosphorylation is dependent on viral entry...
- Figure 40 Blocking early but not late gene expression inhibits vaccinia induced loss of actin stress fibers and cell rounding
- Figure 41 Cartoon of the possible interactions of proteins involved in vaccinia actin tail formation
- Figure 42 Actin tails in the absence of infection