Supporting information

Figure S1. Intracellular behavior of *E. coli* strains in wt MEFs versus atg5-/- MEFs. Infections were performed with the non pathogenic *E. coli* K-12 strain MG1655, the environmental *E. coli* strain SMS 3.5, the commensal *E. coli* strain HS, enteropathogenic *E. coli* (EPEC) strain E2348/69 (D), the diffusely adhering *E. coli* (DAEC) strain C1845, the enterotoxigenic *E. coli* (ETEC) strain H10407, the enteroinvasive *E. coli* (EIEC) strain E12860/0, and the CD-associated adherent-invasive *E. coli* (AIEC) strain LF82. The numbers of intracellular bacteria were determined at various times post-infection ranging from 1 h to 6 h. Results are expressed as percentage of intracellular bacteria after various times of gentamycin treatment relative to that obtained at 1 h after gentamycin exposure, taken as 100% .

Figure S2. **Kinetics of the acquisition of lysosomal marker LAMP-1 and vacuole acidification in MEFs.** Experiments were performed with AIEC LF82-infected wt MEFs (black lines) and Atg5-/- MEFs (grey lines). MEFs were infected with GFP-expressing AIEC strain LF82 for 1 h at MOI of 10 and then incubated for 1 h, 2 h, 4 h or 6 h in cell culture medium containing gentamycin (80µg/mL). A. Labelling of LAMP-1 was performed using rat antibodies to the murine LAMP-1. B. AIEC LF82 containing vacuole acidification was assessed by using Lysotracker Red DND-99 as described in *Experimental procedure*. For each point, at least 100 AIEC LF82-containing vacuoles were counted and scored for the presence or absence of the markers. The results shown are the mean percentages of marker colocalization on AIEC LF82-containing phagosome ± standard error of the mean (SEM). At least three independent experiments were performed.

Figure S3. Representative confocal micrographs of AIEC LF82-GFP-infected wt MEFs or Atg5-/- MEFs at 6 h post-infection labelled free cytosolic AIEC LF82 with O83 antibodies (red) after permeabilization of plasma membrane with digitonin. Vacuoles were stained with LAMP-1 antibodies (purple).

Figure S4. Kinetics of the acquisition of lysosomal marker LAMP-1 (black lines) and vacuole acidification (grey lines) in AIEC LF82-infected Hela cells. Cells were infected with GFP-expressing AIEC strain LF82 for 3 h at MOI of 10 and then incubated for 1 h, 2 h, 4 h or 6 h in cell culture medium containing gentamycin (80µg/mL). Labelling of LAMP-1 was performed using mouse antibodies to the human LAMP-1. AIEC LF82 containing vacuole acidification was assessed by using Lysotracker Red DND-99 as described in *Experimental procedure*. For each point, at least 100 AIEC LF82 containing vacuoles were counted and scored for the presence or absence of the markers. The results shown are the mean percentages of marker colocalization on AIEC LF82-containing phagosome ± standard error of the mean (SEM). At least three independent experiments were performed.

Figure S5. Confocal analysis of percentage of colocalization of AIEC LF82-containing vacuoles with autophagic marker LC3 either in untreated cells (black bars) or in cells treated during 3 h with rapamycin (40µg/mL, white bars) to induce autophagy. Hela cells were infected with GFP-expressing AIEC strain LF82 for 3 h at MOI of 10 and then incubated in gentamycin containing medium. Cells were treated for additional 3 h with rapamycin at 1 h or 20 h post-infection. At least 100 AIEC LF82 containing vacuoles were counted and scored for the presence or absence of endogenous LC3 immunostaining. The results shown are the mean percentage of LC3 colocalization on AIEC LF82-containing phagosomes ± standard error of the mean (SEM). At least three independent experiments were performed.