TLR2 and RIP2 Pathways Mediate Autophagy of *Listeria monocytogenes* via ERK activation

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Figure S1



(A) BMDMs or (B) DCs were exposed to *L. monocytogenes* and the uptake of bacteria was determined by bacterial counts at 1h post-infection relative to input numbers.

Figure S2



(A) Quantification of GFP-LC3 positive *L. monocytogenes* wild-type strain in WT and Rip2^{-/-} at 4h post-infection.

(B) Western blots of LC3-II in wild-type (WT) and *Tlr2-/-Nod2-/-* macrophages that were uninfected (C) or infected with *L. monocytogenes* (*Lm*) for different periods. Actin was used as a loading control.

Figure S3



(A) Western blots of LC3-II in wild-type (WT) and *Nod2-/-* macrophages that were uninfected (C) or infected with *L. monocytogenes* (*Lm*) in the absence or presence of ERK-inhibitor PD98059 for different periods. Actin was used as a loading control.

(B) Intracellular growth curve of *L. monocytogenes* in Wild-type and *Nod2*^{-/-} macrophages in the absence or presence of ERK-inhibitor PD98059 (* $p \le 0.05$).