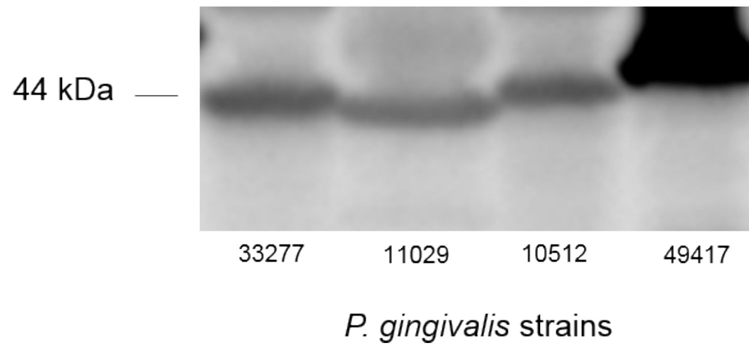

Figure S1. Immunoblot of whole cell lysates of *P. gingivalis* strains probed with polyclonal antibodies to the FimA protein of strain 33277.

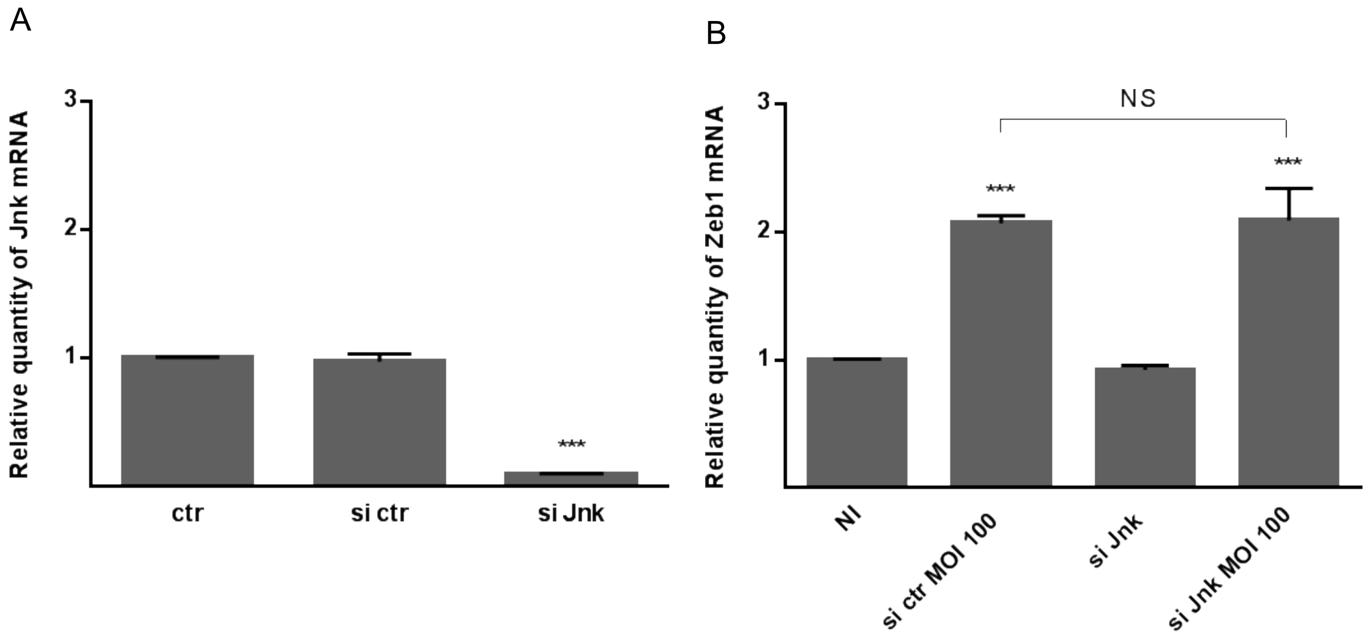

Figure S2. JNK knockdown does not affect regulation of Zeb1 by *P. gingivalis.*
A. TIGK cells were transiently transfected with siRNA to JNK1/2 (si Jnk, 100 nM), Sigma) or scrambled siRNA (si ctr). Control (ctr) cells were nontransfected. JNK mRNA levels in transfected cells were measured by qRT-PCR. Data were normalized to GAPDH mRNA and expressed relative to ctr. Results are means ± SD, n = 3; *** $P < 0.001$;
B. Transfected TIGK cells were infected with *P. gingivalis* 33277 for 24 h at MOI 100. ZEB1 mRNA was measured by qRT-PCR, the data were normalized to GAPDH mRNA and are expressed reletive to the noninfected (NI) control. Results are means ± SD, n = 3; *** $P < 0.001$ compared to NI; NS: not significant.

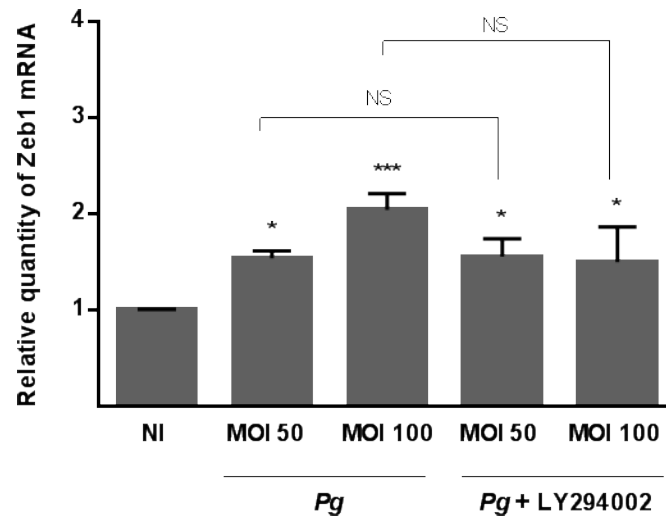

Figure S3. Pharmacological inhibition of Akt does not affect regulation of Zeb1 by *P. gingivalis.*

TIGK cells were preincubated with 10 μM LY294002 or vehicle (DMSO) only for 1 h and infected with *P. gingivalis* 33277 MOI 50 or 100 for 6 h. Zeb1 mRNA levels were measured by qRT-PCR, normalized to GAPDH mRNA and expressed relative to noninfected (NI) controls. Results are means ± SD, n = 3; * $P < 0.05$; *** $P < 0.001$; NS: not significant.

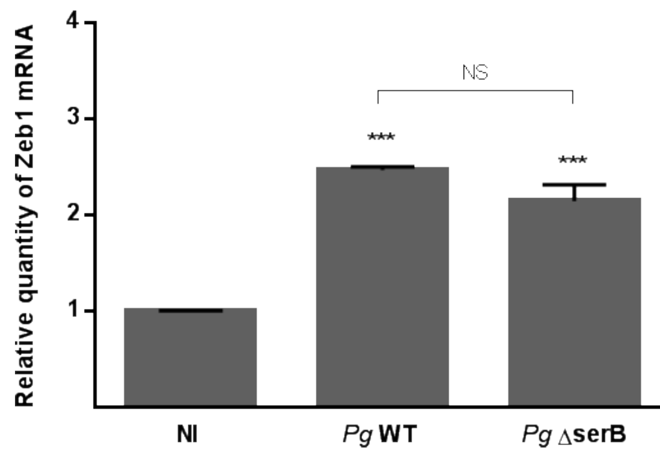

Figure S4. A non-invasive mutant of *P. gingivalis* can induce ZEB1 expression.
qRT-PCR of ZEB1 mRNA expression in TIGK cells infected with *P. gingivalis* 33277 (Pg WT) or a Δ*serB* mutant. Data were normalized to GAPDH mRNA and are expressed relative to noninfected (NI) controls. Results are means ± SD, n = 3; *** $P < 0.001$ compared to NI; NS: not significant.

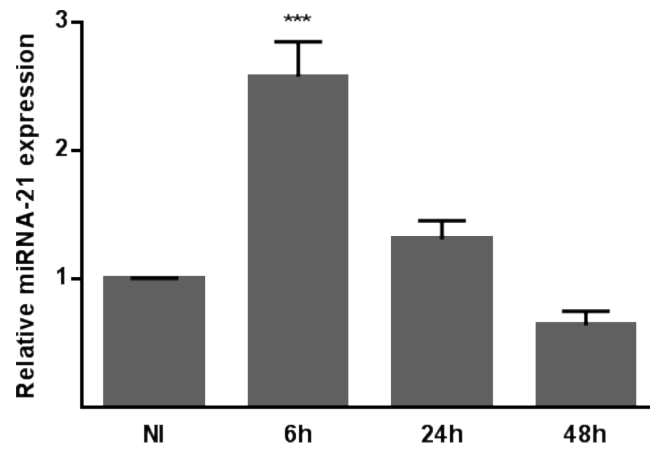

Figure S5. Expression of miRNA-21 is not down-regulated by *P. gingivalis*.
TIGK cells were infected with *P. gingivalis* 33277 (Pg) at MOI 100 for the time indicated. miRNA levels were measured by qRT-PCR, normalized to RNU48 miRNA, and expressed relative to noninfected (NI) controls. Results are means ± SD, n = 3;
*** $P < 0.001$ compared to NI.

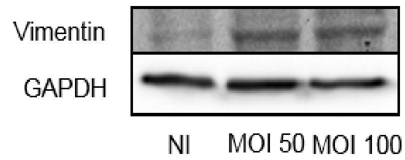

Figure S6. *P. gingivalis* increases expression of vimentin.
Immunoblot of lysates of TIGK cells infected with *P. gingivalis* 33277 for 24 h at the MOI indicated. Control cells were uninfected (NI). Duplicate blots were probed with antibodies to vimentin or GAPDH (loading control).

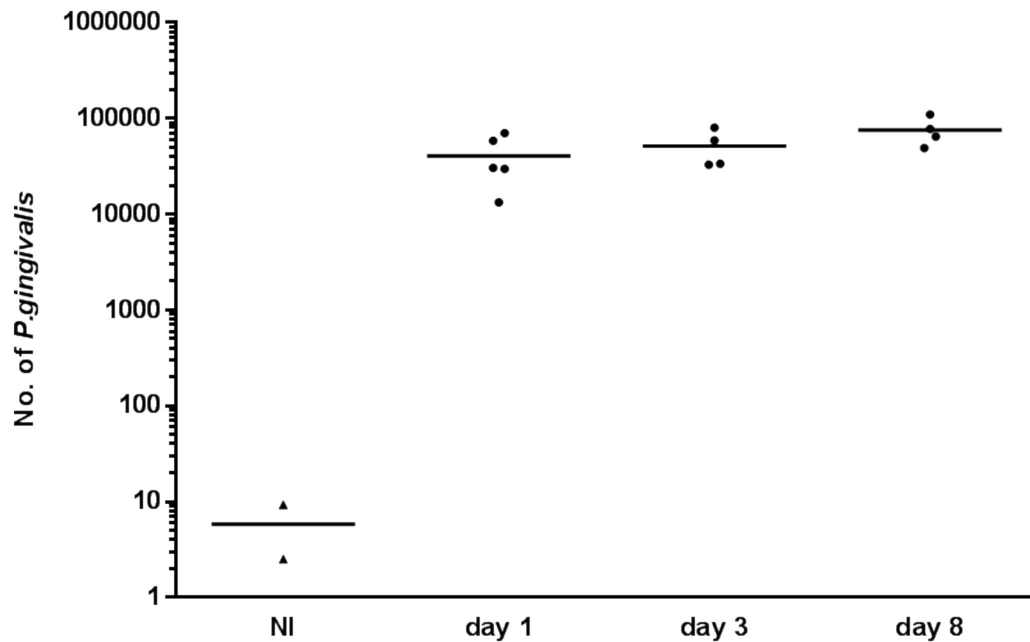

Figure S7. Colonization of mice. Mice were orally infected with 10^7 cfu *P. gingivalis* five times at 2-days intervals. Bacterial samples were collected along the gingiva of the upper molars. Samples were lysed, DNA extracted and qPCR performed with primers specific for *P. gingivalis* 16S DNA. For enumeration, genomic DNA was isolated from laboratory cultures of *P. gingivalis* 33277 (numbers determined by viable counting) and a series of dilutions prepared. The number of gene copies in the oral samples was determined by comparison with the standard curve. In the sham infected animals, 2 of 5 mice were colonized with low levels of organisms sufficient similar to *P. gingivalis* to give a positive result. *P. gingivalis* levels from day 1, 3 and 8 were statistically greater than sham infected ($P < 0.0001$) but were not statistically different from each other.

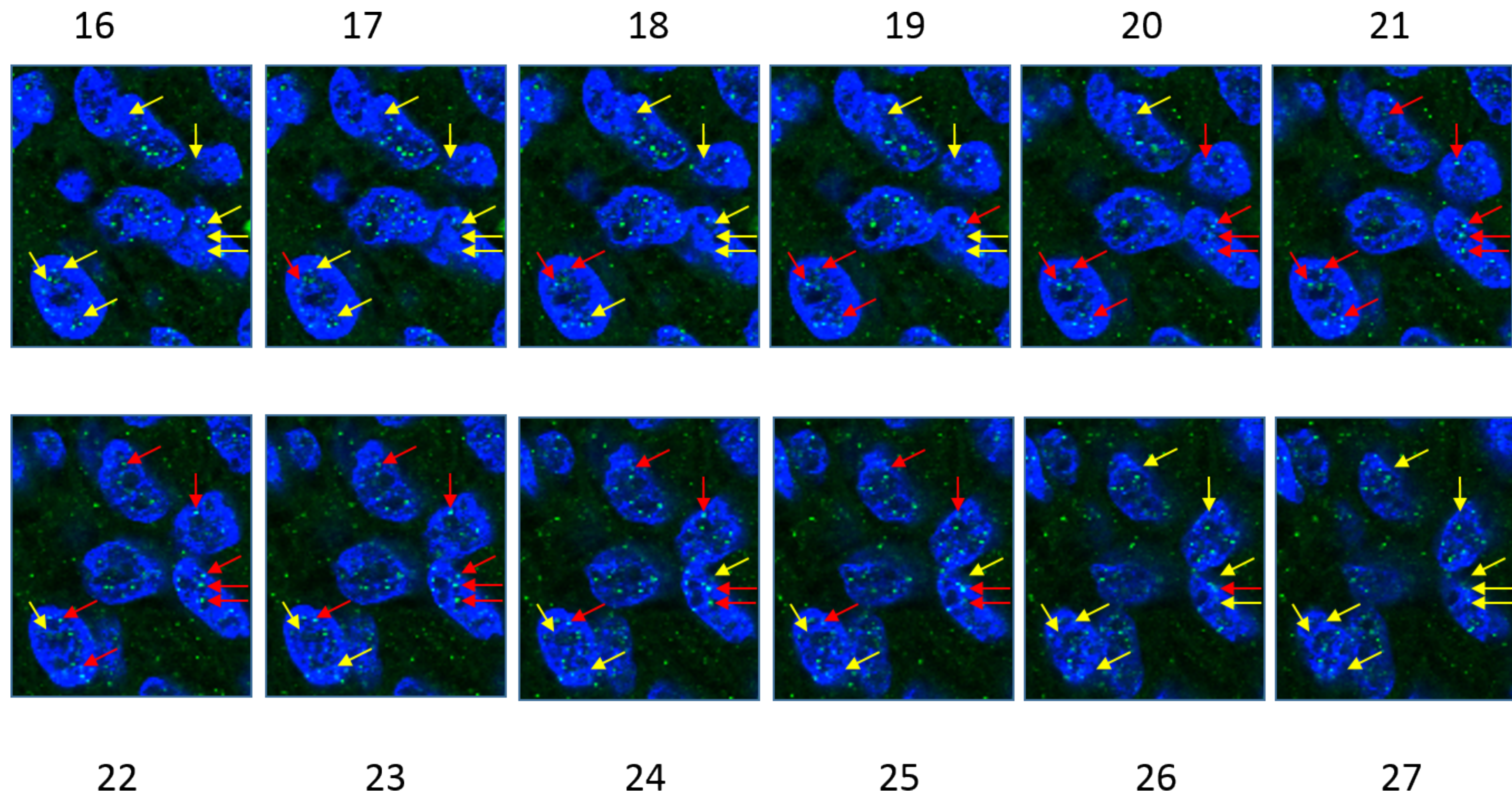

Figure S8. Fluorescent confocal microscopy of a carcinoma in situ biopsy sample probed with *P. gingivalis* antibodies (green) and stained with DAPI (blue). Cells were imaged at magnification x63. Red arrows point to a discrete fluorescent spot, yellow arrows indicate the same position where that spot is absent. Numbers are the slice number in an optical stack of 40 slices at 0.4 μm. Fluorescent spots are present in typically 5 to 7 adjacent optical slices (0.4 μm slices), indicating that the fluorescent particles are about 2.0 to 2.8 μm in size, consistent with the size of *P. gingivalis*.