Supplemental Table 1. Bacterial Strains			
Descriptive name	**Genotype**	**Strain Number**	**Source**
CO92 Lux_{PcysZK}	CO92 pCD1+, pgm+, pMT+, pst+, $Luxp_{cysZK}$	MBLYP043	(1)
CO92 T3+	MBLYP043 pgm-	YPA143	This work
CO92 T3-	CO92 pCD1-, pgm+, pMT+, pst+, Lux_{PtolC}	YPA050	(2)
KIM T3+	KIM1001 pCD1+, pgm-, pMT+, pst+	JG150A	(3)
KIM T3-	KIM1001 pCD1-, pgm-	JG152B	This work
KIM T3E-	KIM1001 pCD1+ (*yopH*$^{\Delta 3\text{-}467}$ *yopE*$^{\Delta 40\text{-}197}$ *yopK*$^{\Delta 4\text{-}181}$ *yopM*$^{\Delta 3\text{-}408}$ *ypkA*$^{\Delta 3\text{-}731}$ *yopJ*$^{\Delta 4\text{-}288}$ *yopT*$^{\Delta 3\text{-}320)}$), pgm-, pMT1+, pPCP1+	JG714	This work
+A	JG917::+*ypkA*	JG730	(3)
+E	JG917::+*yopE*	JG733	(3)
+H	JG917::+*yopH*	JG734	(3)
+J	JG917::+*yopJ*	JG735	(3)
+K	JG917::+*yopK*	JG736	(3)
+M	JG917::+*yopM*	JG732	(3)
+T	JG917::+*yopT*	JG708	(3)
ΔA	JG150A Δ*ypkA*	JG593	(3)
ΔE	JG150A Δ*yopE*	JG517	(3)
ΔH	JG150A Δ*yopH*	JG589	(3)
ΔJ	JG150A Δ*yopJ*	JG525	(3)
ΔK	JG150A Δ*yopK*	JG523	(3)
ΔM	JG150A Δ*yopM*	JG583	(3)
ΔT	JG150A Δ*yopT*	JG713	(3)

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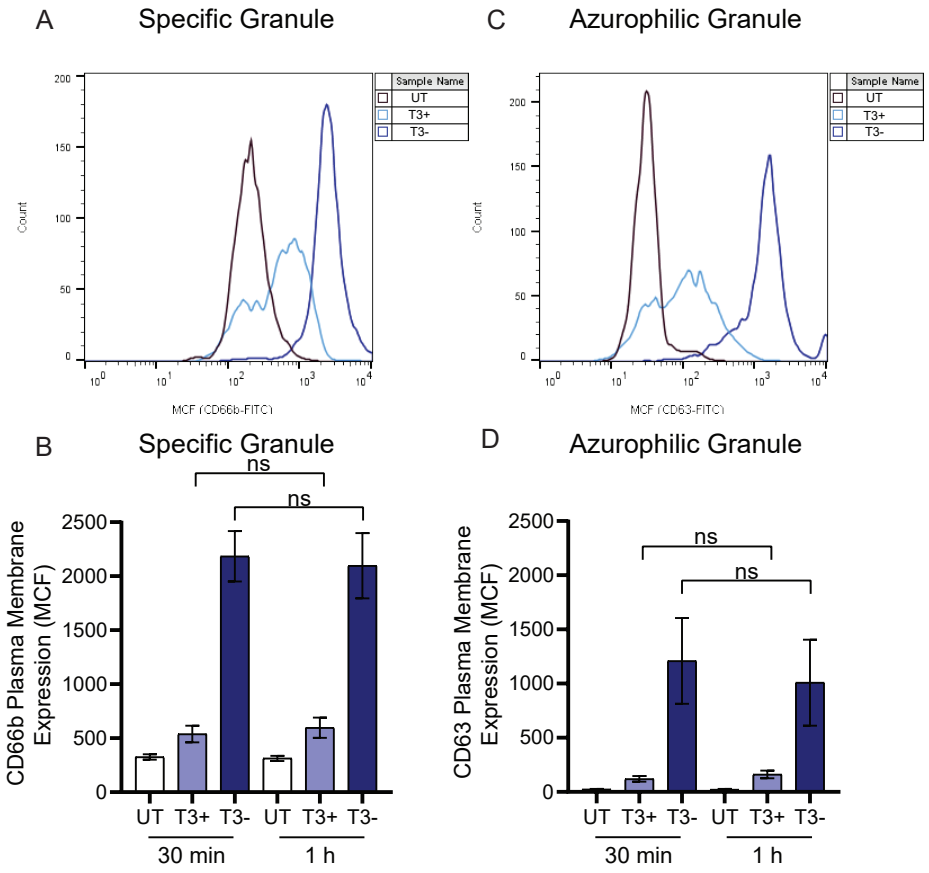
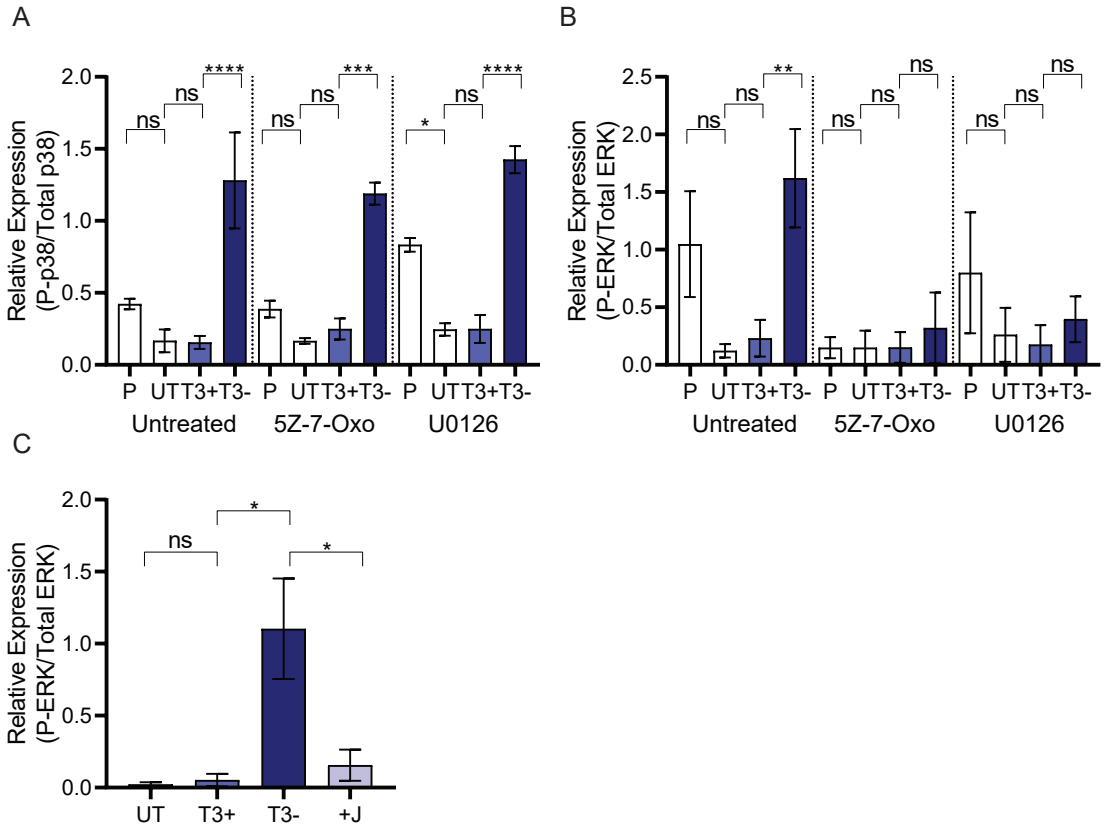
3. Palace SG, Proulx MK, Szabady RL, Goguen JD. 2018. Gain of Function Analysis Reveals Important Virulence Roles for the *Yersinia pestis* Type III Secretion System Effectors YopJ, YopT, and YpkA. Infect Immun doi:10.1128/iai.00318-18.


A
Specific Granule
C
Azurophilic Granule
Sample Name
UT
T3+
T3-
Count
MCF (CD66b-FITC)
MCF (CD63-FITC)
B
Specific Granule
D
Azurophilic Granule
CD66b Plasma Membrane Expression (MCF)
CD63 Plasma Membrane Expression (MCF)
ns
UT T3+ T3- UT T3+ T3-
30 min
1 h

Sup. Fig. 1

Supplemental Figure 1. Degranulation of specific and azurophilic granules peaks by 30 min post-infection. $4x10^6$ human neutrophils were infected with *Y. pestis* CO92 with or without the pCD1 plasmid encoding the T3SS (T3+ or T3-, respectively); MOI of 100. Degranulation of (A and B) specific and (C and D) azurophilic granules was measured 30 min and 1 h post-infection by flow cytometer. (A) and (C) Representative histogram for one experiment from (B) and (D) respectively. For (B) and (D) Mean ±SEM from 4 biologically independent experiments. One-way ANOVA with Sidak's post-hoc; ns = not significant. UT = untreated cells.


A
Relative Expression (P-p38/Total p38)
2.0
1.5
1.0
0.5
0.0
ns
ns
****
ns
ns
***
ns
ns
****
*
P UT T3+ T3- P UT T3+ T3- P UT T3+ T3-
Untreated
5Z-7-Oxo
U0126
B
Relative Expression (P-ERK/Total ERK)
2.5
2.0
1.5
1.0
0.5
0.0
ns
ns
**
ns
ns
ns
ns
ns
ns
P UT T3+ T3- P UT T3+ T3- P UT T3+ T3-
Untreated
5Z-7-Oxo
U0126
C
Relative Expression (P-ERK/Total ERK)
2.0
1.5
1.0
0.5
0.0
ns
*
*
UT
T3+
T3-
+J

Sup. Fig. 2

Supplemental Figure 2. Quantification of p38 and ERK phosphorylation. $8x10^6$ human neutrophils were infected with *Y. pestis* KIM1001 with or without the pCD1 plasmid encoding the T3SS (T3+ or T3-, respectively); MOI = 100. (A) Mean relative expression phosphorylated p38 or (B) phosphorylated ERK during infection with indicated strains after pre-treatment with vehicle control (Untreated), the TAK1 inhibitor (5Z)-7-Oxozeaenol ((5Z)-7-Oxo), or the ERK inhibitor U0126. (C) Mean relative expression of phosphorylated ERK during infection with indicated *Y. pestis* strains. *Y. pestis* KIM1001 = T3+; KIM1001 $T3^{(-)}$ = T3-; KIM1001 expressing only *yopJ* = +J; Uninfected = UT. Mean ±SEM from 3 biologically independent experiments. One-way ANOVA with Sidak's post-hoc; ns= not significant, *=$p<0.05$.