

## Supporting Information Appendix:

### Ornithine Decarboxylase Regulates M1 Macrophage Activation and Mucosal Inflammation via Chromatin Modifications

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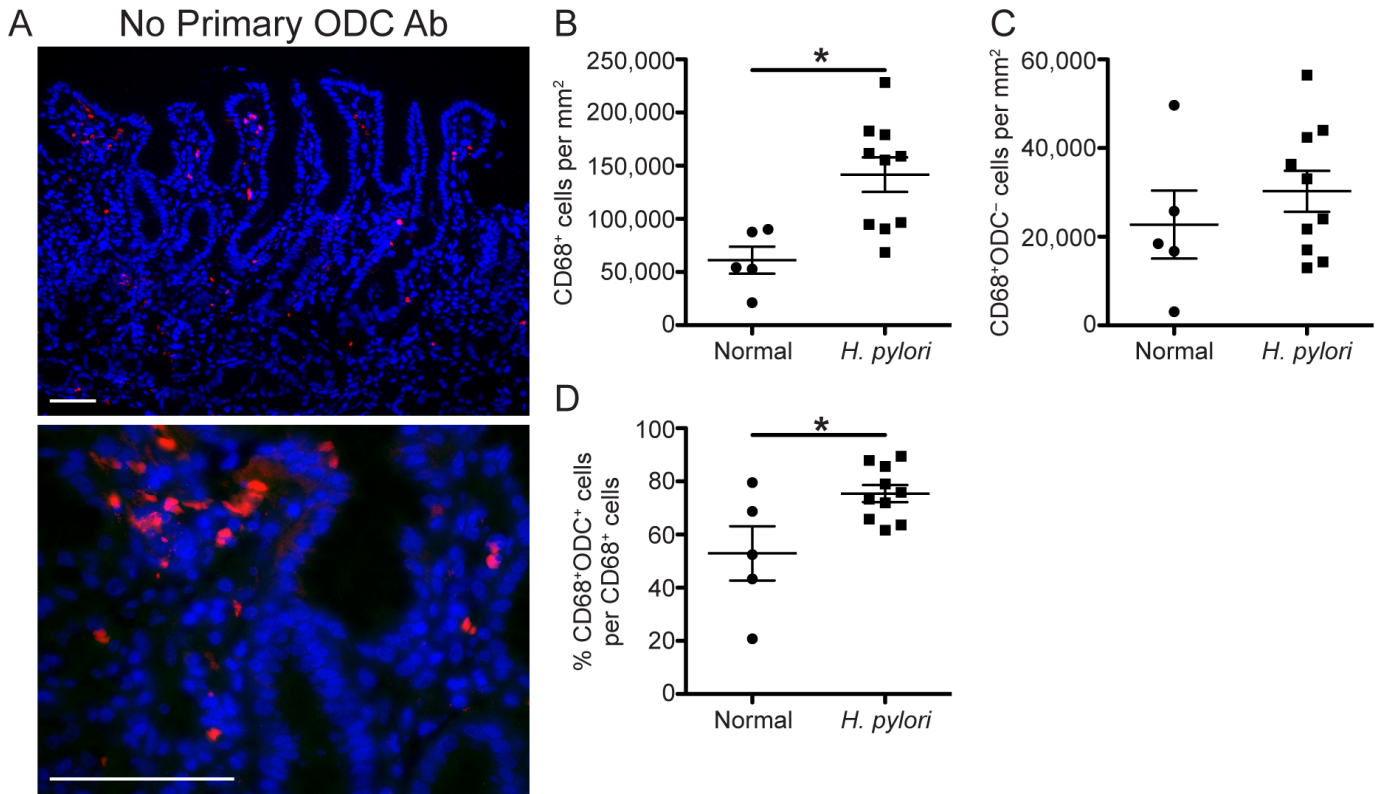
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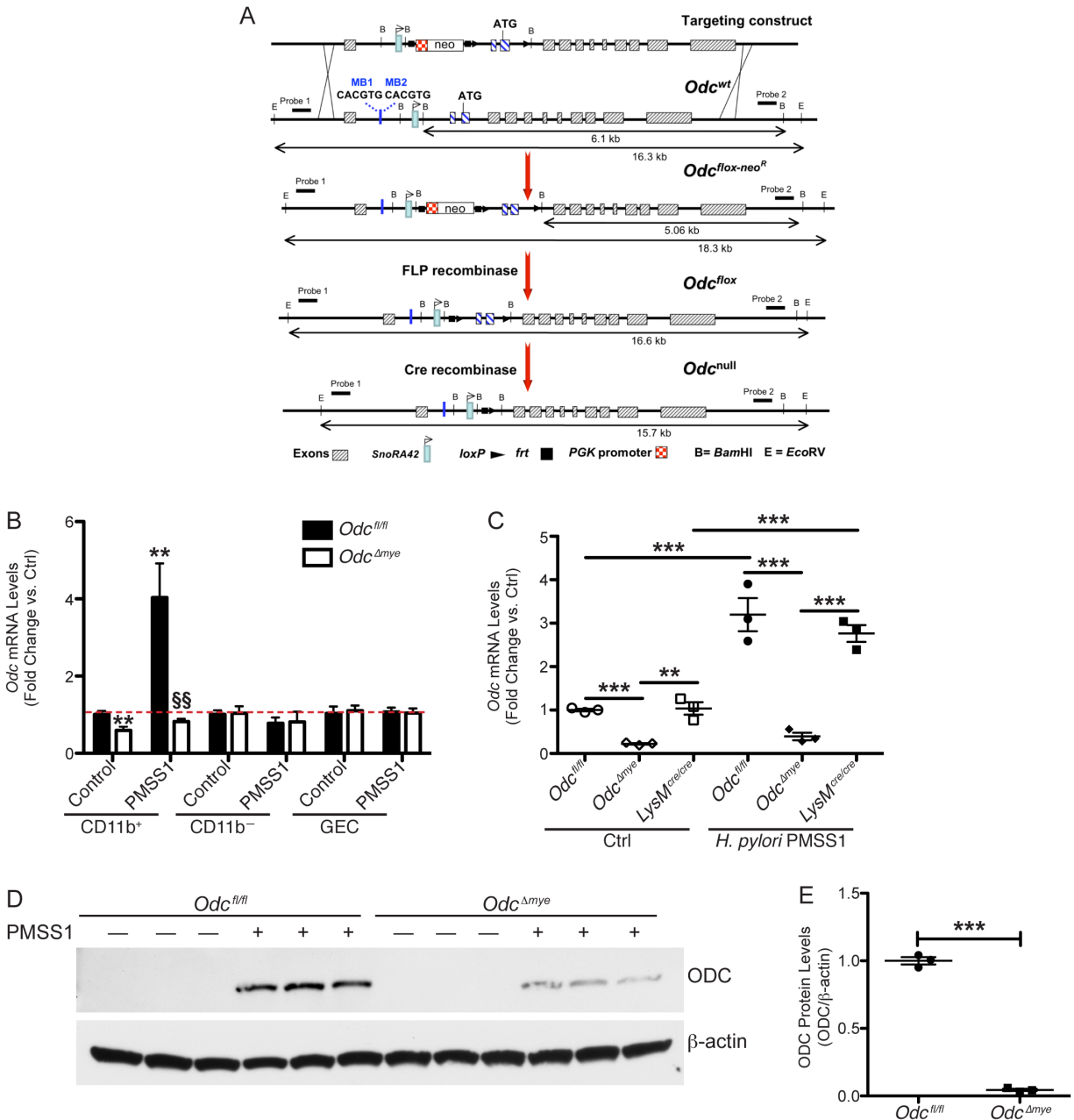
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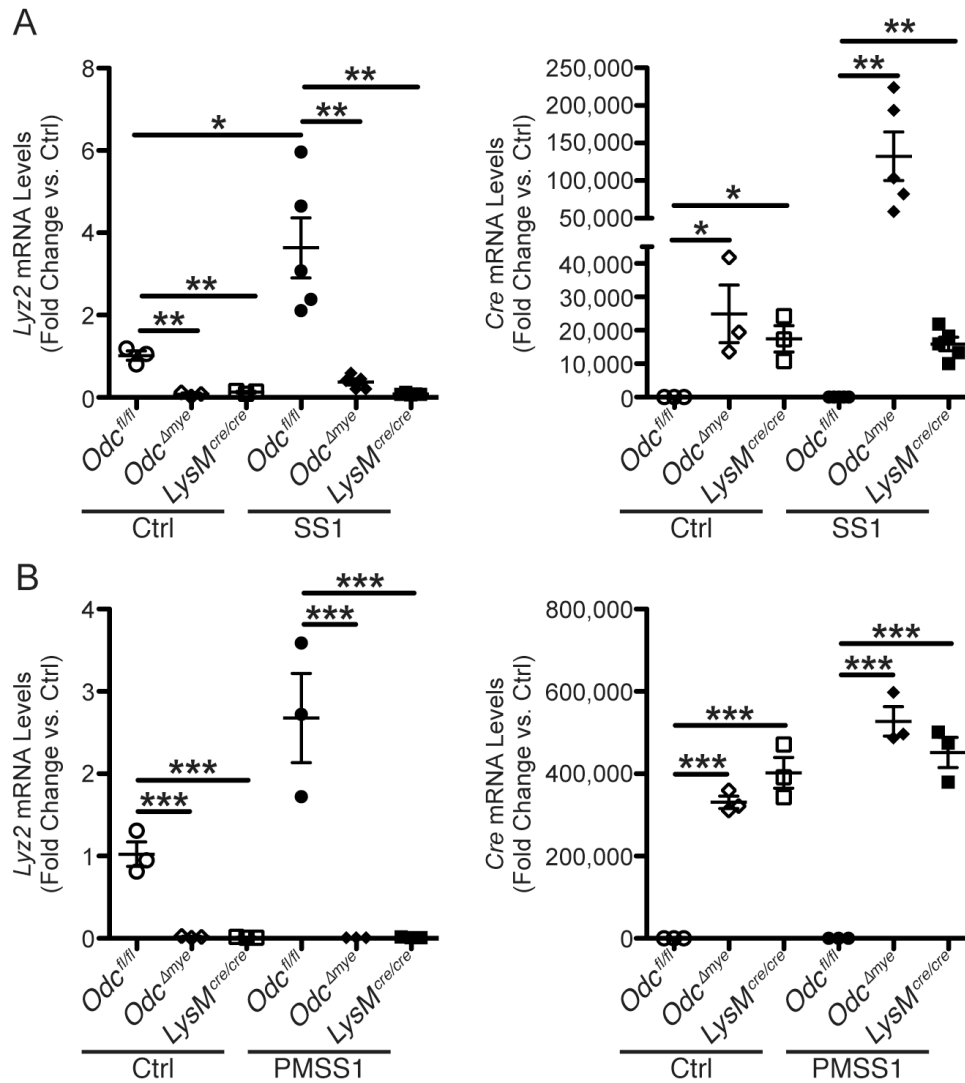
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**Figure S1. Immunofluorescence staining for CD68 and ODC in human gastric biopsies.** (a) Representative images of staining where no primary anti-ODC antibody was utilized, in order to confirm specificity of the ODC antibody. Note that the case utilized is the same *H. pylori*<sup>+</sup> case as in Figure 1c. Scale bar = 50  $\mu\text{m}$ . (b) Number of CD68<sup>+</sup> macrophages per tissue area ( $\text{mm}^2$ ) as determined by the average counts of 4 blinded observers. \* $P < 0.05$  by Student's *t* test.  $n = 5$  normal and 10 *H. pylori*<sup>+</sup> cases. (c) Number of CD68<sup>+</sup>ODC<sup>-</sup> macrophages per tissue area ( $\text{mm}^2$ ) as determined in (b).  $n = 5$  normal and 10 *H. pylori*<sup>+</sup> cases. (d) Percentage of CD68<sup>+</sup>ODC<sup>+</sup> macrophages per the total number of CD68<sup>+</sup> macrophages as determined in (b). \* $P < 0.05$  by Student's *t* test.  $n = 5$  normal and 10 *H. pylori*<sup>+</sup> cases. Data displayed as mean  $\pm$  S.E.M.

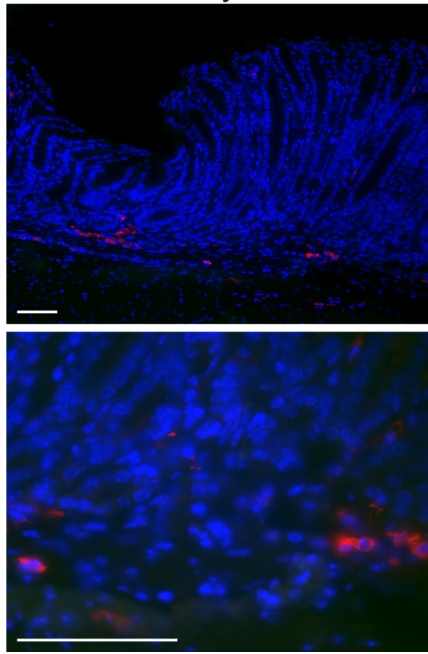


**Figure S2. Creation of *Odc*<sup>Δmye</sup> mice and confirmation of *Odc* deletion in myeloid cells.** (a) A schematic representation of the insertion of *loxP* sites within the *Odc* gene to generate *Odc*<sup>fl/fl</sup> mice and the creation of *Odc*<sup>Δmye</sup> mice by crossing with *LysM*<sup>cre/cre</sup> mice, to create a myeloid-specific knockout of *Odc*. (b) *Odc* mRNA levels were assessed by RT-PCR in isolated gastric lamina propria CD11b<sup>+</sup> (myeloid cells), isolated gastric lamina propria CD11b<sup>-</sup> (non-myeloid cells) and gastric epithelial cells from *Odc*<sup>fl/fl</sup> and *Odc*<sup>Δmye</sup> mice 48 h p.i. with *H. pylori* PMSS1. \*\**P* < 0.01 versus *Odc*<sup>fl/fl</sup> control. §§*P* < 0.01 versus *Odc*<sup>fl/fl</sup> + *H. pylori* PMSS1. *n* = 3 uninfected and 5 infected mice per genotype. Dashed line indicates basal level of *Odc* mRNA in *Odc*<sup>fl/fl</sup> CD11b<sup>+</sup> cells. (c) *Odc* mRNA levels were assessed by RT-PCR in *Odc*<sup>fl/fl</sup>, *Odc*<sup>Δmye</sup>, and *LysM*<sup>cre/cre</sup> bone marrow-derived macrophages (BMmacs) 6 h p.i. with *H. pylori* PMSS1. \*\**P* < 0.01, \*\*\**P* < 0.001. *n* = 3 biological replicates per genotype. Statistical significance in (b) and (c) was calculated by one-way ANOVA with Newman-Keuls post-test. (d) Representative western blot of ODC levels in *Odc*<sup>fl/fl</sup> and *Odc*<sup>Δmye</sup> BMmacs at 6 h p.i. with *H. pylori* PMSS1. (e) Quantification of ODC levels from (d). \*\*\**P* < 0.001. *n* = 3 biological replicates per genotype. Statistical significance was calculated by two-tailed Student's *t* test. Data displayed as mean ± S.E.M.

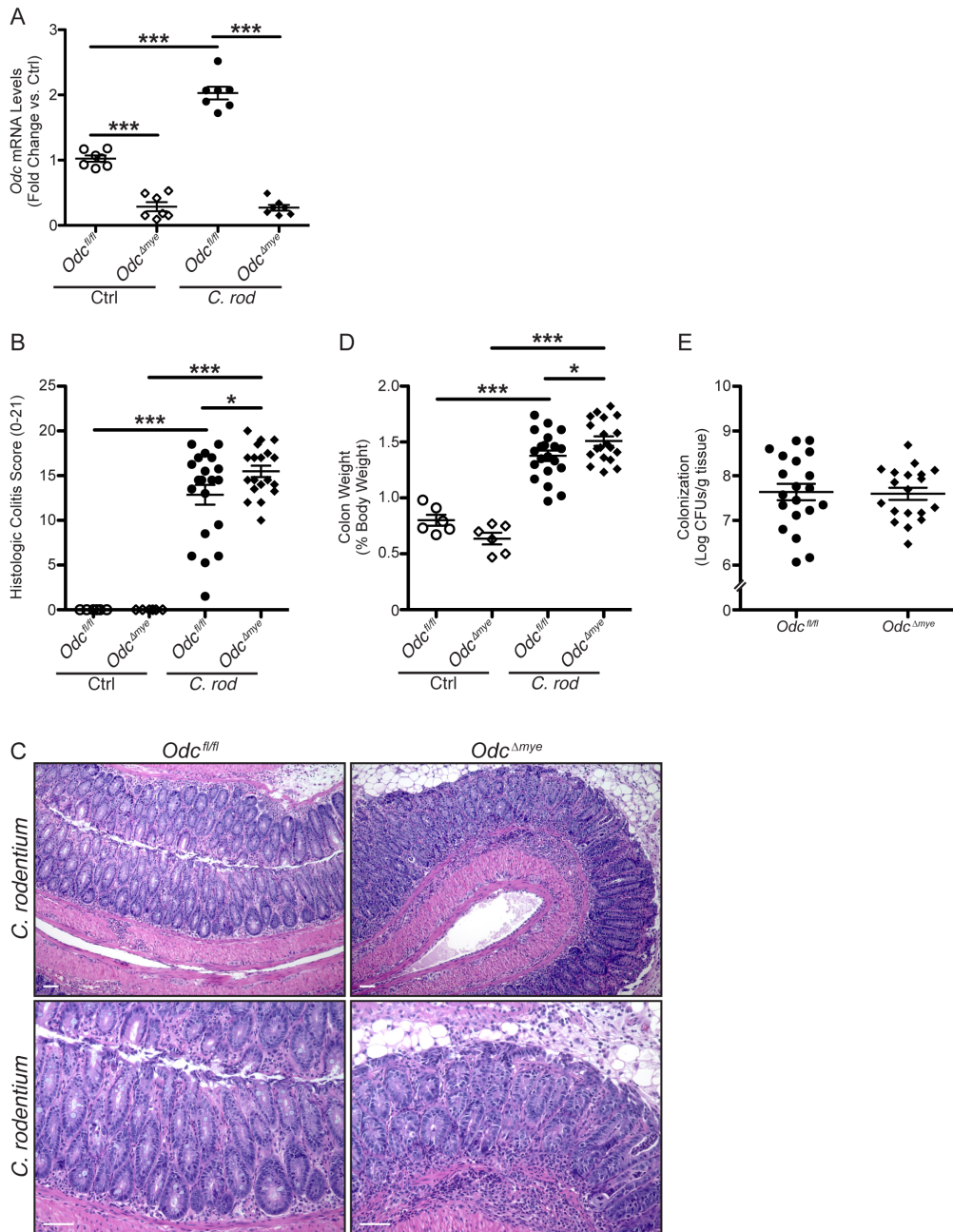


**Figure S3. *Odc* <sup>$\Delta$ mye</sup> gastric tissues and BMmacs have no detectable *Lyz2* expression and robust *Cre* expression.** (A) *Lyz2* (also referred to as *LysM*) and *Cre* mRNA levels were assessed by RT-PCR in *Odc*<sup>fl/fl</sup>, *Odc* <sup>$\Delta$ mye</sup>, and *LysM*<sup>cre/cre</sup> gastric tissues 4 mo p.i. with *H. pylori* SS1. \**P* < 0.05, \*\**P* < 0.01. *n* = 3 uninfected and 5 infected mice per genotype. (B) *Lyz2* and *Cre* mRNA levels were assessed by RT-PCR in *Odc*<sup>fl/fl</sup>, *Odc* <sup>$\Delta$ mye</sup>, and *LysM*<sup>cre/cre</sup> BMmacs 24 h p.i. with *H. pylori* PMSS1. \*\*\**P* < 0.001. *n* = 3 biological replicates per genotype. Statistical significance in all panels was calculated by one-way ANOVA with Newman-Keuls post-test. Data displayed as mean  $\pm$  S.E.M.

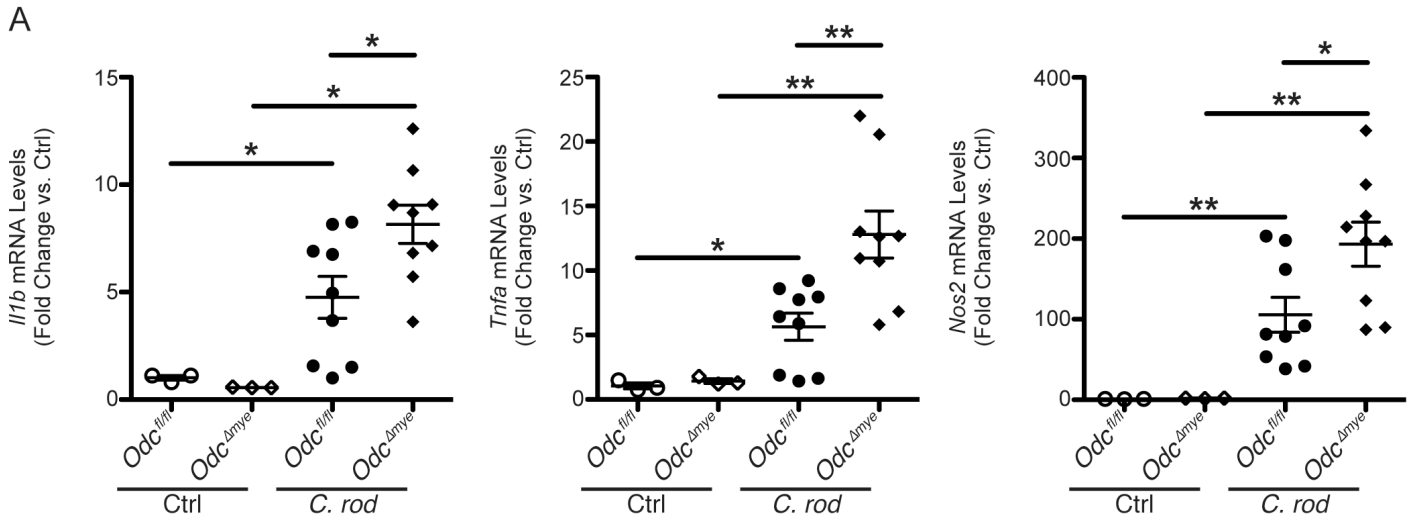
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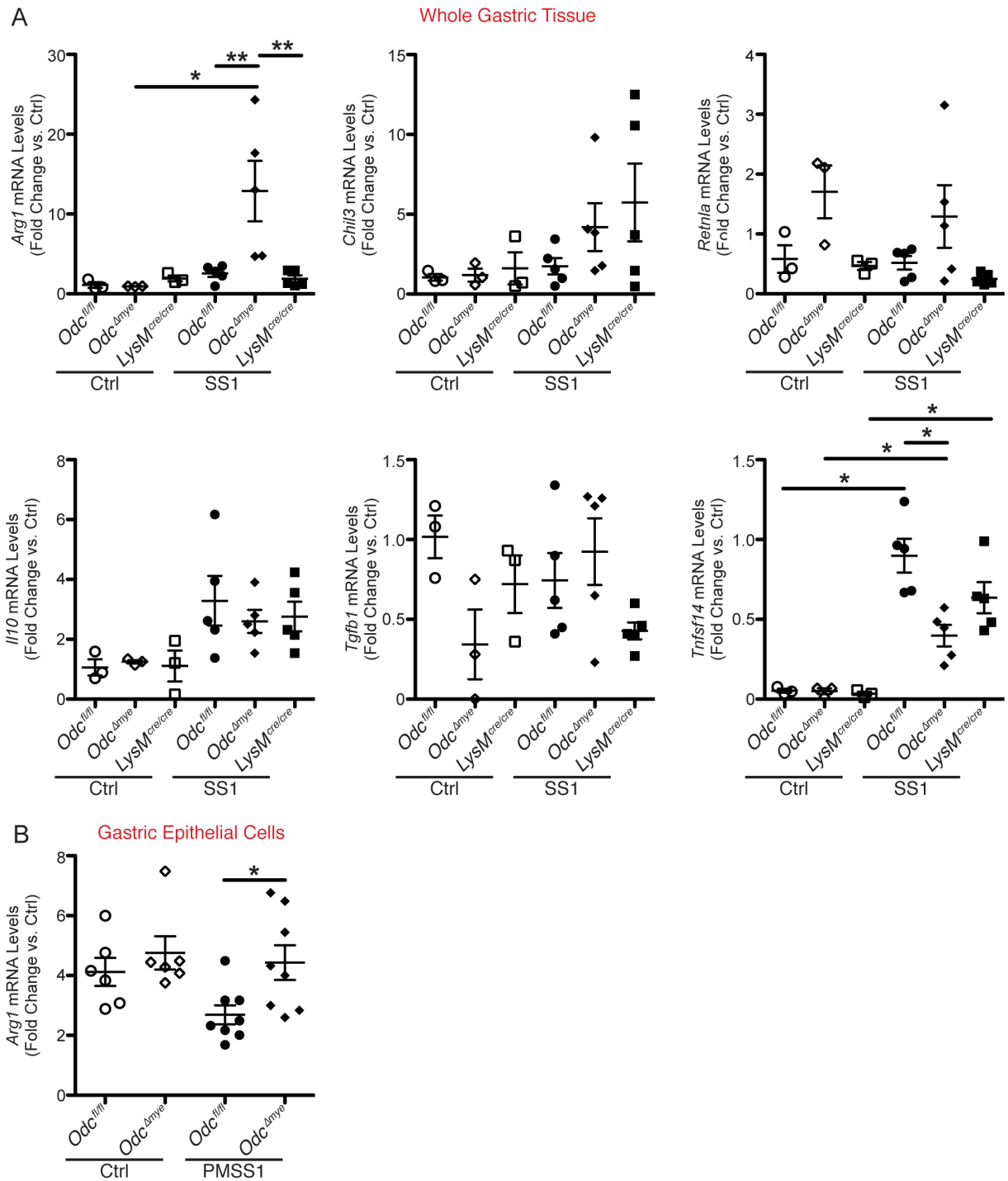
**Figure S4. Immunofluorescence staining for CD68 and ODC in murine gastric tissues.** (a) Representative images of staining where no primary anti-ODC antibody was utilized to confirm specificity of the ODC antibody. Note that the case utilized is the same *Odc<sup>fl/fl</sup>* case as in Figure 1i. Scale bar = 50  $\mu$ m.



**Figure S5. *Odc*<sup>Δmye</sup> mice exhibit significantly increased histologic colitis and increased disease severity during *Citrobacter rodentium* infection.** (a) *Odc* mRNA levels were assessed by RT-PCR in *Odc*<sup>fl/fl</sup> and *Odc*<sup>Δmye</sup> BMmacs 6 h p.i. with *C. rodentium*. \*\*\**P* < 0.001. Statistical significance was calculated by one-way ANOVA with Newman-Keuls post-test. *n* = 7 biological replicates per genotype. (b) Histologic colitis scores were assessed 14 d p.i. with *C. rodentium* by a gastrointestinal pathologist in a blinded manner. \**P* < 0.05, \*\*\**P* < 0.001. *n* = 6 uninfected and 19-20 *C. rodentium*-infected mice per genotype. (c) Representative H&E images from infected mice in (b). Note the transmural inflammation, crypt abscesses, and loss of goblet cells in the *Odc*<sup>Δmye</sup> mouse that is not present in the *Odc*<sup>fl/fl</sup> mouse. Scale bars = 100 μm. (d) Colon weight as a percentage of body weight on the day of sacrifice. \**P* < 0.05, \*\*\**P* < 0.001. *n* = 6 uninfected and 19-20 *C. rodentium*-infected mice per genotype. In (b), and (d), statistical significance was calculated by one-way ANOVA with Newman-Keuls post-test on square-root transformed data. (e) Colonization of *C. rodentium* was assessed by serial dilution and culture 14 d p.i. *n* = 6 uninfected and 19-20 *C. rodentium*-infected mice per genotype. Data displayed as mean ± S.E.M.

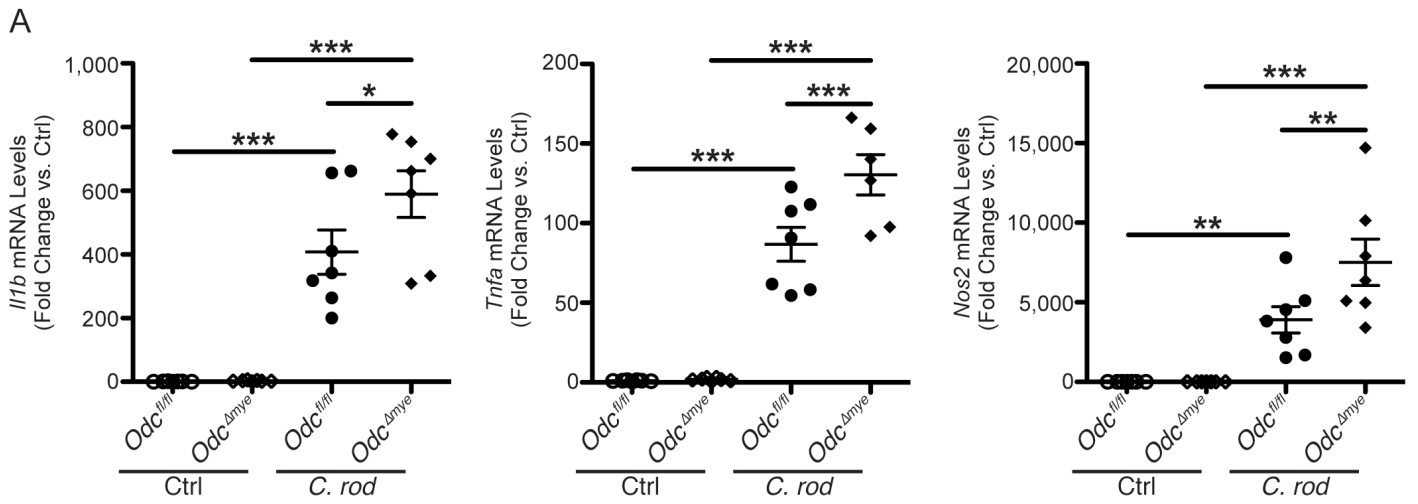


**Figure S6. *Odc<sup>Δmye</sup>* colonic tissues express significantly increased levels of M1 macrophage markers during *C. rodentium* infection.** (a) *Il1b*, *Tnfa*, and *Nos2* mRNA levels were assessed by RT-PCR in *Odc<sup>fl/fl</sup>*, *Odc<sup>Δmye</sup>*, and *LysM<sup>cre/cre</sup>* colonic tissues 14 d p.i. with *C. rodentium*. \* $P < 0.05$ , \*\* $P < 0.01$ .  $n = 3$  uninfected and 9 infected mice per genotype. Statistical significance was calculated by one-way ANOVA with Newman-Keuls post-test. Data displayed as mean  $\pm$  S.E.M.

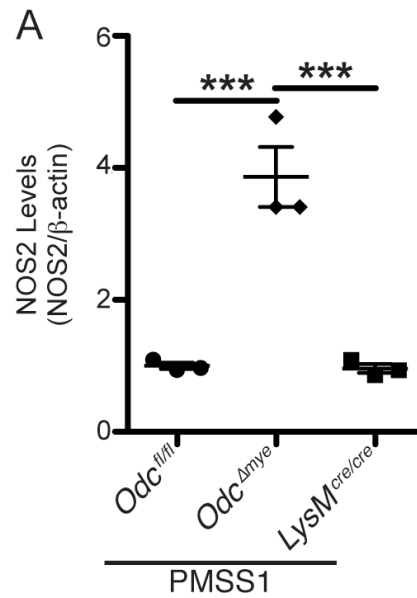


**Figure S7. Markers of M2 macrophage activation are not substantially altered in *Odc*<sup>Δmye</sup> gastric tissues during *H. pylori* infection.** (a) *Arg1*, *Chil3*, *Retnla*, *Il10*, *Tgfb1*, and *Tnfsf14* mRNA levels were assessed by RT-PCR in *Odc*<sup>fl/fl</sup>, *Odc*<sup>Δmye</sup>, and *LysM*<sup>cre/cre</sup> gastric tissues 4 mo p.i. with *H. pylori* SS1. \**P* < 0.05, \*\**P* < 0.01. *n* = 3 uninfected and 5 infected mice per genotype. (b) *Arg1* mRNA levels were assessed by RT-PCR *Odc*<sup>fl/fl</sup>, and *Odc*<sup>Δmye</sup> gastric epithelial cells 48 h p.i. with *H. pylori* PMSS1. \**P* < 0.05. *n* = 4 uninfected and 8 infected mice per genotype. In all panels, statistical significance was calculated by one-way ANOVA with Newman-Keuls post-test. Data displayed as mean ± S.E.M.

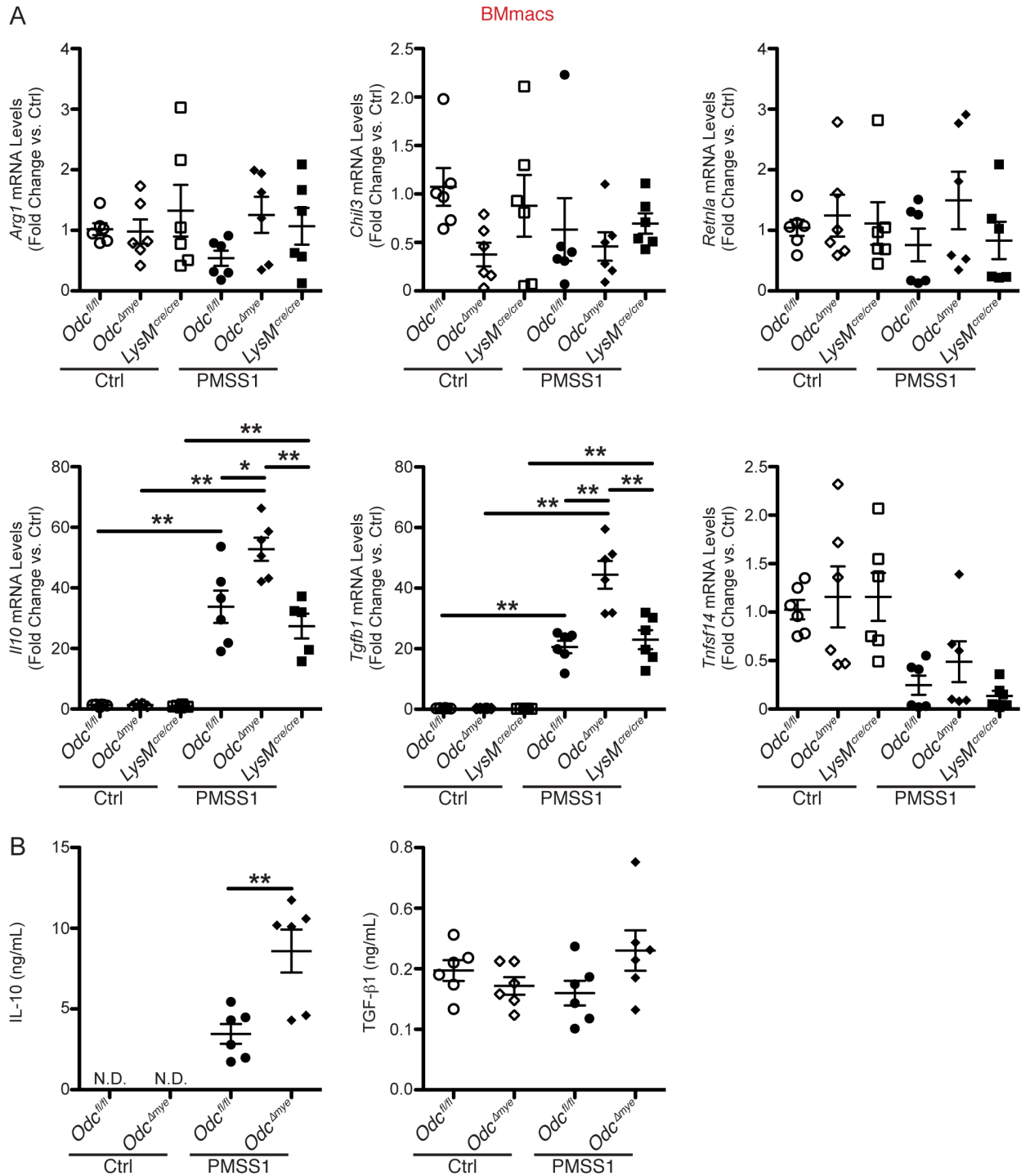




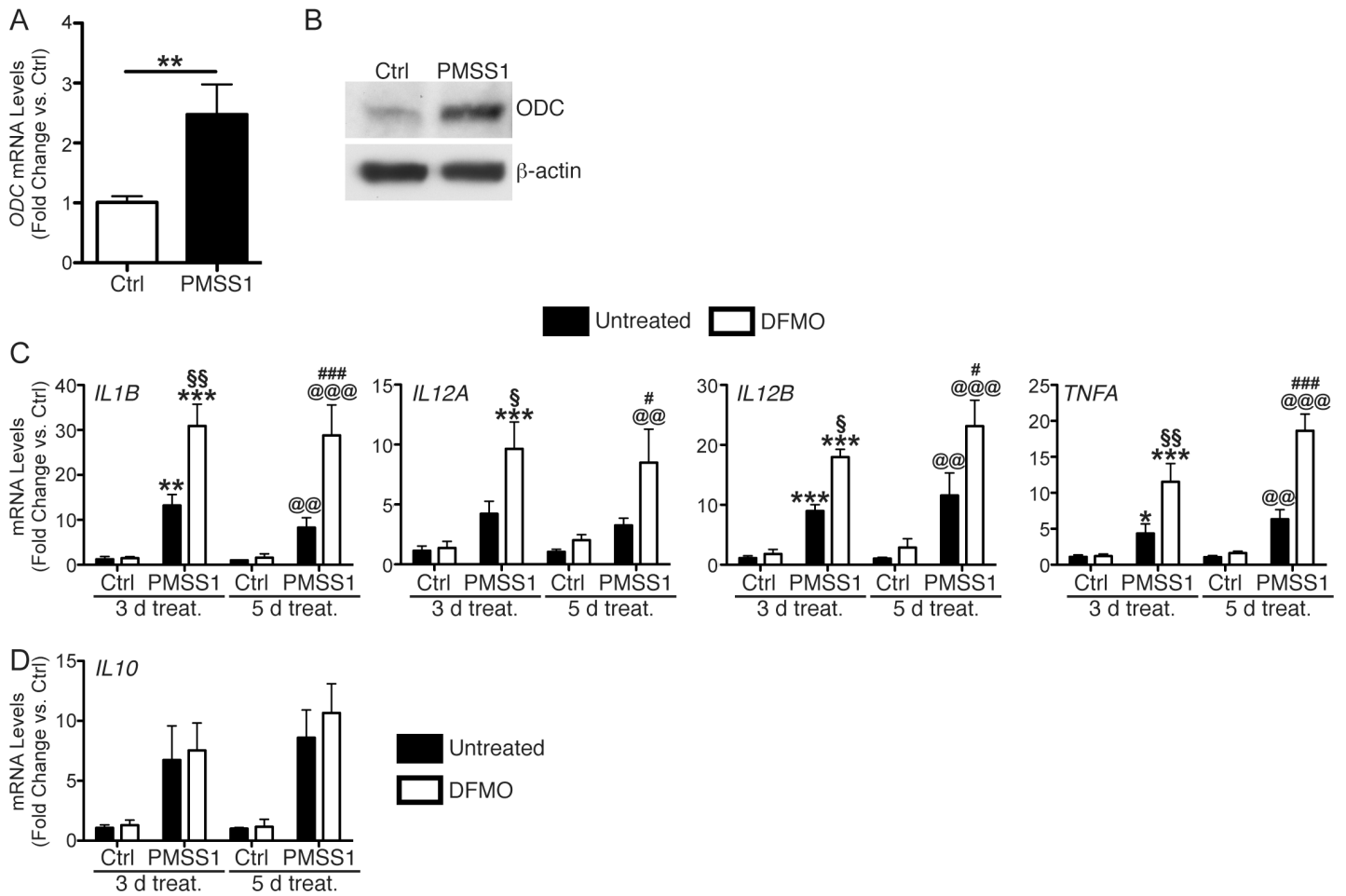
**Figure S8. *Odc<sup>Δmye</sup>* BMmacs express significantly increased levels of M1 macrophage markers during *C. rodentium* infection.** (a) *Il1b*, *Tnfa*, and *Nos2* mRNA levels were assessed by RT-PCR in BMmacs from *Odc<sup>fl/fl</sup>* and *Odc<sup>Δmye</sup>* mice 6 h p.i. with *C. rodentium*. \**P* < 0.05, \*\**P* < 0.01, \*\*\**P* < 0.001. *n* = 7 biological replicates per genotype. Statistical significance was calculated by one-way ANOVA with Newman-Keuls post-test. Data displayed as mean ± S.E.M.



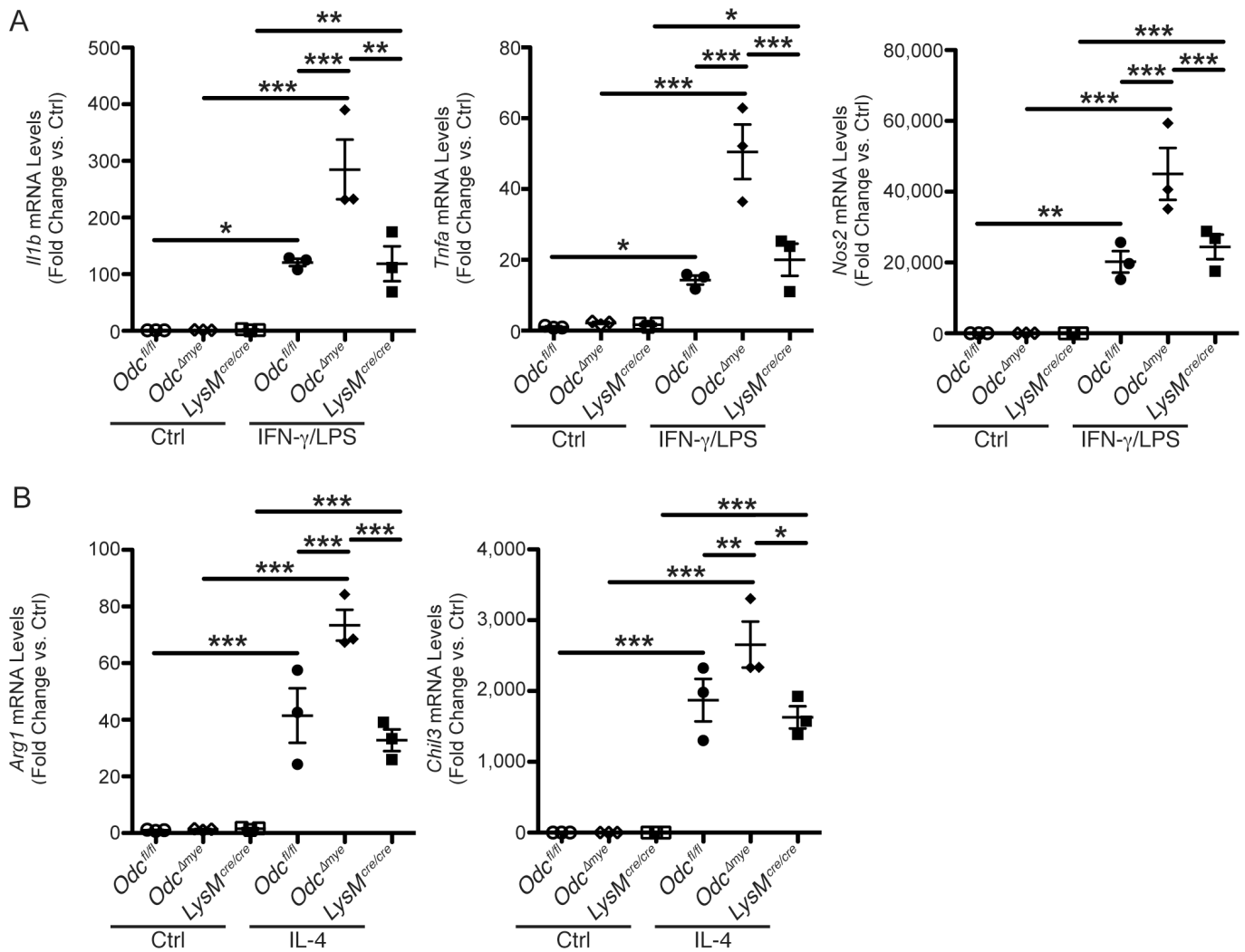
**Figure S9. Densitometric analysis of NOS2 levels in *Odc*<sup>fl/fl</sup>, *Odc* <sup>$\Delta$ mye</sup>, and *LysM*<sup>cre/cre</sup> BMmacs (a)** Densitometric analysis of NOS2 protein levels from the representative western blot in Figure 3d. \*\*\* $P < 0.001$ .  $n = 3$  biological replicates per genotype. Statistical significance was calculated by one-way ANOVA with Newman-Keuls post-test. Data displayed as mean  $\pm$  S.E.M.



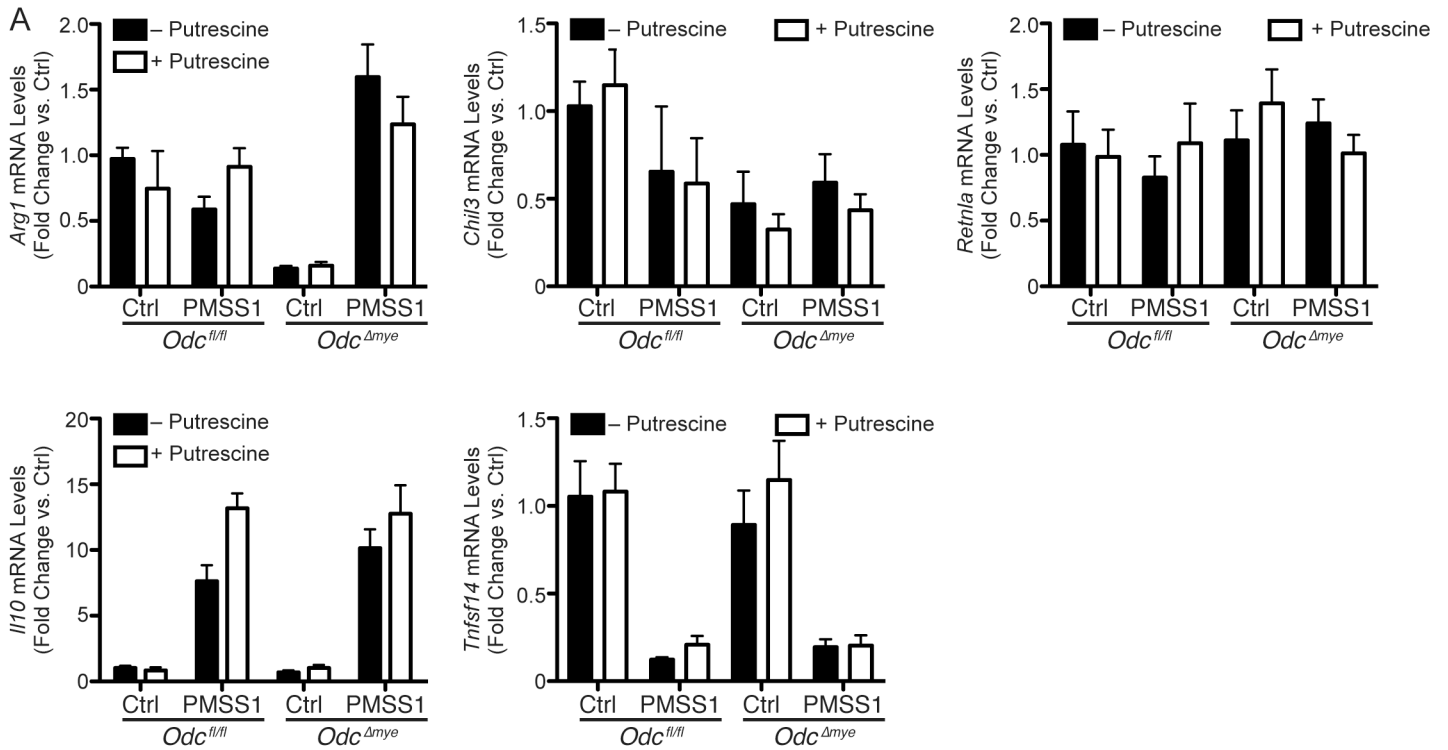
**Figure S10. Markers of M2 macrophage activation are not substantially altered in  $Odc^{\Delta mye}$  BMmacs during *H. pylori* infection.** (a) *Arg1*, *Chil3*, *Retnla*, *Il10*, *Tgfb1*, and *Tnfsf14* mRNA levels were assessed by RT-PCR in BMmacs from *Odc<sup>fl/fl</sup>*, *Odc<sup>Δmye</sup>*, and *LysM<sup>cre/cre</sup>* mice 24 h p.i. with *H. pylori* PMSS1. \* $P < 0.05$ , \*\* $P < 0.01$ .  $n = 6$  mice per genotype. (b) Secreted levels of IL-10 and TGF- $\beta$ 1 were measured by ELISA from supernatants of BMmacs 24 h p.i. with *H. pylori* PMSS1. \*\* $P < 0.01$ .  $n = 6$  mice per genotype. In all panels, statistical significance was calculated by one-way ANOVA with Newman-Keuls post-test. Data displayed as mean  $\pm$  S.E.M.



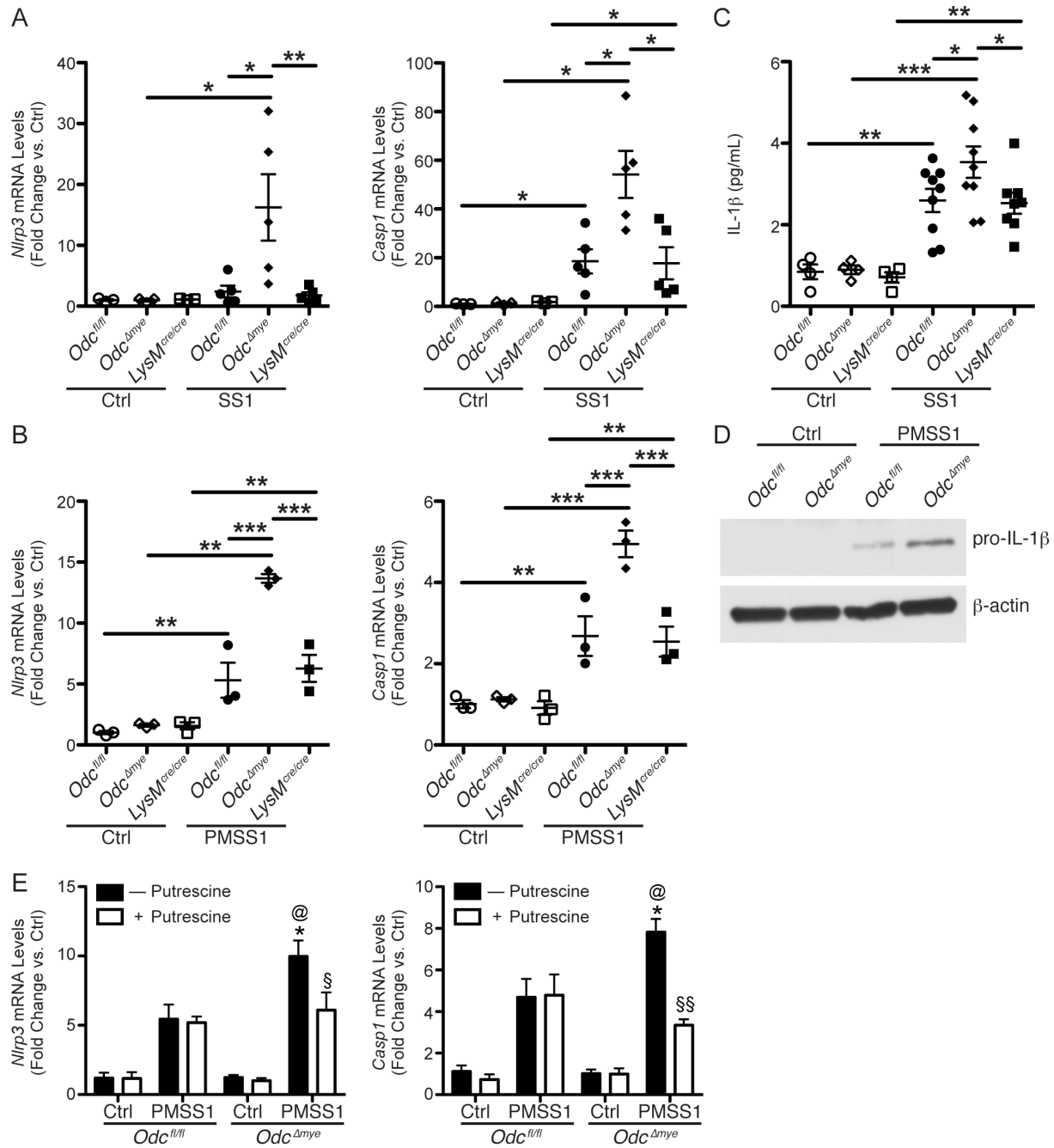
**Figure S11. DFMO-mediated ODC inhibition leads to enhanced M1 macrophage activation in human THP1 cells during *H. pylori* infection.** (a) ODC mRNA was assessed in THP1 cells that were differentiated into macrophages following 18 h treatment with PMA (10 ng/mL) and subsequent 6 h infection with *H. pylori* PMSS1.  $**P < 0.01$ .  $n = 3$  biological replicates. Statistical significance was determined by Student's *t* test. (b) Representative western blot of ODC protein levels in THP1 cells that were differentiated into macrophages following 18 h treatment with PMA (10 ng/mL) and subsequent 6 h infection with *H. pylori* PMSS1.  $n = 3$  biological replicates. (c) *IL1B*, *IL12A*, *IL12B*, and *TNFA* mRNA levels were assessed by RT-PCR in THP1 cells  $\pm$  DFMO (5 nM) for 3 or 5 d prior to 18 h treatment with PMA (10 ng/mL) and subsequent 6 h infection with *H. pylori* PMSS1.  $*P < 0.05$ ,  $**P < 0.01$ ,  $***P < 0.001$  versus 3 d ctrl.  $\$P < 0.05$ ,  $\$\$P < 0.01$  vs. 3 d PMSS1.  $@@P < 0.01$ ,  $@@@P < 0.001$  vs. 5 d ctrl.  $\#P < 0.05$ ,  $###P < 0.001$  vs. 5 d PMSS1.  $n = 3$  biological replicates. Statistical significance was determined by one-way ANOVA with Newman Keuls post-test on square-root transformed data. (d) *IL10* mRNA levels were assessed by RT-PCR in THP1 cells  $\pm$  DFMO (5 nM) for 3 or 5 d prior to 18 h treatment with PMA (10 ng/mL) and subsequent 6 h infection with *H. pylori* PMSS1.  $n = 3$  biological replicates. Data displayed as mean  $\pm$  S.E.M.



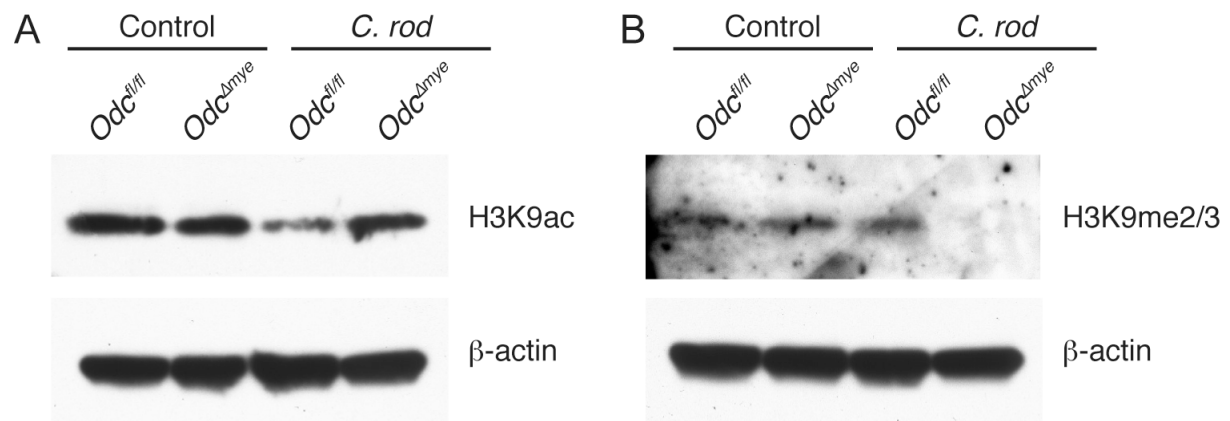
**Figure S12. *Odc* deletion results in enhanced M1 and M2 macrophage activation during treatment with classical stimuli.** (a) *Il1b*, *Tnfa*, and *Nos2* mRNA levels were assessed by RT-PCR in BMmacs from *Odc<sup>fl/fl</sup>*, *Odc<sup>Δmye</sup>*, and *LysM<sup>cre/cre</sup>* mice 24 h post-stimulation with IFN- $\gamma$  (200 U/mL) and LPS (10 ng/mL). \* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$ .  $n = 3$  biological replicates per genotype. (b) *Arg1* and *Chil3* mRNA levels were assessed by RT-PCR in BMmacs from *Odc<sup>fl/fl</sup>*, *Odc<sup>Δmye</sup>*, and *LysM<sup>cre/cre</sup>* mice 24 h post-stimulation with IL-4 (10 ng/mL). \* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$ .  $n = 3$  biological replicates per genotype. Statistical significance in all panels was calculated by one-way ANOVA with Newman-Keuls post-test. Data displayed as mean  $\pm$  S.E.M.



**Figure S13. Addition of excess putrescine does not alter M2 macrophage activation marker expression during *H. pylori* infection.** (a) *Arg1*, *Chil3*, *Retnla*, *Il10*, and *Tnfsf14* mRNA levels were assessed by RT-PCR in BMmacs from *Odc<sup>fl/fl</sup>*, *Odc<sup>Δmye</sup>*, and *LysM<sup>cre/cre</sup>* mice 24 h p.i. with *H. pylori* PMSS1 ± 25 μM putrescine added 60 min prior to infection. *n* = 4 biological replicates per genotype. Data displayed as mean ± S.E.M.

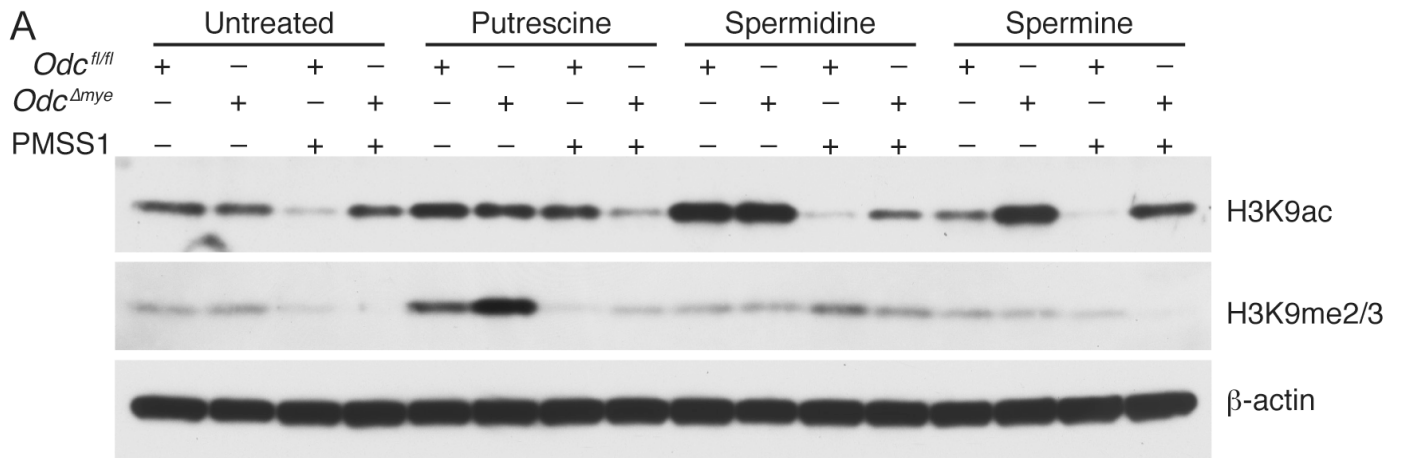


**Figure S14. *Odc* deletion in macrophages enhances NLRP3-inflammasome activation during *H. pylori* infection.** (a) mRNA levels of *Nlrp3* and *Casp1* were assessed by RT-PCR in gastric tissues 4 mo p.i. with *H. pylori* SS1. \* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$  by one-way ANOVA with Kruskal-Wallis post-test, followed by Mann-Whitney *U* test.  $n = 3$  uninfected and 5 *H. pylori* SS1 infected mice per genotype. (b) mRNA levels of *Nlrp3* and *Casp1* were assessed by RT-PCR in BMmacs 24 h p.i. with *H. pylori* PMSS1. \*\* $P < 0.01$ , \*\*\* $P < 0.001$  by one-way ANOVA with Newman-Keuls post-test.  $n = 3$  mice per genotype. (c) IL-1 $\beta$  protein levels were assessed by ELISA in gastric tissues 4 mo p.i. with *H. pylori* SS1. \* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$  by one-way ANOVA with Kruskal-Wallis post-test, followed by Mann-Whitney *U* test.  $n = 4$  uninfected and 8-9 *H. pylori* SS1-infected mice per genotype. (d) pro-IL-1 $\beta$  protein levels were assessed by western blotting in BMmacs 24 h p.i. with *H. pylori* PMSS1.  $n = 4$  mice per genotype. (e) mRNA levels of *Nlrp3* and *Casp1* were assessed by RT-PCR in BMmacs 24 h p.i. with *H. pylori* PMSS1  $\pm$  25 mM putrescine added 60 min prior to infection. \* $P < 0.05$  vs. *Odc*<sup>fl/fl</sup> + PMSS1; @ $P < 0.05$ , @@@ $P < 0.001$  vs. *Odc*<sup>fl/fl</sup> + PMSS1 + putrescine; § $P < 0.05$ , §§ $P < 0.01$  vs. *Odc* <sup>$\Delta$ mye</sup> + PMSS1 by one-way ANOVA with Newman-Keuls post-test.  $n = 4$  biological replicates. Data displayed as mean  $\pm$  S.E.M.

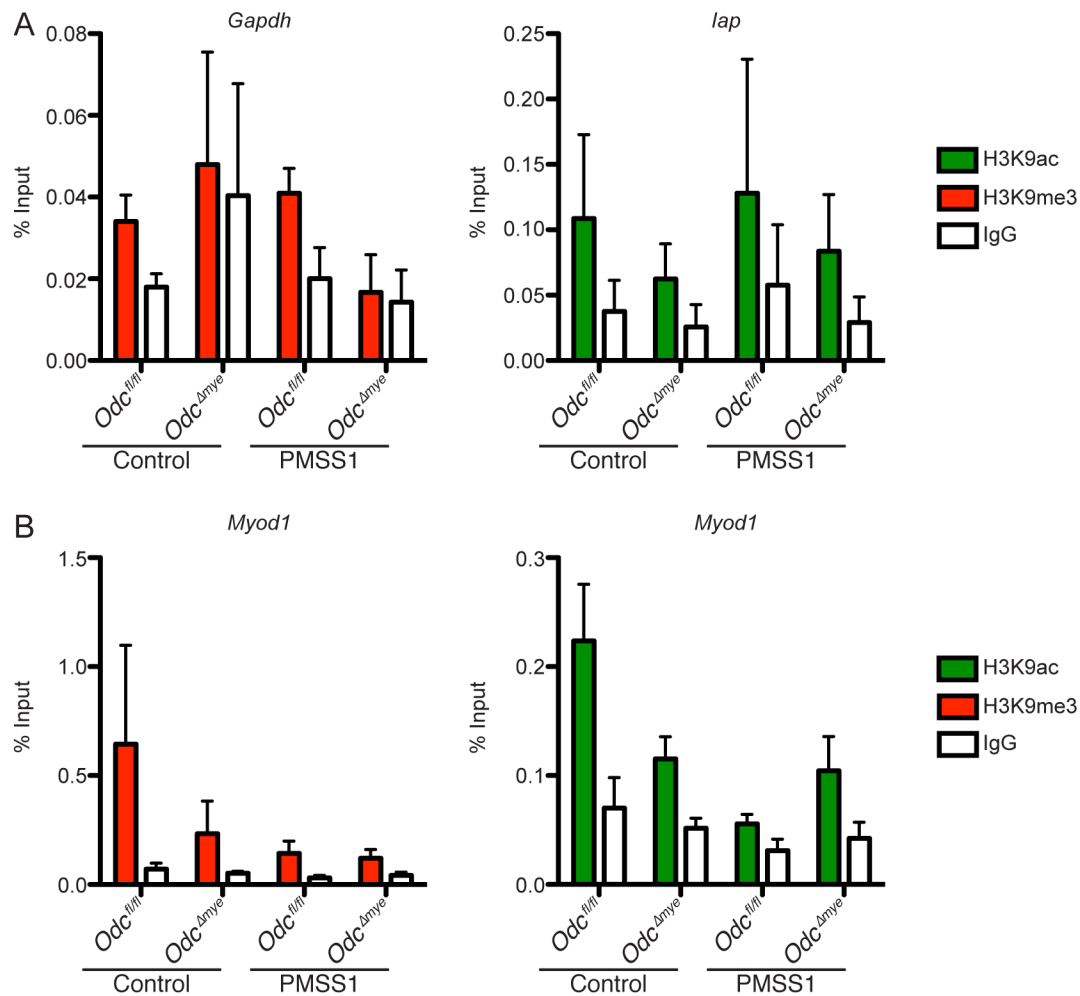


**Figure S15. *Odc* deletion in macrophages alters acetylation and di/tri-methylation of histone 3, lysine 9 during *C. rodentium* infection.** (a) Representative western blot of H3K9ac and levels in BMmacs 6 h p.i. with *C. rodentium*.  $n = 3$  biological replicates. (b) Representative western blot of H3K9me2/3 levels in BMmacs 24 h p.i. with *C. rodentium*.  $n = 3$  biological replicates.

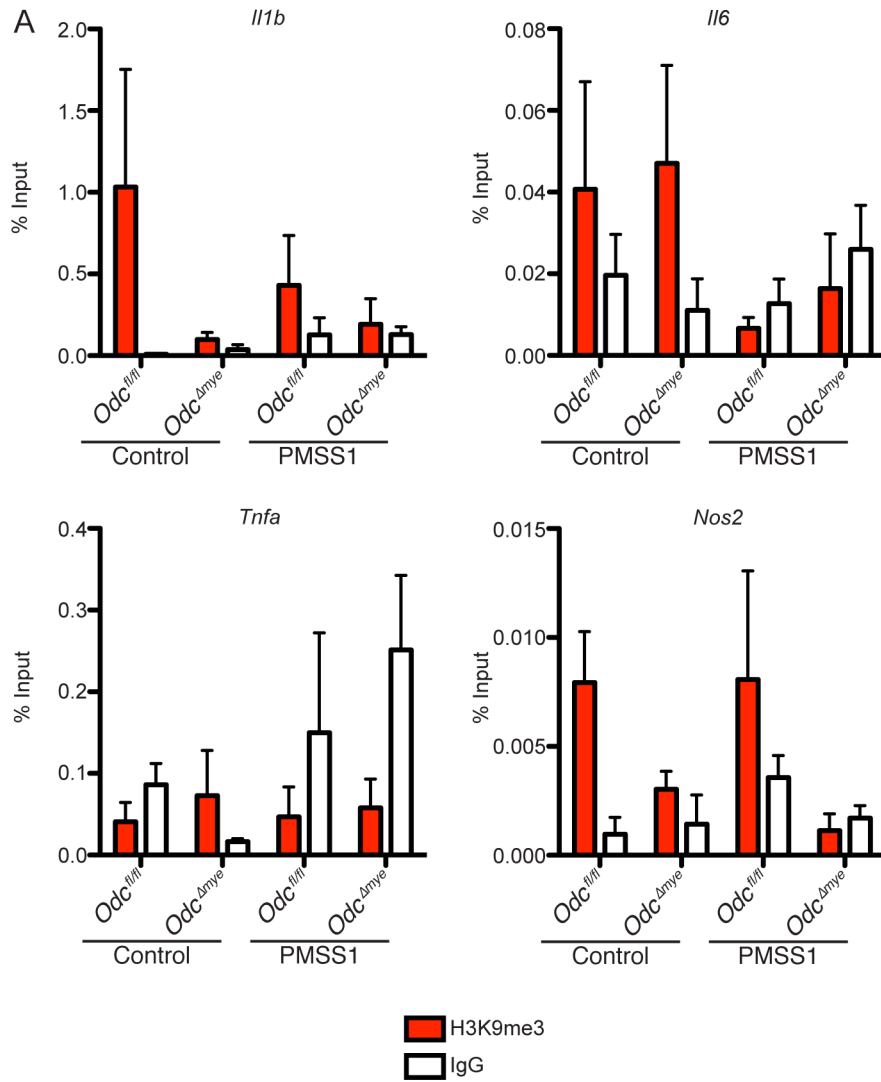




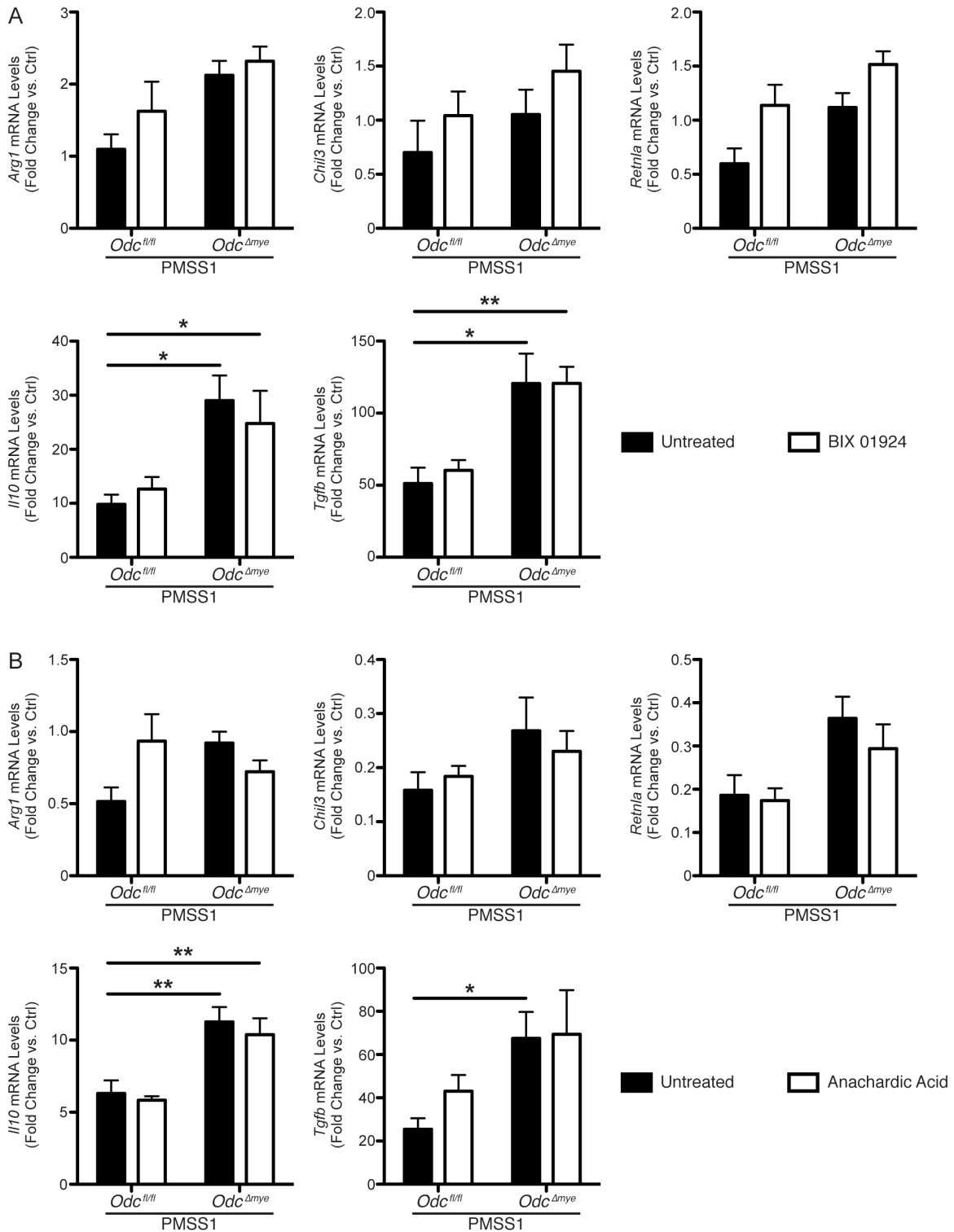
**Figure S16. Putrescine add-back returns the H3K9ac and H3K9me2/3 levels in *Odc<sup>Δmye</sup>* BMmacs to those in *Odc<sup>fl/fl</sup>* BMmacs.** (a) Representative western blot of H3K9ac and H3K9me2/3 levels in BMmacs 24 h p.i. with *H. pylori* PMSS1 ± 25 mM putrescine, 10 mM spermidine, or 10 mM spermine added 60 min prior to infection. *n* = 3 biological replicates.



**Figure S17. Gene expression of unchanged markers of general transcriptional levels following ChIP with anti-H3K9ac and anti-H3K9me3 antibodies in *Odc*-deficient BMmacs.** (a) Expression of *Gapdh* and *lap* was assessed by RT-PCR in BMmacs from *Odc<sup>fl/fl</sup>* and *Odc<sup>Δmye</sup>* mice 24 h p.i. with *H. pylori* PMSS1 with subsequent ChIP with the denoted antibodies.  $n = 3$  biological replicates. (b) Expression of *Myod1* was assessed by RT-PCR in BMmacs from *Odc<sup>fl/fl</sup>* and *Odc<sup>Δmye</sup>* mice 24 h p.i. with *H. pylori* PMSS1 with subsequent ChIP with the denoted antibodies. Data displayed as mean  $\pm$  S.E.M.



**Figure S18. Gene expression of unchanged pro-inflammatory markers following ChIP with anti-H3K9ac and anti-H3K9me3 antibodies in *Odc*-deficient BMmacs.** (a) Expression of *Il1b*, *Il6*, *Tnfa*, and *Nos2* promoter sequences was assessed by RT-PCR in BMmacs from *Odc<sup>fl/fl</sup>* and *Odc<sup>Δmye</sup>* mice 24 h p.i. with *H. pylori* PMSS1, followed by subsequent ChIP with the denoted antibodies.  $n = 3$  biological replicates. Data displayed as mean  $\pm$  S.E.M.



**Figure S19. Alterations in histone modifications and chromatin structure in ODC-deficient macrophages does not alter M2 macrophage activation during *H. pylori* infection.** (a) M2 markers, *Arg1*, *Chil3*, and *Retnla*, *Il10*, and *Tgfb* were assessed at the mRNA level by RT-PCR in BMmacs 24 h p.i. with *H. pylori* PMSS1 ± 5 μM BIX 01924 added 60 min prior to infection. \* $P < 0.05$ , \*\* $P < 0.01$ , by one-way ANOVA with Kruskal-Wallis post-test, followed by Mann-Whitney *U* test.  $n = 5$  mice per genotype. (b) M2 markers, *Arg1*, *Chil3*, and *Retnla*, *Il10*, and *Tgfb* were assessed at the mRNA level by RT-PCR in BMmacs 24 h p.i. with *H. pylori* PMSS1 ± 10 μM anacardic acid added 60 min prior to infection. \*\*\* $P < 0.001$  by one-way ANOVA with Kruskal-Wallis post-test, followed by Mann-Whitney *U* test.  $n = 5$  mice per genotype. Data displayed as mean ± S.E.M.

Analyte	Concentration of Analyte (pg/mg protein); Mean $\pm$ S.E.M.					
	<i>Odc</i> <sup>fl/fl</sup>		<i>Odc</i> <sup><math>\Delta</math>mye</sup>		<i>LysM</i> <sup>cre/cre</sup>	
	Uninfected	<i>H. pylori</i> SS1	Uninfected	<i>H. pylori</i> SS1	Uninfected	<i>H. pylori</i> SS1
CSF2	3.70 $\pm$ 0.77	5.30 $\pm$ 0.53	3.42 $\pm$ 0.24	6.77 $\pm$ 0.86*	2.61 $\pm$ 0.56	4.09 $\pm$ 0.64
CSF3	0.79 $\pm$ 0.35	0.95 $\pm$ 0.20	0.30 $\pm$ 0.04	0.44 $\pm$ 0.11	0.38 $\pm$ 0.10	0.29 $\pm$ 0.07
IFN- $\gamma$	0.84 $\pm$ 0.11	1.33 $\pm$ 0.21	0.86 $\pm$ 0.20	2.02 $\pm$ 0.47***	0.67 $\pm$ 0.13	0.53 $\pm$ 0.07
IL-1 $\alpha$	24.01 $\pm$ 2.41	24.30 $\pm$ 4.01	31.80 $\pm$ 3.52	32.00 $\pm$ 2.97	31.01 $\pm$ 5.44	28.17 $\pm$ 2.44
IL-2	1.08 $\pm$ 0.08	0.65 $\pm$ 0.09	0.90 $\pm$ 0.20	0.53 $\pm$ 0.07	1.19 $\pm$ 0.26	0.74 $\pm$ 0.14
IL-4	0.22 $\pm$ 0.03	0.20 $\pm$ 0.03	0.18 $\pm$ 0.03	0.18 $\pm$ 0.04	0.20 $\pm$ 0.06	0.21 $\pm$ 0.05
IL-5	0.84 $\pm$ 0.24	0.70 $\pm$ 0.08	0.40 $\pm$ 0.04	0.56 $\pm$ 0.12	0.40 $\pm$ 0.07	0.58 $\pm$ 0.17
IL-6	1.30 $\pm$ 0.40	1.07 $\pm$ 0.12	1.23 $\pm$ 0.27	1.76 $\pm$ 0.64	2.15 $\pm$ 1.27	0.93 $\pm$ 0.10
IL-7	0.68 $\pm$ 0.14	1.31 $\pm$ 0.33	0.69 $\pm$ 0.06	0.96 $\pm$ 0.11	0.46 $\pm$ 0.09	0.60 $\pm$ 0.08
IL-9	10.84 $\pm$ 2.01	8.91 $\pm$ 1.40	10.73 $\pm$ 3.35	7.46 $\pm$ 1.63	10.81 $\pm$ 3.25	5.49 $\pm$ 1.28
IL-10	0.24 $\pm$ 0.09	0.26 $\pm$ 0.05	0.14 $\pm$ 0.02	0.24 $\pm$ 0.08	0.12 $\pm$ 0.01	0.10 $\pm$ 0.03
IL-12p40	1.96 $\pm$ 0.54	1.01 $\pm$ 0.15	1.13 $\pm$ 0.28	0.49 $\pm$ 0.15	0.62 $\pm$ 0.53	0.69 $\pm$ 0.21
IL-12p70	0.56 $\pm$ 0.10	0.48 $\pm$ 0.09	0.47 $\pm$ 0.09	0.78 $\pm$ 0.22	0.46 $\pm$ 0.09	0.29 $\pm$ 0.04
IL-13	0.06 $\pm$ 0.04	0.57 $\pm$ 0.29	0.04 $\pm$ 0.02	0.99 $\pm$ 0.40	0.54 $\pm$ 0.52	0.37 $\pm$ 0.18
IL-15	0.51 $\pm$ 0.19	2.13 $\pm$ 0.41	1.59 $\pm$ 0.40	1.77 $\pm$ 0.29	1.11 $\pm$ 0.36	0.87 $\pm$ 0.10

**Table S1. Luminex analytes that did not demonstrate significant differences in gastric tissues from uninfected and infected *Odc*<sup>fl/fl</sup>, *Odc* <sup>$\Delta$ mye</sup>, and *LysM*<sup>cre/cre</sup> mice.** A total of 25 distinct analytes were assessed in gastric tissue from uninfected and infected mice from each of the three genotypes. Listed are the analytes that were not significantly induced by infection, or demonstrated few or no significant differences between genotypes. Note that IL-1 $\beta$  is not shown, as this analyte was measured by ELISA.  $n = 4$  uninfected and 8-9 *H. pylori* SS1 infected mice per genotype. \* $P < 0.05$  and \*\*\* $P < 0.001$  versus *LysM*<sup>cre/cre</sup> + *H. pylori* SS1.

<b>Antibody</b>	<b>Dilution</b>	<b>Application</b>	<b>Source (Location)</b>
Rabbit polyclonal anti-NOS2	1:5,000	WB	EMD Millipore (Darmstadt, Germany) Cat. no. ABN26
Mouse monoclonal anti-IL-1 $\beta$	1:1,000	WB	Cell Signaling Technology (Danvers, MA) Cat. no. 12242
Rabbit polyclonal anti-ODC	1:5,000 1:2,000 1:2,000	WB IF IHC-P	Lisa Shantz (Penn State College of Medicine) David Feith (University of Virginia) (1, 2)
Mouse monoclonal anti-H3K9me2/3	1:1,000	WB	Cell Signaling Technology (Danvers, MA) Cat. no. 5327
Rabbit polyclonal anti-H3K9ac	1:1,000 1:200	WB IF	Cell Signaling Technology (Danvers, MA) Cat. no. 9649
Rabbit polyclonal anti-H3K4me1	1:1,000	WB	Cell Signaling Technology (Danvers, MA) Cat. no. 9723
Mouse monoclonal anti- $\beta$ -actin	1:10,000	WB	Sigma-Aldrich (St. Louis, MO) Cat. no. A1978
Goat anti-mouse IgG, HRP labeled	1:30,000	WB	Sigma-Aldrich (St. Louis, MO) Cat. no. 115-035-003
Goat anti-rabbit IgG, HRP labeled	1:3,000	WB	Sigma-Aldrich (St. Louis, MO) Cat. no. 111-035-003
Rabbit polyclonal anti-CD68	1:200	IF	Boster Biological Technology (Pleasanton, CA) Cat. no. PA1518
Goat anti-rabbit IgG, Alexa488	1:400	IF	ThermoFisher Scientific (Waltham, MA) Cat. no. A11078
Goat anti-rabbit IgG, Alexa555	1:500	IF	ThermoFisher Scientific (Waltham, MA) Cat. no. A21429

**Table S2.** List of all antibodies used for this study, including the dilution, application and company from which the antibodies were purchased. WB = western blotting, IF = immunofluorescence. IHC-P = immunoperoxidase. <sup>1,2</sup> = See **Supporting Information References** for further antibody information.

Species	Target	Sequence
Mouse	<i>β-actin</i>	F: CCAGAGCAAGAGAGGTATCC
		R: CTGTGGTGGTGAAGCTGTAG
Mouse	<i>Nos2</i>	F: CACCTTGGAGTTCACCCAGT
		R: ACCACTCGTACTTGGGATGC
Mouse	<i>Tnfa</i>	F: CTGTGAAGGGAATGGGTGTT
		R: GGTCACGTCCCAGCATCTT
Mouse	<i>Il1b</i>	F: ACCTGCTGGTGTGTGACGTTCC
		R: GGGTCCGACAGCACGAGGCT
Mouse	<i>Il6</i>	F: AGTTGCCTTCTTGGGACTGA
		R: TCCACGATTTCCAGAGAAC
Mouse	<i>Il12a</i>	F: AAATGAAGCTCTGCATCCTGC
		R: TCACCCTGTTGATGGTCACG
Mouse	<i>Il12b</i>	F: GAAAGACCCTGACCATCACT
		R: CTTTCTCTGCAGACAGAGAC
Mouse	<i>Il23a</i>	F: CCAGCAGCTCTCTCGGAATC
		R: TCATAGTCCCGCTGGTGC
Mouse	<i>Arg1</i>	F: AAGAAAAGGCCGATTACCT
		R: CACCTCCTCTGCTGTCTTCC
Mouse	<i>Chil3</i>	F: ACTTTGATGGCCTCAACCTG
		R: AATGATTCCTGCTCCTGTGG
Mouse	<i>Retnla</i>	F: GGGATGACTGCTACTGGGTG
		R: TCAACGAGTAAGCACAGGCA
Mouse	<i>Il10</i>	F: CCAAGCCTTATCGGAAATGA
		R: TCACTCTTACCTGCTCCAC
Mouse	<i>Tgfb1</i>	F: TCCTTGCCTGCGGAAGTG
		R: GGAGAGCATTGAGCAGTTCGA
Mouse	<i>Tnfsf14</i>	F: CTGCATCAACGTCTTGGAGA
		R: GATACGTCAAGCCCCTCAAG
Mouse	<i>Odc</i>	F: CCTTGTGAGGAGCTGGTGATA
		R: GGTCCAGAATGTCCTTAGCAGT
Mouse	<i>Nlrp3</i>	F: ATGCTGGCTTCGACATCTCCT
		R: GTTTCTGGAGGTTGCAGAGC
Mouse	<i>Casp1</i>	F: AGATGCCCACTGCTGATAGG
		R: TTGGCACGATTCTCAGCATA
Mouse	<i>Nos2 (Promoter)</i>	F: ATGGCCTTGCATGAGGATAC
		R: CACCAAGGTGGCTGAGAAGT
Mouse	<i>Il1b (Promoter)</i>	F: CCCCTAAGAATTCCCATCAAGC
		R: GAGCTGTGAAATTTCCCTTGG
Mouse	<i>Tnfa (Promoter)</i>	F: CCCAGATTGCCACAGAATC
		R: CCAGTGAGTAAAGGGACAG
Mouse	<i>Il6 (Promoter)</i>	F: CCCACCCTCCAACAAAGATT
		R: GCTCCAGAGCAGAATGAGCTA
Human	<i>IL1B</i>	F: TGAAGTGCACGCTCCGG
		R: GAACACCACTTGTTGCTC
Human	<i>TNFA</i>	F: ATGAGCACTGAAAGCATGATCC
		R: GAGGGCTGATTAGAGAGAGGTC
Human	<i>IL6</i>	F: GTAGCCGCCCCACACAGA
		R: CATGTCTCCTTTCTCAGGGCTG
Human	<i>IL12A</i>	F: CAAAACCTGCTGAGGGCCGTC
		R: GGAGGCCAGGCAACTCCCATTAG
Human	<i>IL12B</i>	F: CCAAGAACTTGCAGCTGAAG
		R: TGGGTCTATTCCGTTGTGTC
Human	<i>IL10</i>	F: GCCTAACATGCTTCGAGATC
		R: TGATGTCTGGGTCTTGGTTC

**Table S3.** List of primers used for RT-PCR. (Promoter) = primers utilized for ChIP-PCR.

### **Supporting Information References:**

1. Shantz LM, Guo Y, Sawicki JA, Pegg AE, & O'Brien TG (2002) Overexpression of a dominant-negative ornithine decarboxylase in mouse skin: Effect on enzyme activity and papilloma formation. *Carcinogenesis* 23(4):657-664.
2. Shantz LM & Pegg AE (1998) Ornithine decarboxylase induction in transformation by H-Ras and RhoA. *Cancer Res.* 58(13):2748-2753.