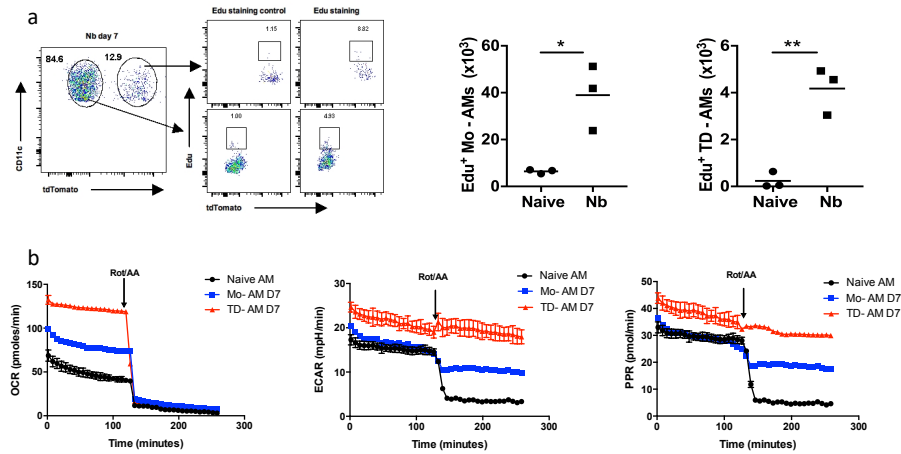


Supplementary fig. 1

Suppl. Fig. 1 Gene expression profile tdTomato<sup>±</sup> subpopulation of alveolar macrophages during *N. brasiliensis* infection.

*Cx3cr1*<sup>CreERT2-EYFP-IRES-YFP/+</sup>*Rosa26*<sup>flxed-tdTomato/+</sup> reporter mice received tamoxifen at days -1, +1, after *N. brasiliensis* (*Nb*) inoculation. At day 7 and day 14, Lung tdTomato<sup>+</sup> (Mo-AM) and tdTomato<sup>-</sup> (TD-AM) alveolar macrophages were sort-purified for RNA-seq analysis. (a-b) Venn diagram illustrating overlap of respectively upregulated or downregulated significant genes (FDR adjusted p-value <0.05) across all sample groups as compared to AMs from naïve mice. (c) Selected Ingenuity Pathway (IPA- Qiagen) enrichment analyses for respective samples compared to AMs from naïve mice representing either upregulation or downregulation. Dotted red bar illustrates significant enrichment cutoff (p<0.05). (d) Expression of selected characteristic wound healing response markers in TD-AMs and Mo-AMs at day 7 and day 14 after *Nb* inoculation as expressed relative to AMs from naïve mice (Log<sub>2</sub> fold change).



Supplementary Fig. 2

Suppl. Fig. 2 Cell proliferation and metabolic analysis of lung macrophage subsets after *N. brasiliensis* infection.

(a)  $Cx3cr1^{CreERT2-EYFP-IRES-YFP/+}Rosa26^{floxed-tdTomato/+}$  mice received tamoxifen at days -1, +1, and Edu at days 0, +3, +5 after *N. brasiliensis* (Nb) inoculation. At day 7 after Nb inoculation, whole lung tdTomato<sup>+</sup> monocyte-derived alveolar macrophages (Mo-AMs) and tdTomato<sup>-</sup> tissue-derived alveolar macrophages (Td-AMs) were analyzed by flow cytometry for Edu incorporation. Mean and SE for 3 mice/treatment group are shown. (b)  $Cx3cr1^{CreERT2-EYFP-IRES-YFP/+}Rosa26^{floxed-tdTomato/+}$  were administered tamoxifen and Nb inoculated as in (a). At day 7 after inoculation, Mo-AMs, TD-AMs, and naïve alveolar macrophages were sort-purified and real-time metabolic activity assessed by Seahorse analysis. The oxygen consumption rate (OCR) and extracellular acidification rate (ECAR) were measured and Rotenone (Rot) and Antimycin A (AA) inhibitors were added to block mitochondrial activity. Experiments in (a) and (b) were repeated two times with similar results.