**S1 Figure, related to Figure 2. Genetic and pharmacological inhibition of Nampt alleviates skin inflammation and restores epithelial integrity in spint1a–deficient larvae.** (A) Neutrophil distribution of wild type and Spint1a-deficient larvae treated with the pharmacological inhibitors of Nampt GMX1778 and FK-866. (B) Representative merge images (brightfield and red channel) of lyz:dsRED zebrafish larvae of every group are shown. (C) For genetic inhibition using CRISPR/Cas9 technology, one-cell stage zebrafish eggs were microinjected and imaging was performed in 3 dpf larvae. (D) Quantification of the percentage of neutrophils out of the CHT in Spint1a-deficient larvae upon knockdown of Nampta/Namptb. (E) Representative merge images (brightfield and red channel) of lyz:dsRED zebrafish larvae of every group are shown. (F) Analysis of genome editing efficiency in larvae injected with control or nampta and namptb crRNA/Cas9 complexes and quantification rate of non-homologous end-joining (NHEJ) showing all insertions and deletions (INDELs) (https://tide.deskgen.com/). Each dot represents one individual and the mean ± S.E.M. for each group is also shown. P values were calculated using one-way ANOVA and Tukey multiple range test and t-Test. **p<0.001, ****p<0.0001. (G) Inflammation and oxidative stress determination in zebrafish larvae (Candel et al., 2014). Inflammation was scored by using two different approaches: i) the lyz:dsRED zebrafish transgenic line was used to quantify the percentage of neutrophils out of the CHT, i.e. neutrophil dispersion and ii) the nfkb:eGFP zebrafish transgenic line was used to determine NFkB activity by quantification of fluorescence intensity in the drawn white box. For oxidative stress, analysis of fluorescence intensity of the region of interest (white box) of larvae preloaded with an H2O2 fluorogenic probe. The data underlying this figure can be found in S1 Data.