**Supplemental Material**

**Synthesis of Affibody molecules**

$Z_{\text{CAM241}}$ is an Affibody molecule selected for binding to the extracellular domain of human recombinant CD69 as described previously (21). $Z_{\text{CAM241}}$ demonstrated nanomolar affinity ($K_D=34$ nM) to both human and murine recombinant CD69 by surface plasmon resonance. $Z_{\text{CAM241}}$ also demonstrated high thermal stability and refolding after denaturation, which is important for radiolabeling at harsh conditions, and Indium-111 labeled DOTA-$Z_{\text{CAM241}}$ demonstrated suitable biodistribution and properties for CD69 imaging in vivo (21).

$Z_{\text{CAM241}}$ is a 58-mer peptide, and like all Affibody molecules, consists of 3 alpha helices, where the binding motif is located on helix 1 and 2 (Supplemental Table 1) (21-22). A unique cysteine is added at the C-terminal to enable site specific conjugation. $Z_{\text{CAM241}}$ was generated by chemical solid phase peptide synthesis, and an $2,2',2'',2'''-(1,4,7,10$-Tetraazacyclododecane-$1,4,7,10$-tetrayl)tetraacetic acid (DOTA) chelator added to the C-terminal cysteine by malemide chemistry (Almac). DOTA- $Z_{\text{CAM241}}$ was purified by Reverse Phase-HPLC, freeze-dried and aliquoted into vials with 100 µg each.

$Z_{\text{AM106}}$ was used as a non-binding control peptide. It has the same size (58 residues plus a c-terminal cysteine) and tertiary structure as $Z_{\text{CAM241}}$, including three helices. However, $Z_{\text{AM106}}$ has 13 residues on the binding helices 1 and 2 altered compared to $Z_{\text{CAM241}}$ (Supplemental Table 1, previously unpublished). $Z_{\text{AM106}}$ instead binds to recombinant human DGCR2, which is absent from most healthy tissues and immune cells. The control, non-binding Affibody molecule DOTA-$Z_{\text{AM106}}$ was synthesized and analyzed (New England Peptides) in the same manner as described for DOTA-$Z_{\text{CAM241}}$ above.

**Radiolabeling of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$**

The $^{68}\text{Ge}/^{68}\text{Ga}$ generator (50 mCi, Cyclotron Co., Ltd, Russia) was eluted with 0.1 M HCl and the fraction with the highest activity (0.5 mL, ~200 MBq) was used for the labelling reaction. A stock solution of DOTA-$Z_{\text{CAM241}}$ was prepared by dissolving 100 µg of the Affibody molecule (Figure 1A) in 100 µL NaOAc buffer (1M, pH 4.6). An aliquot of 25 µL of the stock solution was transferred to a low protein binding vial and an additional 10 µL of NaOAc buffer was added, followed by 0.2 mL of the $^{68}\text{Ga}$ eluent (~80 MBq). The reaction mixture was heated at 75°C for 5 min and then cooled to room temperature before passing through a NAP-5 column which removed the unreacted $^{68}\text{Ga}$. The final purified product was formulated in PBS with 10% ethanol. The radiochemical yield (RCY) and
radiochemical purity of the resulting tracer $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ were assessed by HPLC using a C4 column with 10-100% acetonitrile/water at a flow rate of 1 mL/min for 10 minutes. Control, non-binding peptide $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{AM106}}$ was radiolabeled using the same protocol.

**In vivo biodistribution of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ in rat**

In vivo distribution was evaluated in healthy Sprague Dawley (SPD) rats, which is a standard strain and species to use for yielding data suitable for e.g. identify optimal time-point for imaging or to understand clearance mechanisms. We opted for a PET imaging-based approach to study the dynamic biodistribution, instead of the traditional ex vivo organ distribution design. The imaging approach has the benefit of substantially reducing the number of animals used ($n=3$ instead of $n>16$) as we obtain the full kinetic distribution in each scanned animal.

$[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ (13.8±2.2 MBq) was administered as a bolus in a catheter in the lateral tail vein of Sprague Dawley rats ($n=3$, 312±24g, male). Dynamic PET images were acquired from injection to 150 min using a small animal PET-MRI system (nanoPET/MRI, 3T, Mediso, Hungary). Dynamic whole-body PET scanning was performed using multiple whole-body sweeps (3 beds per pass; 2 x 5 min, 2 x 10 min, 4 x 30 min). After the PET acquisition, the animals were euthanized by sodium thiopental (Apoteket AB, Stockholm, Sweden) while still lying in the scanner. MRI sequences were then realized post-mortem for 30 min (Gradient Echo 3D Multi-FOV). PET images were reconstructed by the use of Maximum Likelihood Estimation Maximized (MLEM) algorithm (10 iterations). PET images were analyzed in PMOD 4.0 (PMOD Technologies, Zürich, Switzerland). Tissues and organs were segmented on PET summation images and co-registered MRI images. PET uptake was read out for all time points, decay corrected and expressed as Standardized Uptake Values (SUV).

**Plasma stability of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ in vivo in rat**

In vivo plasma stability of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ was evaluated in two additional rats. A target dose of 20 MBq $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ was administered intravenously in the tail vein in wake animals, who were then allowed back into their cage. After 1 or 2 h, one animal was euthanized by CO$_2$ followed by heart puncture. The blood was immediately collected in LiHep tubes and centrifuged at 4000 rpm for 5 minutes to separate the plasma. The plasma was mixed with ACN in a ratio 1:1 for precipitation, and the mixture was centrifuged at 16000g for 2 min (Eppendorf 5415R centrifuge) at 4 °C. The supernatant was filtered through a 2X0.2 μm nylon membrane (low protein binding). The
resulting plasma was injected to HPLC (5-10µl) for separation, and measured by both UV and radiodetector. The pellet, leftover plasma and filter was measured in well counter for recovery. The radiochromatogram peak corresponding to intact $[^{68}\text{Ga}]$Ga-DOTA-Z$\text{CAM}_{241}$ was measured by integration of the signal, and compared to all other peaks, and expressed as a percentage.

**In vivo biodistribution of $[^{68}\text{Ga}]$DOTA-Z$\text{CAM}_{241}$ in TCRb ko mice**

In order to understand the uptake and binding of $[^{68}\text{Ga}]$Ga-DOTA-Z$\text{CAM}_{241}$ in animals without induced inflammatory arthritis, and to identify the optimal timepoint for PET imaging in the main study, an ex vivo biodistribution study was performed in a relevant mouse strain (TCRb ko, n=7, 18.7±1.3g, female). Wake mice were administered a target dose of 2 MBq $[^{68}\text{Ga}]$Ga-DOTA-Z$\text{CAM}_{241}$ (1.9±0.2 MBq). Individual mice were euthanized after 5, 10, 20, 40, 60, 90 or 120 minutes, dissected and tissues (including hind paws, relevant tissue for the main study in inflammatory arthritis) were weighed and measured from radioactivity in a gamma counter. The tissue uptake was corrected for body weight and administered activity and expressed as SUV.

**Generation of CD69 transfected CHO cells**

The CHO-K1 cell line was purchased from ATCC and cultured in Ham’s F-12 (Biowest, Riverside MO, USA), 10% FBS (Sigma) and 1% Penicillin/Streptomycin (Merck Millipore). The human CD69 cDNA clone sequence was constructed from the NM_001781.2 NCBI Reference Sequence Database (RefSeq) and purchased from Genscript Biotech Corporation as a pcDNA3.1+/C-(K)DYK vector. The CHO-K1 cells were cultured to 80% confluence before transfection. The CD69 cDNA clone sequence was mixed with Lipofectamine 3000 reagents (Invitrogen, Waltham, Massachusetts, USA) and prepared according to the manufacturer’s guideline. Transfected CHO-K1 clones were selected using 1mg/mL of Geneticin (ThermoFisher) before moving to a pressure concentration of 0.4mg/mL to maintain clone selection.

Surface expression of CD69 was verified by fluorescence activated cell sorting (FACS) using APC conjugated anti-human CD69 antibody (Clone: FN50, Biolegend). The CHO-K1 and transfected CHO-CD69$^+$ cells were passaged twice a week and grown at 37°C in a humified air incubator with 5% CO$_2$. The media was changed every two days and the cells were harvested by using trypsin-ethylenediaminetetraacetic acid (Trypsin-EDTA).
In vitro binding of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ to CD69 positive CHO cells

Interactions between Z$_{\text{CAM241}}$ and human and murine CD69 at nanomolar concentrations was previously demonstrated using Surface Plasmon Resonance and confirmed using $[^{111}\text{In}]\text{In-DOTA-Z}_{\text{CAM241}}$ and CD3 activated human Peripheral Blood Mononuclear Cells (PBMCs) (21). In vitro binding of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ was here verified in a CD69 transfected CHO-K1 cell line.

Cells were initially seeded in poly-L-lysine coated Petri dishes 48h prior to the start of the assay. At the time of experiment, each dish contained a cell monolayer of approximately $10^6$ cells/dish. For blocking of binding, a subset CHO-CD69$^+$ cell dishes were pre-saturated with unlabeled Z$_{\text{CAM241}}$ precursor (200 nM, 250 μL) 10 min prior to adding the radioactive solution. On the remaining dishes 250 μL of complete Ham’s F-12 medium was added, in order to equalize the volume on all the dishes. Afterwards 250 μL of solution containing $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ corresponding 3 nM or 30 nM peptide was added to each dish. The dishes were incubated 1h at 37°C at 5% CO$_2$ in an incubator. Following the incubation, the media was aspirated, and the cells were washed with 1 ml of incomplete media. Subsequently, 500 μL of trypsin–EDTA solution was added for 10 min to detach the cells. After the trypsinization the cells were diluted in 1 ml of complete media, carefully resuspended and collected. An automated gamma counter (Wizard, PerkinElmer) was used to measure the radioactivity of the samples of which the percentage of cell-bound radioactivity was then defined.

Ethical considerations and longitudinal study design

The study design was a longitudinal imaging study to follow each mouse by four subsequent PET scans over 12 days; from before disease induction and during disease progression. A total of 8 animals were enrolled in the study. Five of the animals underwent longitudinal imaging from baseline, throughout the disease progression from induction to full blown inflammatory arthritis and euthanasia (followed by post-mortem phenotyping), thus enabling direct comparison within individuals. The remaining three individuals were sentinel animals used for biopsies and correlative staining, while undergoing a similar disease progression as the animals used for longitudinal PET scanning. The sentinel animals (n=3) were euthanized at three different time points (at baseline before disease induction and day 3 and 7 after induction, to provide representative biopsy material for staining to compare to imaging).

The study design was inspired by the 3R principles (Replace, Reduce, Refine) of humane animal research. The selected model of inflammatory arthritis has a predictable time-course which reduces the
number of animals required for reproducible results, as well as minimizing the risk of individuals reaching unacceptable severity of disease before the end of the study. The longitudinal imaging design is estimated to have reduced the number of animals in the study to n=8, compared to n=20 if ex vivo post-mortem analysis of similar group sized would have been used at the same time points (group sizes of n=5 multiplied with 4 time points). The number of splenocyte donor animals could also be reduced accordingly.

**Primers for genotyping transgenic mice**

The following primers were used for genotyping. KRN TCR alpha chain-forward: AGGTCCACAGCTCCTTCTGA, KRN TCR alpha chain-reverse: GTATTGGAAGGGGCCAGAG, KRN TCR beta chain-forward: GGGCAAAAAACTGACCTTGAA, KRN TCR beta chain-reverse: GAGCCTGGTTTGTGGAT, I-A<sup>8</sup> forward: TTCAAGGGCGAGTGCTACTT, I-A<sup>8</sup> reverse: GTTCGCTCCAGGTACTGCTT, I-A<sup>b</sup> forward: TTCATGGGCGAGTGCTACTT, I-A<sup>b</sup> reverse: CGTTCGCTCCAGGATCTC.

**PET scanning protocol in arthritic mice**

The background uptake of [<sup>68</sup>Ga]Ga-DOTA-Z<sub>CAM241</sub> (n = 5 female mice, weight: 20–23 g at the start of the study) was first established on PET-CT at baseline before induction of disease. Next, disease was induced by splenocytes transfer, as described above. Then each mouse (n = 5) was again examined by PET after 3 days, 7 days and 12 days post induction (p.i.) of inflammatory arthritis (Figure 1B). Co-registered MRI (baseline) or CT (Day 3, 7 and 12 days) scans was obtained in each scanning session, to provide anatomic information to support tissue segmentation. The MRI at baseline was changed to a CT at subsequent occasions as the CT provided much improved spatial resolution over the hind paw regions in shorter duration compared to the MRI sequence used. Before each scan, the mice were administered with [<sup>68</sup>Ga]Ga-DOTA-Z<sub>CAM241</sub> (target dose of 2 MBq) i.v. in tail vein and after 1 h, they were anesthetized with isoflurane 3% and placed on the camera bed with heated pad on. A 30-min static whole-body PET scan (nanoPETMRI, Mediso Medical Imaging Systems, Hungary, 283 × 283 × 321 matrix) was acquired, followed by an MRI scan (baseline; gradient echo 2D axial sequence, TR/TE 260/4.1 ms, 0.4-mm spatial resolution, 19 slices) or a CT scan (all other time points; 50 kVp, 610 μA) of 7 min. The timing of the PET imaging (from 60-90 minutes after injection of tracer) was guided by the results of the ex vivo biodistribution pilot study (Supplemental Figure 2B), which showed low
background at this time-point, while the remaining Gallium-68 signal was still high enough for
detection.

After the first three time points, the mice were observed an hour after the anesthesia before
returning to their housing. The mice were euthanized by CO₂ after the last time point. All PET images
were reconstructed for attenuation, dead time, positron range and scatter correction (Tera-Tomo 3D: 0.3-
mm³ spatial resolution, OSEM, Monte Carlo DOI estimation, 4 iterations, 6 subsets, voxel size 0.4 mm³.
The data was decay- and weight-corrected to SUV.

Data analysis of PET images of arthritic mice

The volumes of interest (VOIs) were manually segmented over the rear paws, on standard uptake
value (SUV) corrected axial projections using PMOD software (PMOD Technologies LLC, Zürich,
Switzerland). Uptake of PET tracer in tissue was presented as SUV_{mean}, i.e. the average SUV value of all
voxels in a segmented volume. SUV was defined as the uptake in tissue expressed as Bq/cc, normalized
for injected radioactivity (Bq) as well as body weight (g). Thus, SUV is a unitless value of PET tracer in
tissue which can be compared inter- and intra-individually.

The proximity of lymph nodes and spleen to regions with very high uptake (kidney and bladder)
made reproducible localization and segmentation challenging. However, the right and left axillary lymph
nodes could be segmented without interference from the excretory organs at the late time points. The
data was summarized and illustrated on GraphPad Prism (GraphPad Software Inc., La Jolla, CA, USA)
and presented as mean and standard deviation of the mean.

Correlation was assessed by Pearson’s correlation coefficient and simple linear regression.
Differences between were evaluated by the unpaired t-test where p < 0.05 was considered significant.

Histology of mouse biopsies

For each of the three first imaging time points (baseline, day 3 and day 7), one sentinel animal
(n=3 in total) was euthanized and the rear paws, the spleen, and the rear popliteal lymph nodes were
dissected and placed into separate cassettes (Q Path MicroStar 2, Avantor). Each of these biopsies were
assumed to present a similar histopathology as the animals used for longitudinal PET imaging, as they
followed a similar disease progression as evaluated by clinical severity scoring and weight loss. Finally,
biopsies were also taken postmortem for n=2 of the mice undergoing the longitudinal PET scanning after
euthanasia at day 12, to provide direct histopathological correlate to the last PET scan. For the remaining
n=3 mice euthanized at day 12, tissues (axillary, brachial, inguinal, mesenteric, and popliteal lymph nodes, as well as spleen and joint) were instead used for analysis of CD69 expression levels by flow cytometry after processing.

All biopsies were fixed in PFA for histology for 24 h and later transferred to 70% EtOH at 4°C. The paw biopsies were decalcified in 10% ethylenediaminetetraacetic acid (EDTA, MolDecal 10, Histolab, Gothenburg, Sweden) at a neutral pH for approximately 5 months at 4°C. All biopsies were then paraffin embedded, and then sectioned into 6 µm consecutive sections. Sections were immunostained for CD69 (anti-CD69, 8B6, Invitrogen) according to the manufacturers protocol. Briefly, the sections were de-paraffinized and blocked by 5% goat serum (30 minutes). Sections were incubated with the primary anti-CD69 antibody (1:175 dilution for 60 minutes). Next, the section was incubated with a goat anti-mouse secondary antibody containing a AF647 fluorophore (1:200 dilution for 60 minutes). Nuclear staining was performed by Sytox Orange (1:5000 dilution for 10 minutes). Sections were digitalized using a Vectra Polaris (Akoya Biosciences) slide scanner and visualized using QuPath (v0.3.2, University of Edinburgh).

Adjacent sections were stained with Masson’s trichrome (MTC) according to standard pathology protocols, dehydrated, mounted and analyzed by light microscopy (Leica) or digitalized by a slide scanner (PathScan Enabler IV).

**Enzyme-linked immunosorbent assay (ELISA)**

ELISA plate (Invitrogen, Cat. No. 44-2404-21) was coated with 50 µl of 10 µg/ml Glucose 6 Phosphate Isomerase from baker’s yeast (SIGMA, Cat. No. P5381) at 4°C overnight and diluted mouse sera were added into the plate. Subsequently, the plate was washed, and the HRP conjugated anti-mouse IgG (Santa Cruz Biotechnology, Cat. No. sc-2005) antibodies were added and incubated for 2 hours at room temperature. After the final wash, the TMB substrate (Medicago, Cat. No. 10-9405-250) was added, and the plate was read by ELISA reader.
Radiosynthesis of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ and $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{AM106}}$

The average activity yield of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ was 25±5 MBq when starting with ~80 MBq of $^{68}\text{Ga}$ elute. The RCY was 84±9% and the radiochemical purity was 98±1%. Stability testing showed that the purity was still >95% after 1 h with the addition of EDTA. The molar activity ($A_M$) was 7±2 MBq/nmol at the end of synthesis. The average RCY of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{AM106}}$ was 84±9% and the radiochemical purity was 98±1%.

In vitro and in vivo characterization of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$

$[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ demonstrated rapid tissue distribution in rat, with rapid clearance from circulation (Supplemental Figure 1 and 2A). Only low background remained in most tissues after 60 minutes, while being almost non-existent after 150 minutes. $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ exhibited strong and irreversible retention in the renal cortex, which is often seen for Affibody molecules (or other proteins of similar size) labeled with residualizing radiometals such as Gallium-68 (Supplemental Figure 1B and 2A). Further release of radioactivity into bladder was also notable. Liver also exhibited some retention, as well as lymph nodes. The binding in joints and bone was low, a prerequisite for imaging of immune cells in models of inflammatory arthritis (Supplemental Figure 1A and 2A).

The stability of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ in rat plasma in vivo was suitable for further tracer development, with around 80% intact tracer seen after 60 minutes and 60% after 120 minutes (Supplemental Figure 2B).

The biodistribution in TCRb ko mice was consistent with that seen in rats, with progressive clearance of the background and circulatory signal over time (Supplemental Figure 2C). The background signal in hind paws reached an acceptable low background after 60 minutes. Thus, the 60 minutes time-point was deemed as optimal for PET imaging and potential image contrast, both based on the biodistribution in rat and mice, and considering the high stability of the tracer at this point.

CD69 transfected cells demonstrated binding of $[^{68}\text{Ga}]\text{Ga-DOTA-Z}_{\text{CAM241}}$ at 3 nM, blockable by preincubation with 200 nM DOTA-$Z_{\text{CAM241}}$ which is in agreement with the binding seen with previous analogues as well as unlabeled $Z_{\text{CAM241}}$, confirming that Gallium-68 radiolabeling retained binding affinity towards CD69 (Supplemental Figure 2D).
Supplemental Figures and Tables

Supplemental Table 1. Overview of the primary amino acid sequences of Affibody molecules $Z_{\text{CAM241}}$ and $Z_{\text{AM106}}$. Green shading indicates residues involved in helical tertiary structures. White shading indicates constant residues. Yellow shading indicates variable residues (i.e. the binding motif).

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A  Tracer validation
- PET and ex vivo biodistribution in rat + mouse
- In vivo stability
- Binding to CD69+ cells

B  Evaluation in inflammatory arthritis

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Supplemental Figure 1. (A) Schematic structure of the CD69 targeting Affibody molecule $Z_{\text{CAM241}}$ and the basic tracer validation described in this report. (B) Overview of the study design of the PET study using $[^{68}\text{Ga}]\text{Ga-DOTA-}Z_{\text{CAM241}}$ to detect CD69 cells in a model of inflammatory arthritis.
Supplemental Figure 2. Representative biodistribution of $^{68}$GaDOTA-ZCAM241 in rat as evaluated by PET/MRI imaging from injection to 150 minutes later. Panels show the distribution over time at coronal projections at the level of the liver/ bladder (A, all images normalized to SUV 5) and kidney (B, all images normalized to SUV 60). Ln stands for lymph nodes, Li stands for liver, Bl stands for bladder and Ki stands for kidney.
Supplemental Figure 3. The in vivo biodistribution of [68Ga]DOTA-Z\textsubscript{CAM241} in rats quantified as SUV (A). Stability in plasma of [68Ga]DOTA-Z\textsubscript{CAM241} in rat over two hours after injection (B). In vivo biodistribution of [68Ga]DOTA-Z\textsubscript{CAM241} in TCRb ko mice as assessed by ex vivo organ uptake by gamma counter for up to 120 minutes post injection (C). In vitro binding of [68Ga]DOTA-Z\textsubscript{CAM241} to CD69 transfected CHO cells, either tracer alone or after blocking with different amounts of unlabeled DOTA-Z\textsubscript{CAM241} (D).
Supplemental Figure 4. Baseline/ day 0 is indicated by black dots, the day 3 time point is indicated by red dots, the day 7 time point by blue dots and the day 12 time point by grey dots, n=5. Each point indicates an individual animal examined at an individual time point. Correlation was assessed by Pearson’s correlation coefficient. The uptake of control non-binding peptide $[^{68}\text{Ga}]\text{Ga-DOTA-ZAM106}$ as a function of time after induction of disease (A). Correlation of the $[^{68}\text{Ga}]\text{Ga-DOTA-ZAM106}$ uptake to clinical score before induction (black dots), and 3 days (red dots), 7 days (blue dots) or 10 days (gray dots) after the induction of disease (B). Each point indicates an individual animal examined at an individual time point. Correlation was assessed by Pearson’s correlation coefficient. Characterizing the induced joint inflammation in the KRN T cell adoptive transfer model, in the group examined by non-binding peptide $[^{68}\text{Ga}]\text{Ga-DOTA-ZAM106}$. (C) Joint inflammation from baseline to last PET scan (clinical score, 0-12; 0-3 points/paw). (D) Weight change from baseline after induction of inflammatory arthritis shown as % reduction. (E) Representative CD69 fluorescent immunostaining from joint in animals scanned with the non-binding peptide $[^{68}\text{Ga}]\text{Ga-DOTA-ZAM106}$. 
Supplemental Figure 5. Fluorescence CD69 staining of lymph node at baseline (E) and 12 days p.i. induction of inflammatory arthritis (F).