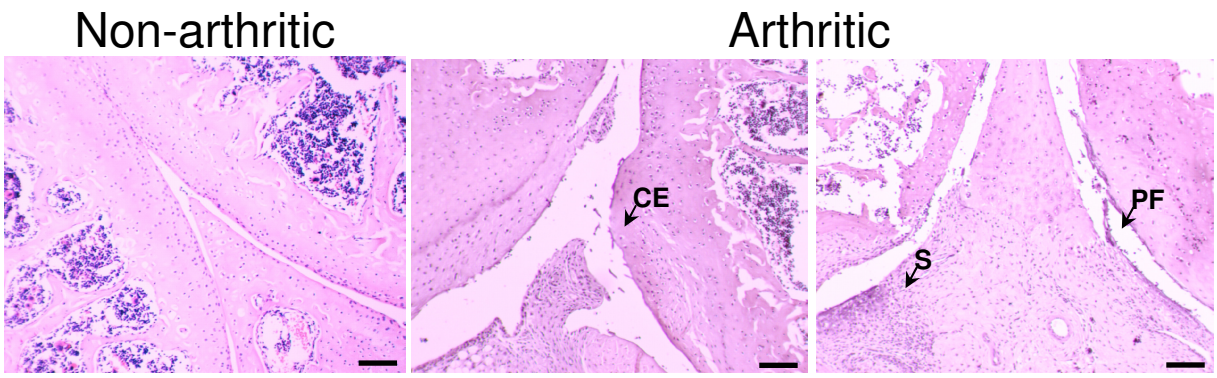
Figure S1

Figure S1. H&E staining of a normal non-arthritic and KxB/N arthritic knee joint



Mice were given an i.p. injection of 50 µl at day 0 and day 2 (50+50 µl) K/BxN serum. Knees of naïve and 50+50 µl K/BxN serum group, taken at day 20 endpoint were decalcified and paraffin embedded. Sections were stained by H&E. Representative images are shown here. Scale bars; 60 µM

Table S1

Table S1. Arthritic disease incidence following low volume K/BxN serum administration

Time (days)	50+50 µl K/BxN serum	200 µl K/BxN serum
0	0% (0/8)	0% (0/8)
6	100% (8/8)	85.7% (6/7)
10	75% (3/4)	66.6% (2/3)
20	0% (0/4)	0% (0/3)

Data report the number of mice with a severe arthritic score, taking a threshold ≥ 8. Mice were given an i.p. injection of 50 µl at day 0 and day 2 (50+50 µl)) or 200 µl at day 0 of K/BxN serum**.**

Table S2

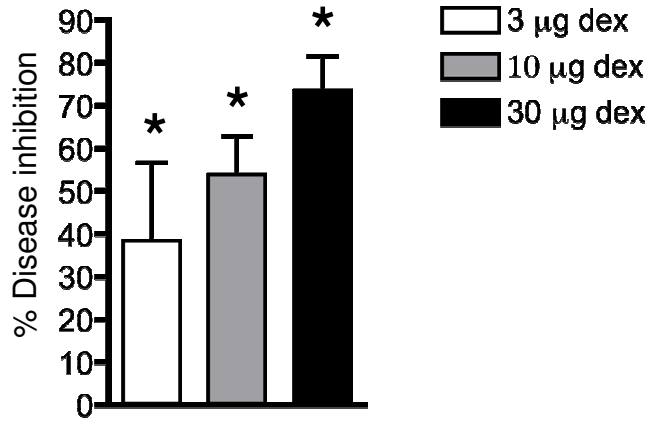
Table S2. Expression of elements of the AnxA1 pathway in naïve ankle joints by quantitative PCR (data normalised to housekeeping genes *Gapdh* or *Rpl32*)

Gene of Interest	Cycle threshold (Ct)	Difference normalised to *Gapdh* (ΔCt)	Difference normalised to *Rpl32* (ΔCt)
Gapdh	18.6 ± 0.32	---	---
Rpl32	18.4 ± 0.16	---	---
Fpr1	30.7 ± 0.69	12.1 ± 0.73	12.3 ± 0.60
Fpr2	30.4 ± 0.68	11.8 ± 0.77	12.0 ± 0.57
Anxa1	21.0 ± 0.15	2.4 ± 0.42	2.6 ± 0.05

Data report the gene expression of elements of the AnxA1 pathway. Naïve mice were culled and quantitative real-time PCR was performed on cDNA from left ankle joints. Data are mean ± SE of 4 mice per group. Ct values were normalised to endogenous *Gapdh* and *Rpl32.*

Figure S2

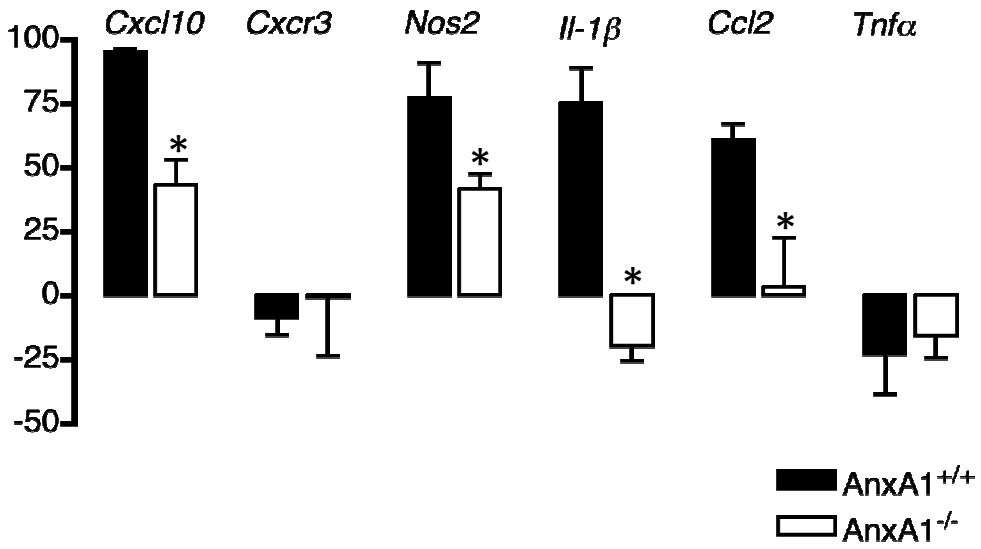
Figure S2. Inhibition of K/BxN arthritis by dexamethasone



Data report percentage disease inhibition in Dex groups relative to vehicle control at day 6. Mice were given an i.p. injection of 50 μl at day 0 and day 2 (50+50 μl)) of K/BxN serum and then received vehicle or Dex (3, 10 or 30 μg i.p. daily).

Figure S3

Figure S3 Dexamethasone attenuates K/BxN arthritis in AnxA1$^{+/+}$ but not AnxA1$^{-/-}$ mice (data normalised to housekeeping gene *Rpl32)*



Mice were given an i.p. injection of 50 µl at day 0 and day 2 (50+50 µl) K/BxN serum and then received vehicle or Dex (10 µg i.p. daily). Quantitative real-time PCR was performed on cDNA from left ankle joints of mice from each experimental group (Day 10); pro-inflammatory genes were analysed with all Ct values normalised to endogenous *Rpl32* (4 mice per group). RQ values were calculated using $2^{-(\Delta\Delta Ct)}$ and data shown here as mean % gene inhibition by Dex relative to vehicle control groups ± SE. Naïve joints were set as the calibrator samples. *p<0.05 *vs.* respective vehicle control (Student's *t* test).

Table S3

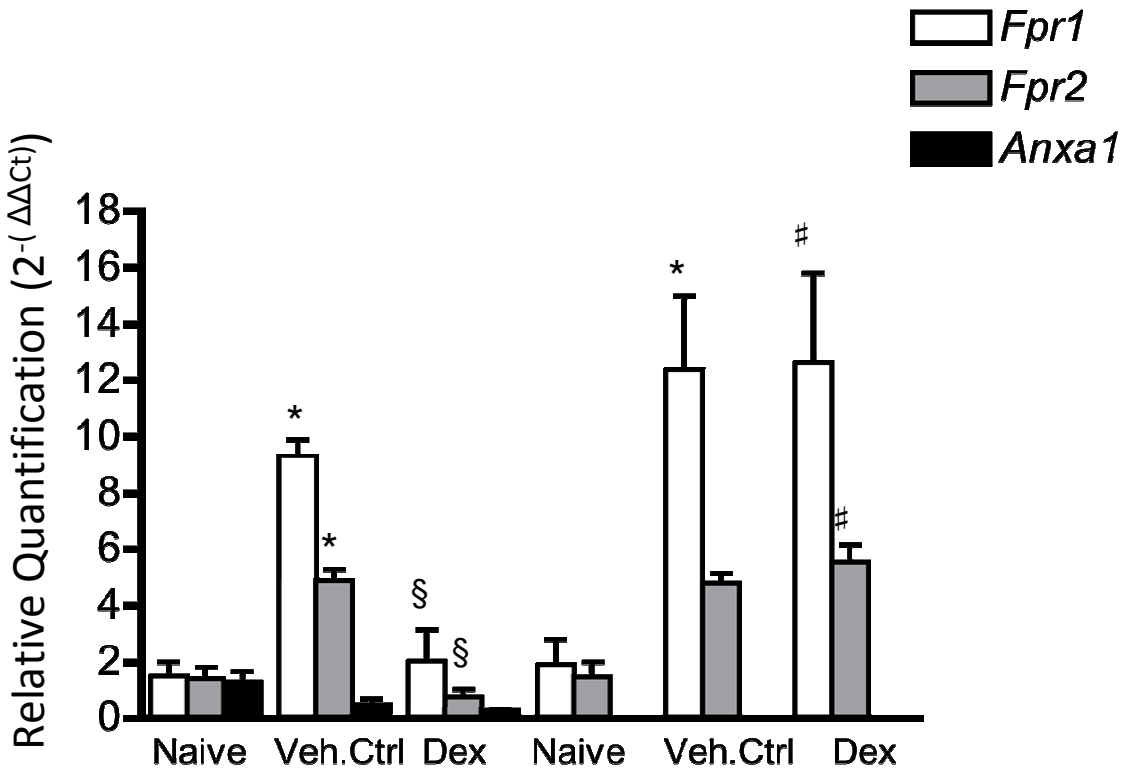
Table S3. Arthritic disease incidence in AnxA1$^{+/+}$ and AnxA1$^{-/-}$ mice treated with Dex

Time (days)	AnxA1$^{+/+}$ + Vehicle control	AnxA1$^{+/+}$ + Dexamethasone	AnxA1$^{-/-}$ + Vehicle control	AnxA1$^{-/-}$ + Dexamethasone
0	0% (0/3)	0% (0/4)	0% (0/4)	0% (0/4)
6	66.7% (2/3)	0% (0/4)	75% (3/4)	75% (3/4)
10	66.7% (2/3)	0% (0/4)	75% (3/4)	25% (1/4)

Data report the number of mice with a severe arthritic score, taking a threshold ≥ 8. AnxA1$^{+/+}$ and AnxA1$^{-/-}$ mice were given an i.p. injection of 50 µl at day 0 and day 2 (50+50 µl) and then received vehicle or Dex (10 µg i.p. daily).

Figure S4

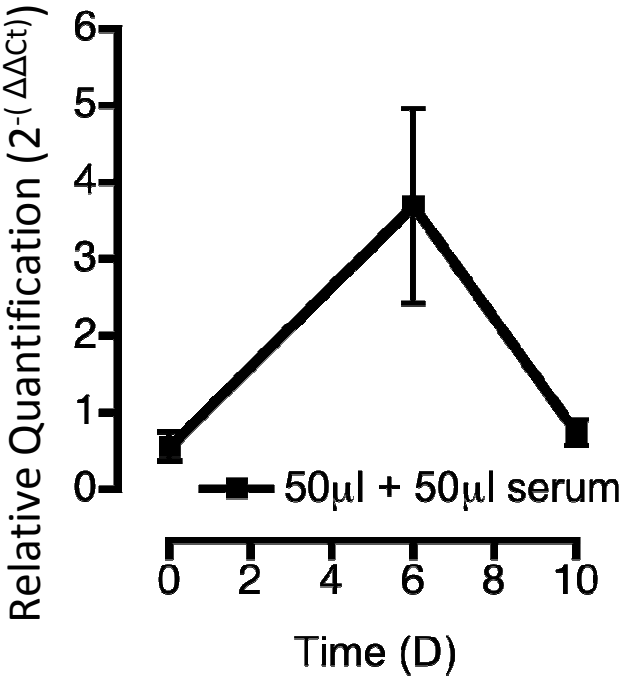
Figure S4. Profile of *Anxa1*, *Fpr1* and *Fpr2* gene product expression in ankle joints of AnxA1$^{+/+}$ and AnxA1$^{-/-}$ mice treated with Dex mice (data normalised to housekeeping gene *Rpl32)*



AnxA1$^{+/+}$ and AnxA1$^{-/-}$ mice were given an i.p. injection of 50 µl at day 0 and day 2 (50+50 µl) K/BxN serum and then received vehicle or Dex (10 µg i.p. daily). Quantitative real-time PCR was performed on cDNA from left ankle joints of mice from each experimental group (Day 10); The *Anxa1*, *Fpr1* and *Fpr2* genes were analysed with all Ct values normalised to endogenous *Rpl32* (4 mice per group). Mean RQ values ± SE were calculated using $2^{-(\Delta\Delta Ct)}$ method. Naïve joints were set as the calibrator samples. *p<0.05 *vs.* respective vehicle control (Student's *t* test). *p<0.05 *vs.* naïve; §p<0.05 *vs.* appropriate vehicle control; $^{\#}$p<0.05 *vs.* AnxA1$^{+/+}$ group (Student's *t* test).

Figure S5

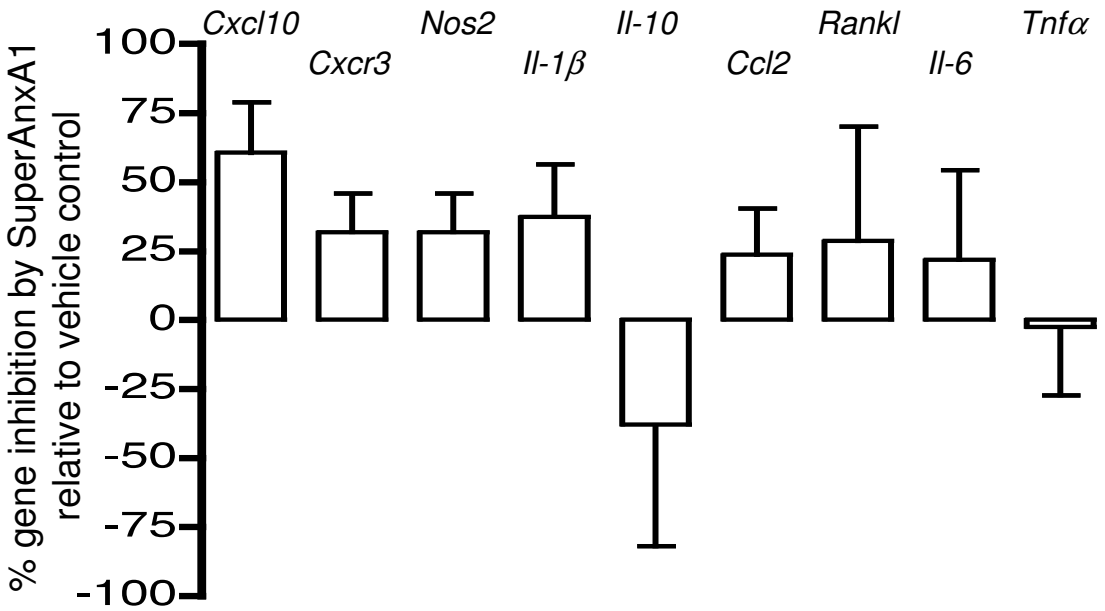
Figure S5. Profile of *Pr3* gene product expression in ankle joints of AnxA1$^{+/+}$ mice over time-course of K/BxN time-course (data normalised to housekeeping gene *Rpl32)*



AnxA1$^{+/+}$ mice were given an i.p. injection of 50 µl at day 0 and day 2 (50+50 µl) K/BxN serum. Quantitative real-time PCR was performed on cDNA from left ankle joints of mice at day 0 (naive), day 6 and day 10; the *Pr3* gene was analysed with all Ct values normalised to endogenous *Rpl32* (6 mice per group). Mean RQ values ± SE were calculated using $2^{-(\Delta\Delta Ct)}$ method. Naïve joints were set as the calibrator samples.

Figure S6

Figure S6. SuperAnxA1 attenuates pro-inflammatory mediators in K/BxN arthritic ankle joints



Mice were given an i.p. injection of 50 µl at day 0 and day 2 (50+50 µl) K/BxN serum and then received vehicle control or SuperAnxA1 (1 µg i.p. daily) (n=6 per group). Quantitative real-time PCR was performed on cDNA from left ankle joints of mice from each experimental group (Day 10); pro-inflammatory genes were analysed with all Ct values normalised to endogenous *Gapdh* (six mice per group). RQ values were calculated using $2^{-(\Delta\Delta Ct)}$ and data shown here as mean % gene inhibition by SuperAnxA1 relative to vehicle control group ± SE.