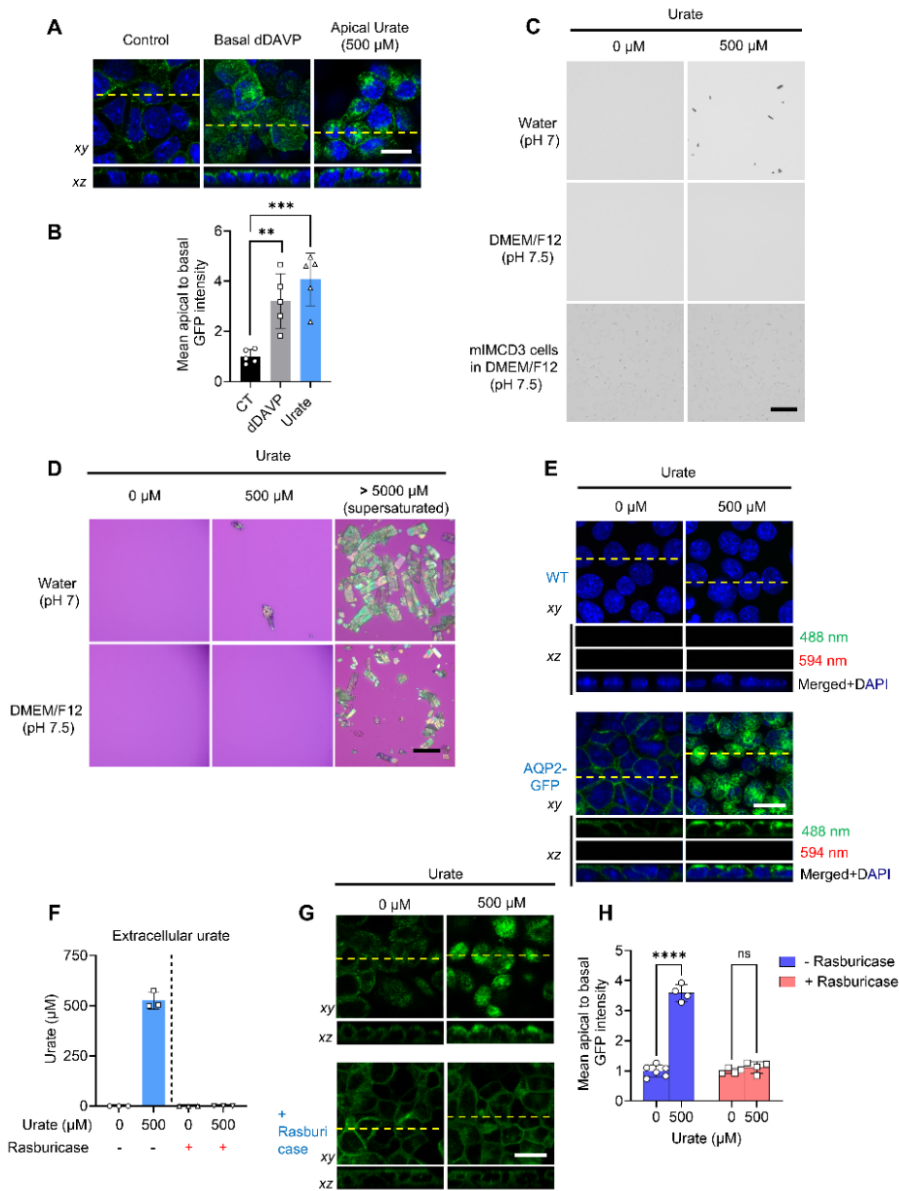


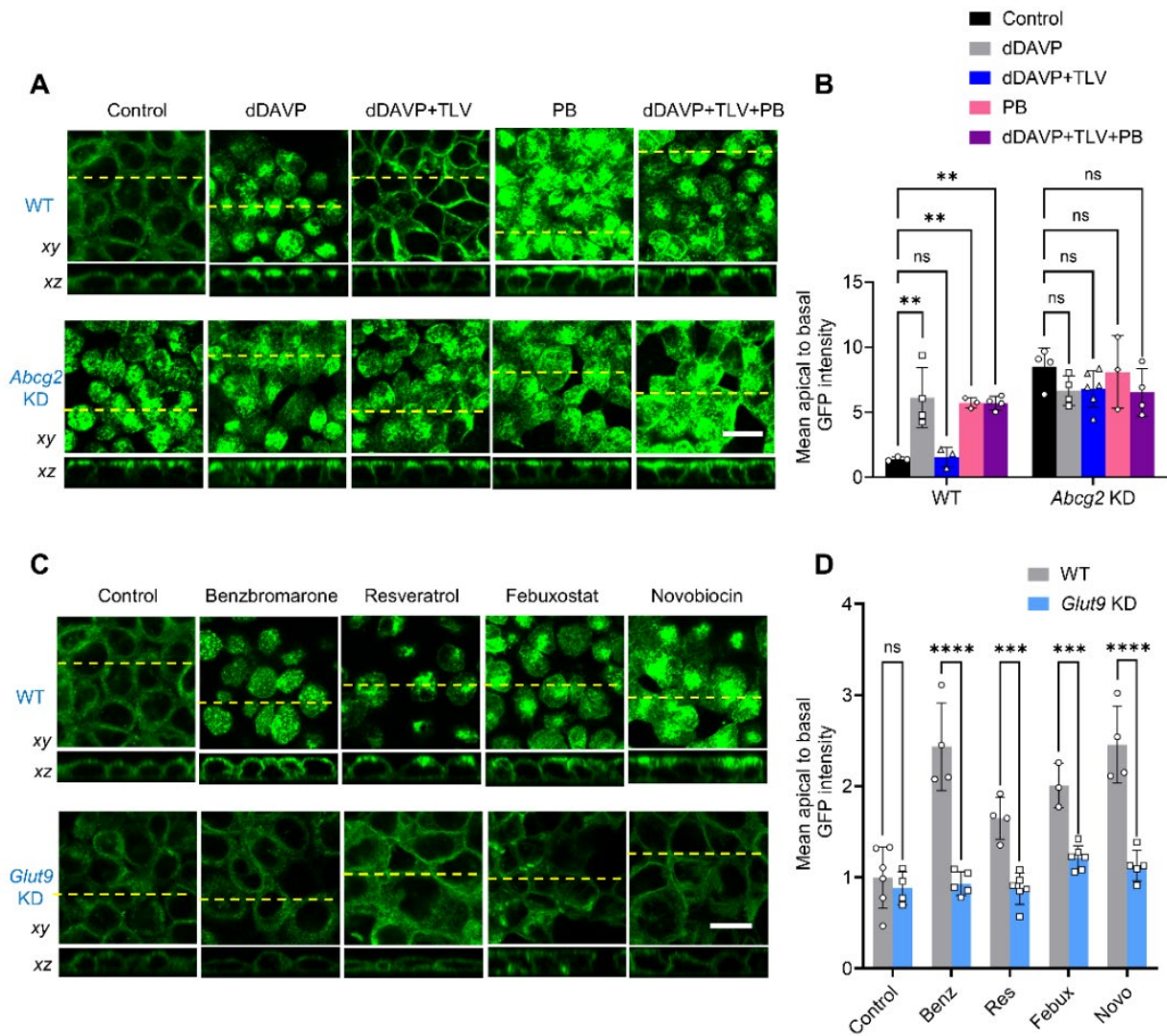
Supplementary materials

- **Supplementary Figure 1:** Urate solubility and specificity controls for the *in vitro* assays and validation of genetic modifications in mIMCD3 cells.
- **Supplementary Figure 2:** Additional validation of urate-induced AQP2 trafficking.
- **Supplementary Figure 3:** ABCG2 inhibition and probenecid increase intracellular urate and promote AQP2 apical accumulation in a GLUT9-dependent manner.
- **Supplementary Figure 4:** Validation of urate-induced AQP2 trafficking in MDCK cells and characterization of transporter localization in human tissue, overexpression, and off-target controls.
- **Supplementary Figure 5:** Urate-induced AQP2 trafficking is independent of PANX1 and of NLRP3.
- **Supplementary Figure 6:** Urate-induced AQP2 trafficking is independent of UT-A.
- **Supplementary Figure 7:** Intracellular urate accumulation activates AMPK and requires intact vesicle trafficking for AQP2 apical accumulation.
- **Supplementary Figure 8:** Probenecid attenuates tolvaptan-induced aquaresis without altering kidney volume or renal function.
- **Supplementary Figure 9:** Design and biochemical outcomes of the SereNDIpity-pb1 clinical trial evaluating probenecid in ADPKD patients on tolvaptan.
- **Supplementary Table 1:** Clinical characteristics, adverse events, and outcomes from the SereNDIpity-pb1 trial.
- **Supplementary Table 2:** CONSORT 2025 Checklist for SereNDIpity-pb1 clinical trial.
- **Supplementary Table 3:** Primer sequences used in gene expression and mutagenesis studies.
- **Supplementary Table 4:** Commercial antibodies used for immunoblotting experiments.

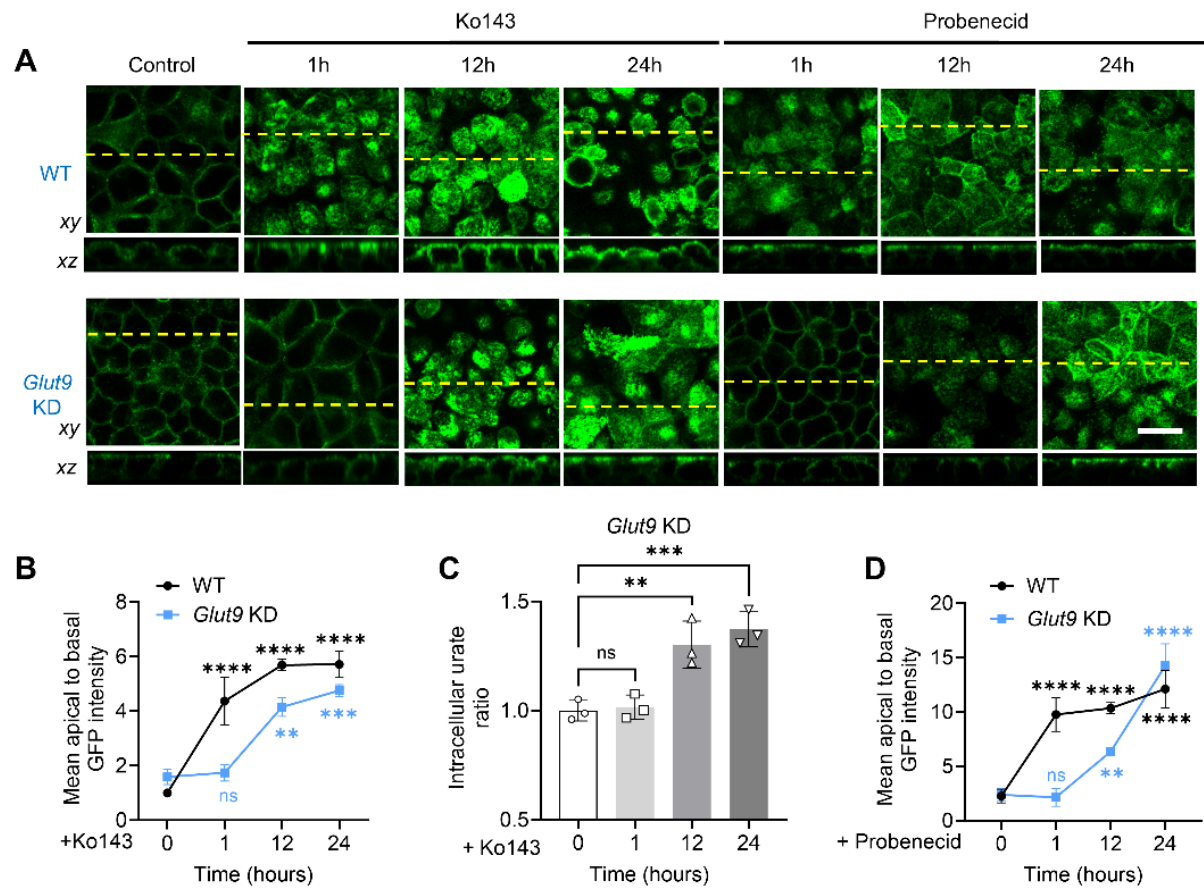
Supplementary Figure 1. Urate solubility and specificity controls for the *in vitro* assays and validation of genetic modifications in mIMCD3 cells. (A) Representative confocal images of apical AQP2 and (B) corresponding apical-to-basal fluorescence intensity (FI) ratios showing apical AQP2 trafficking in AQP2-OE mIMCD3 cells treated for 1 h with basal dDAVP (100 nM) and apical urate (500 μ M). (C) Bright-field imaging mIMCD3 cultures also showed absence of urate crystal formation. (D) Dissolved urate at 500 μ M formed birefringent crystals as observed in polarized light only in water but not in DMEM/F12, whereas supersaturated urate (>5 mM) produced crystals in both conditions. These results confirmed absence of crystal formation under the conditions used for cell experiments. Urate solutions were prepared in milli-Q water (pH 7) or serum-free DMEM/F12 (pH 7.5) at 37 $^{\circ}$ C with 1 h continuous agitation before imaging (E) Confocal imaging demonstrated that non-transfected cells treated for 1 h with 500 μ M urate exhibited no detectable fluorescence emission at 488 nm or 594 nm, and that the signal observed in AQP2-GFP cells treated 1 h with 500 μ M urate reflects AQP2-GFP redistribution rather than urate autofluorescence. (F, G) Urate-induced apical AQP2 accumulation over 1 h was prevented by 30 min pretreatment with rasburicase (5 μ g/mL), confirming the requirement for intact urate. (H) Corresponding apical-to-basal FI ratios for (G). All urate treatments were in FBS-free medium. $n \geq 3$ biological replicates. Statistical significance was determined by one-way ANOVA with Dunnett correction in (B), 2-way ANOVA with Dunnett correction in (H). ** P value < 0.01, *** P value < 0.001, **** P value < 0.0001. Scale bar: 25 μ m.



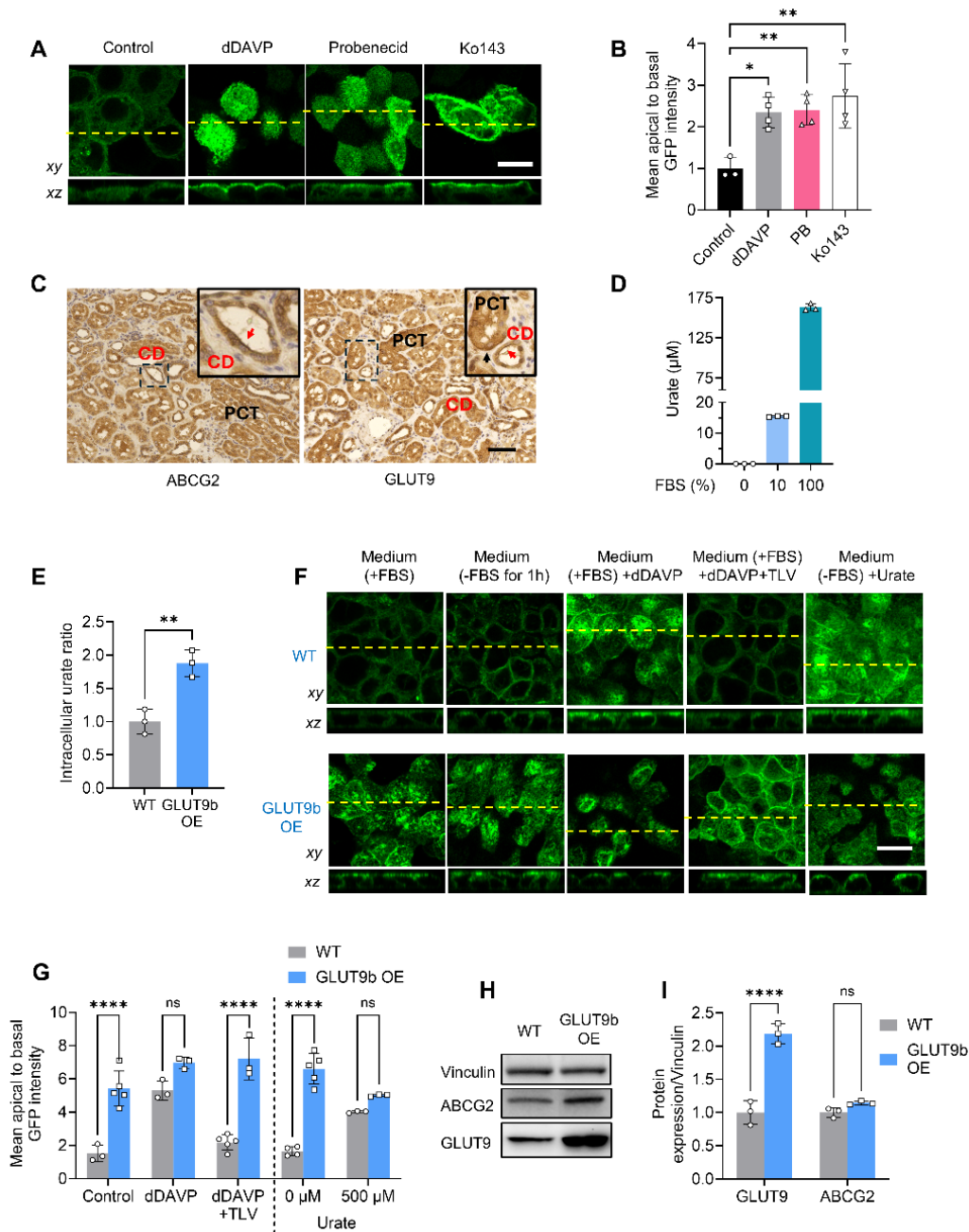
Supplementary Figure 2. Additional validation of urate-induced AQP2 trafficking. (A) Representative confocal images of AQP2-GFP localization in WT and *Abcg2* KD cells in the absence or presence of dDAVP, tolvaftan (TLV) or probenecid (PB), with (B) normalized apical-to-basal FI ratios of AQP2-GFP showing that *ABCG2* KD induces apical accumulation of AQP2. (C) Representative confocal images of apical AQP2-GFP localization in wild-type (WT) and *Glut9* knockdown (KD) mIMCD3 cells treated with ABCG2 inhibitors benzbromarone (Benz, 10 μ M), resveratrol (Res, 0.5 μ M), febuxostat (Febux, 100 μ M), or novobiocin (Novo, 100 μ M). (D) Normalized apical-to-basal FI ratios demonstrating that apical AQP2 trafficking is abolished in GLUT9-deficient cells. Panels (A) and (C) share the same WT control, as experiments were performed in parallel. $n \geq 3$ biological replicates. Statistical significance in (B and D) was determined by 2-way ANOVA with Bonferroni correction. $**P < 0.01$, $***P < 0.001$, $****P < 0.0001$. Scale bar: 25 μ m.



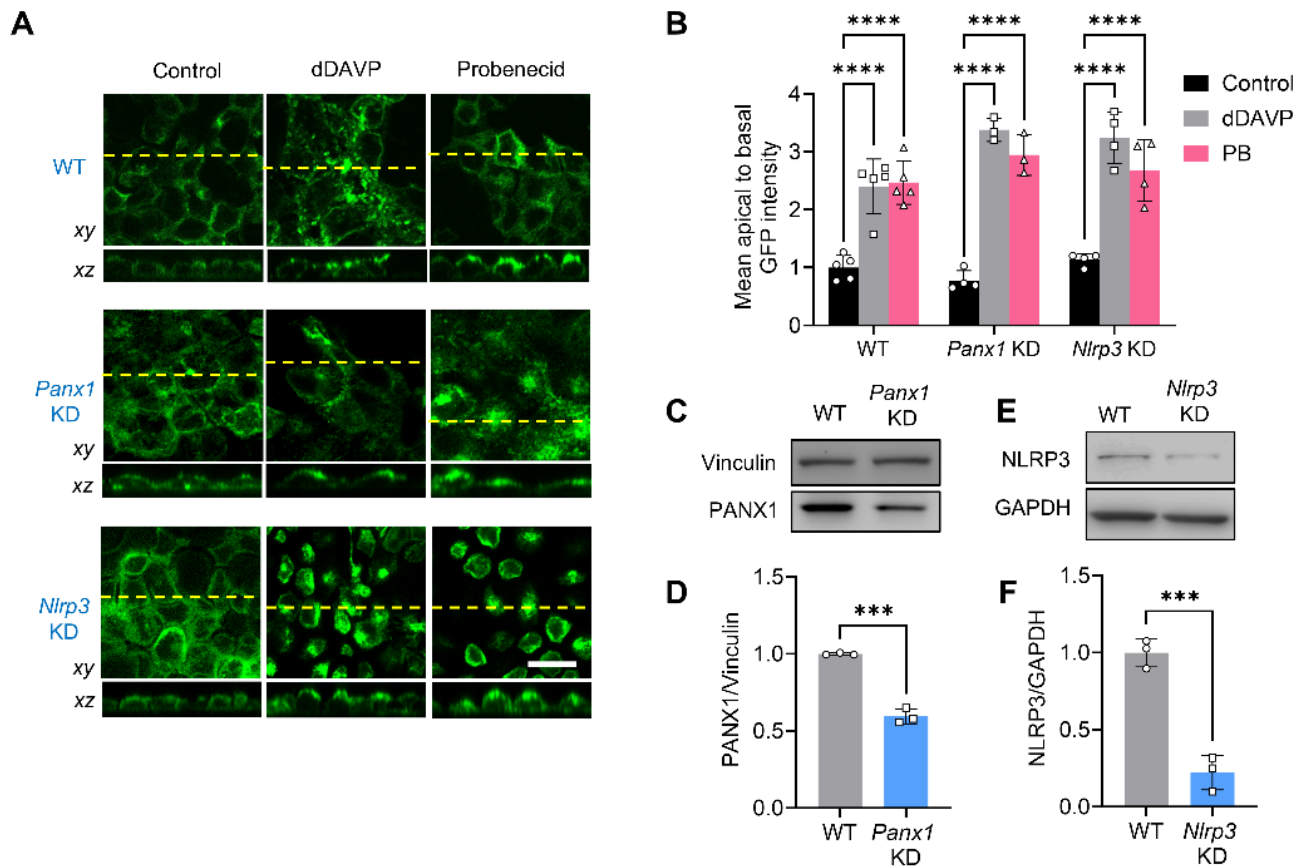
66 **Supplementary Figure 3. Additional validation of urate-induced AQP2 trafficking.** (A) Confocal images of AQP2-GFP
67 localization in representative WT or *Glut9* knock down cells in the absence (control) or presence of Ko143 or probenecid
68 for 1, 12 or 24 h. **B** and **D** show normalized apical-to-basal FI and (**C**) normalized intracellular urate measurements,
69 confirming gradual urate accumulation and corresponding apical AQP2 accumulation over 12–24 hours. $n \geq 3$ biological
70 replicates. Statistical significance was determined by 2-way ANOVA with Dunnett correction. $**P < 0.01$, $****P < 0.0001$.
71 Scale bar: 25 μm .



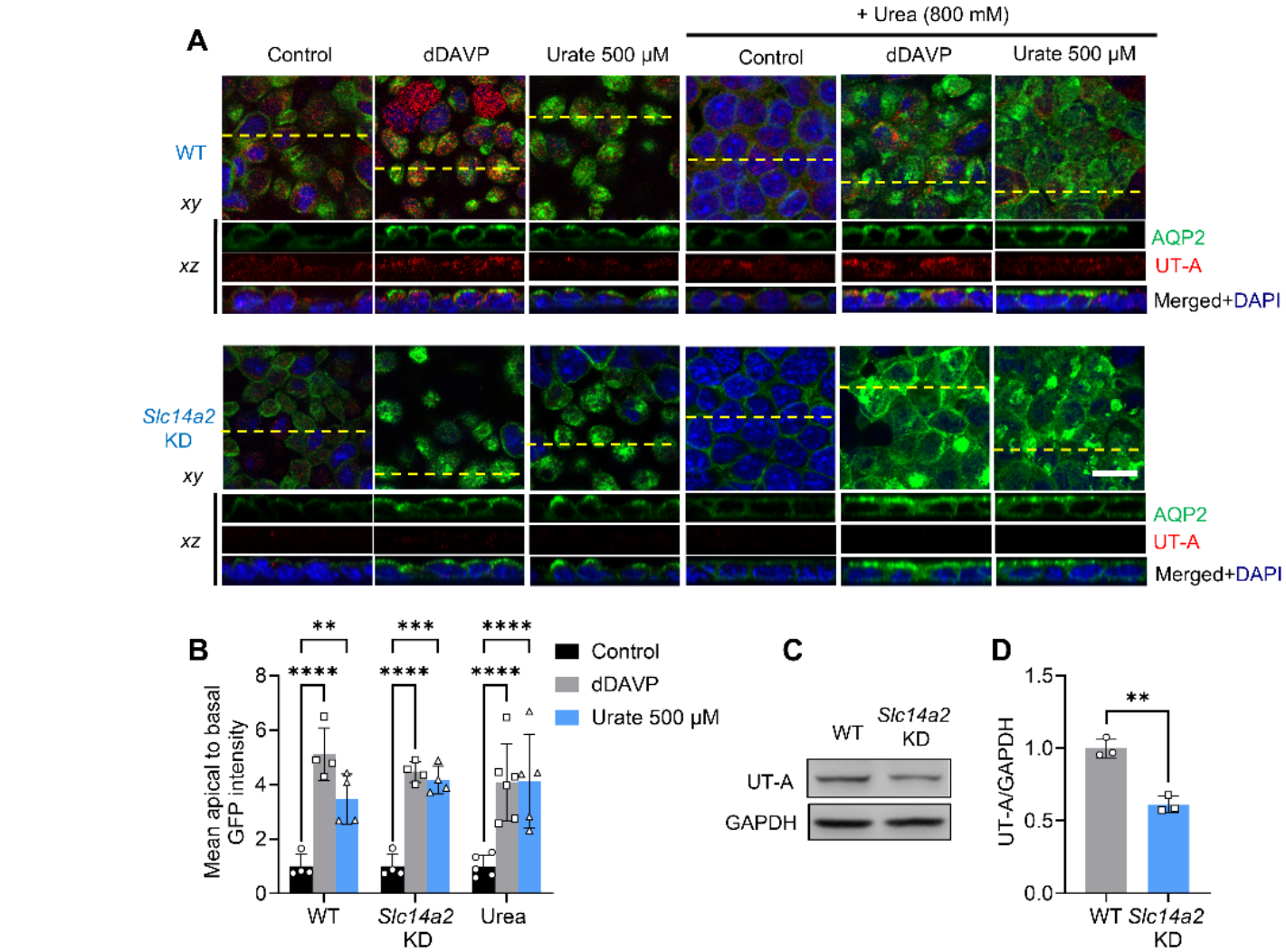
Supplementary Figure 4. Validation of urate-induced AQP2 trafficking in MDCK cells and characterization of transporter localization in human tissue, overexpression and off-target controls. (A) Confocal images of representative polarized MDCK cells expressing AQP2–GFP after 1 h treatment with dDAVP (100 nM), Probenecid (100 μ M), or Ko143 (1 μ M). **(B)** Normalized apical-to-basal FI demonstrating apical AQP2 accumulation across species. **(C)** Immunohistochemically stained human kidney sections showing ABCG2 localization at collecting duct (CD) luminal surface and GLUT9 expression in both CD and proximal convoluted tubule (PCT) basolateral membrane. Scale bar, 75 μ m. **(D)** Urate quantitation in 10% and 100% FBS. **(E)** GLUT9b overexpression in mIMCD3 cells increased intracellular urate and **(F, G)** induced spontaneous apical localization of AQP2. **(H)** Western blot validation of GLUT9b overexpression and **(I)** corresponding mean densitometric quantitation, with accompanying increase in ABCG2 expression. All urate treatments were performed in FBS-free medium. $n \geq 3$ biological replicates. Statistical significance was determined by unpaired t test for **(E)**, one-way ANOVA with Dunnett correction for **(B)**, and 2-way ANOVA with Bonferroni correction for **(G and I)**. $**P < 0.01$, $****P < 0.0001$. Scale bar: 25 μ m unless otherwise indicated.



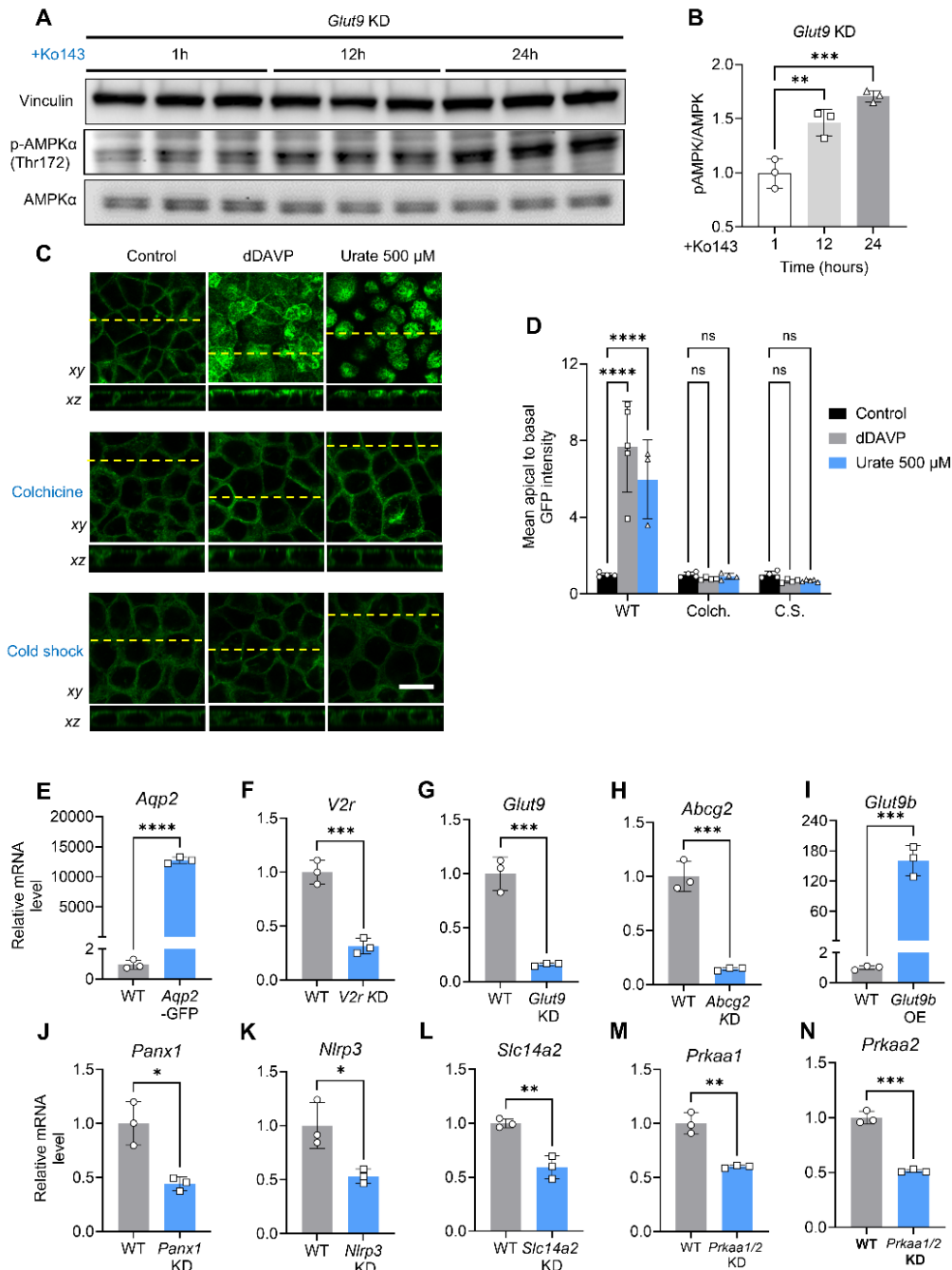
86 **Supplementary Figure 5. Urate-induced AQP2 trafficking is independent of PANX1, NLRP3 (A)** Confocal images of
 87 representative cells with *Panx1* or *Nlrp3* knockdown treated with dDAVP (100 nM) and urate (500 μ M) for 1 h and (B)
 88 normalized apical-to-basal FI ratios for (A). (C, E) Representative immunoblots and (D, F) mean densitometric quantitation
 89 confirming efficient siRNA knockdown of (C, D) PANX1 and (E, F) NLRP3. $n \geq 3$ biological replicates. Statistical significance
 90 was determined by unpaired *t* test for (D and F) and by 2-way ANOVA with Dunnett correction for (B). $**P < 0.01$, $***P <$
 91 0.001 and $****P < 0.0001$. Scale bar, 25 μ m.



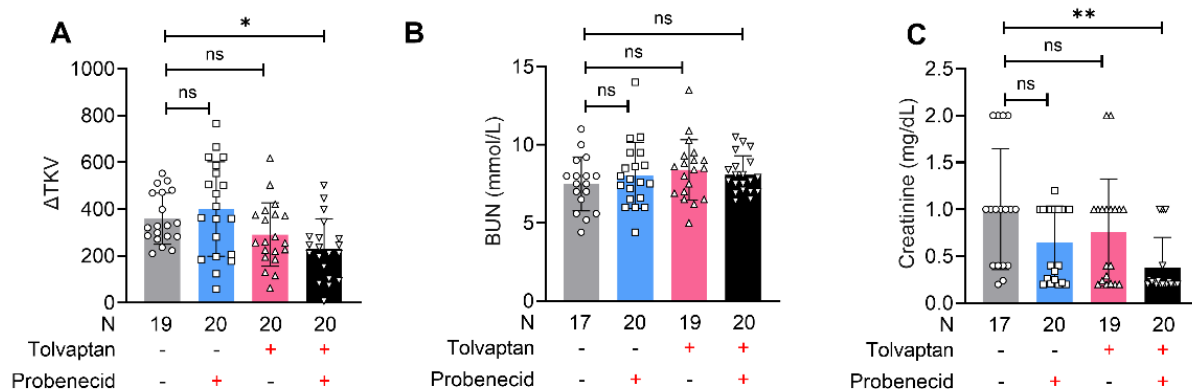
93 **Supplementary Figure 6. Urate-induced AQP2 trafficking is independent of UT-A.** (A) Confocal images of
 94 representative WT and *Slc14a2* KD cells treated for 1 h with dDAVP (100 nM) and urate (500 μ M) in the absence or presence
 95 of 800 mM urea. (B) Corresponding normalized apical-to-basal FI ratios, showing apical AQP2 redistribution in both cell
 96 lines. (C) Representative immunoblot and (D) mean densitometric quantitation confirming *Slc14a2* knockdown. $n \geq 3$
 97 biological replicates. Statistical significance was determined by 2-way ANOVA with Bonferroni correction for (B) and by
 98 unpaired *t* test for (D). $**P < 0.01$, $***P < 0.001$; ns, not significant. Scale bar, 25 μ m.



Supplementary Figure 7. Intracellular urate accumulation activates AMPK and requires intact vesicle trafficking for apical accumulation of AQP2. (A) Immunoblot and (B) densitometric quantitation of total AMPK α and p-AMPK α (Thr172) after 12 h urate treatment, showing elevation in pAMPK/AMPK ratio. (C) Confocal images and (D) corresponding normalized apical-to-basal FI of representative mIMCD3 AQP2-GFP cells, showing that 30 min pre-treatment with colchicine (Colch, 10 μ M) or acute cold shock (C.S.) abolishes AQP2 apical translocation induced by 1 h treatment with dDAVP (10 nM) or urate (500 μ M). (E-N) qPCR analyses validated targeted knockdowns using siRNA or overexpression of *Aqp2*, *V2r*, *Glut9*, *Abcg2*, *Panx1*, *Nlrp3*, *Slc14a2*, *Prkaa1*, and *Prkaa2* in the modified cell lines studied, with *Gapdh* as “housekeeping” normalization control gene. $n \geq 3$ biological replicates. Statistical significance was determined by unpaired *t* test for (E–N), one-way ANOVA with Dunnett correction for (B) and 2-way ANOVA with Dunnett correction for (D). * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$, **** $P < 0.0001$. Scale bar, 25 μ m.

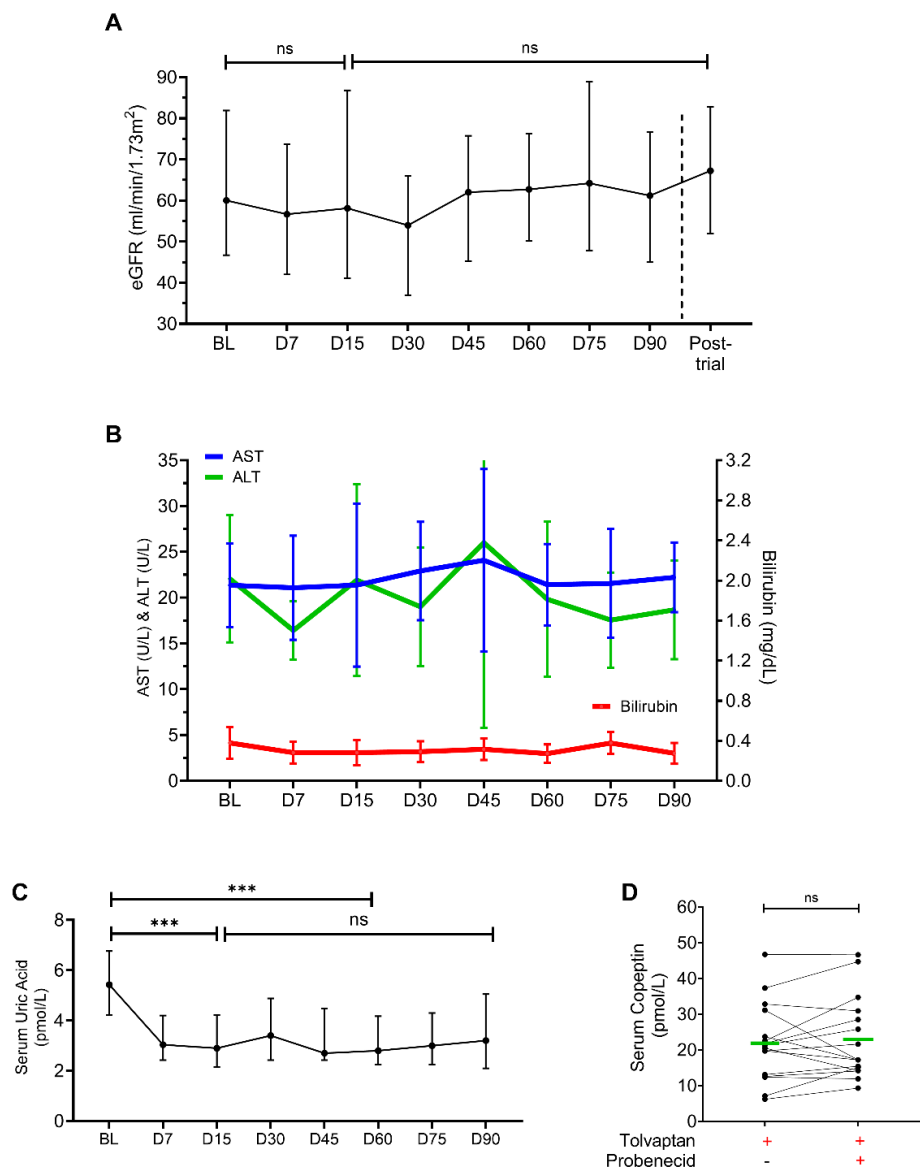


111 **Supplementary Figure 8. Probenecid attenuates tolvaptan-induced aquaresis with improvement in kidney volume**
112 **and renal function. (A)** Change in total kidney volume (Δ TKV), showing improvement with tolvaptan/probenecid
113 combination. **(B)** Blood urea nitrogen (BUN) levels across treatment groups, showing no significant differences. **(C)** Serum
114 creatinine levels, showing reduction with tolvaptan/probenecid combination. Data are presented as mean $\pm$ SD with
115 individual data points representing biological replicates. Statistical significance was determined by one-way ANOVA with
116 Dunnett correction. * $P < 0.05$, ** $P < 0.01$.



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Supplementary Figure 9. Design and biochemical outcomes of the SereNDipity-pb1 clinical trial evaluating probenecid in ADPKD patients on tolvaptan. (A) Mean estimated glomerular filtration rates (eGFR) during the 90-day follow-up. (B) Trends in liver function tests (AST, ALT) and total bilirubin over the study duration. (C) Serum uric acid levels throughout treatment. (D) Paired serum copeptin values during the study period. * $P < 0.05$; ** $P < 0.001$. BL, baseline before initiation of treatment.



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Supplementary Table 1. Clinical characteristics, adverse events, and outcomes from the SereNDipity-pb1 trial. a, Baseline demographic and clinical characteristics of ADPKD patients receiving tolvaptan in the SereNDipity-pb1 trial (N = 17) vs. matched historical control cohort not treated with tolvaptan or probenecid (N = 159), matched by age, sex, and eGFR. **b,** Key clinical outcomes following ≥7 days of probenecid therapy, including changes in 24-hour urine volume, nocturia frequency, morning urine osmolality, serum uric acid, serum copeptin, and ADPKD-Impact Scale (ADPKD-IS) scores. **c,** Safety data for patients with longitudinal follow-up (N = 14), including eGFR, AST, ALT, and bilirubin at baseline, during probenecid treatment, and post-discontinuation. Summary of non-serious adverse events reported during the study period. Two-tailed paired t-tests. P-values < 0.05 were considered statistically significant.

a) Demographics			
	ADPKD on tolvaptan	ADPKD not on tolvaptan	p-value
N	17	159	
Females, n (%)	9 (52.9%)	88 (55.3%)	0.85
Caucasians, n (%)	16 (94.1%)	143 (89.9%)	0.58
Age, years, mean (SD)	46.6 (10.5)	46.2 (12.3)	0.88
BMI, kg/m², mean (SD)	26.7 (4.8)	28.2 (6.6)	0.36
eGFR at baseline, mL/min/1.73 m², mean (SD)	66.6 (22.5)	63.4 (25.9)	0.62
24-hour urine volumes, mL, mean (SD)	6235 (2046)	2343 (1031)	<0.0001
Hypertension, n (%)	15 (88.2%)	149 (97.3%)	0.39
CKD stage at baseline			0.76
Stage 1-2, n (%)	9 (52.9%)	78 (49.1%)	
Stage 3, n (%)	8 (47.1%)	81 (50.9%)	

Tolvaptan dosage		ADPKD on tolvaptan
45/15, n (%)		7 (41.2%)
60/30, n (%)		8 (47.1%)
90/30, n (%)		2 (11.8%)

b) Outcomes				
		Tolvaptan (T)	Tolvaptan and probenecid (TP)	p-value
N		14	14	
24-hour urine volume, mL, mean (SD)		6798 (1693)	4662 (1168)	<0.0001
Urine osmolality, mOsm/Kg, mean (SD)		248 (126)	324 (85)	0.017
ADPKD-IS, mean (SD)		32.4 (11.6)	27.4 (11.4)	<0.0001
Nocturia episode	N	10	10	0.0004
	Mean (SD)	3.9 (1.4)	0.9 (0.6)	
Serum uric acid, mg/L, mean (SD)		5.4 (1.3)	3.2 (1.1)	<0.0001
Serum copeptin, pmol/L, mean (SD)		21.8 (10.9)	22.8 (11.4)	0.55
Serum sodium, mmol/L, mean (SD)		140.7 (2.5)	140.1 (1.6)	0.56

c) Safety Markers					
				p-value	
	Tolvaptan (T)	Tolvaptan and probenecid (TP)	Tolvaptan Post-trial (TpostTP)	T vs. TP	TP vs. TpostTP
N	14	14	14		
eGFR, mL/min/1.73m², mean (SD)	71.9 (21.1)	63.9 (19.8)	70.4 (17.7)	0.003	0.038
AST, U/L, mean (SD)	20.9 (4.1)	21.6 (5.0)	18.9 (2.8)	0.51	0.29
ALT, U/L, mean (SD)	21.4 (4.7)	20.4 (6.5)	17.9 (3.9)	0.51	0.32
Bilirubin, mg/dL, mean (SD)	0.42 (0.19)	0.33 (0.10)	0.52 (0.16)	0.14	0.003

Adverse events*	ADPKD on tolvaptan
N	17
Participants with AEs, n (%)	9 (52.9%)
Serious AEs, n (%)	0 (0%)
AEs leading to withdrawal, n (%)	2 (11.8%)
Gastrointestinal upset, n (%)	6 (35.2%)
<i>Nausea, n (%)</i>	6 (35.2%)
<i>Vomiting, n (%)</i>	1 (5.9%)
<i>Decreased appetite, n (%)</i>	1 (5.9%)
<i>Heartburn, n (%)</i>	1 (5.9%)
Headaches, n (%)	3 (17.6%)
Rash, n (%)	4 (23.5%)
Lower limb edema, n (%)	1 (5.9%)
Hemorrhoids, n (%)	1 (5.9%)
High blood pressure readings, n (%)	1 (5.9%)
Pleuritic chest pain, n (%)	1 (5.9%)

*Adverse events are not mutually exclusive; a single patient may have experienced more than one event.
ADPKD: autosomal dominant polycystic kidney disease; ADPKD-IS: Autosomal Dominant Polycystic Kidney Disease–Impact Scale; ALT: alanine aminotransferase; AST: aspartate aminotransferase; BMI: body mass index; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; SD: standard deviation

Section/topic	No	CONSORT 2025 checklist item description	Reported on page no.
Title and abstract			
Title and structured abstract	1a	Identification as a randomised trial	N/A (non-randomized)
	1b	Structured summary of the trial design, methods, results, and conclusions	P.13, lines. 308-337
Open science			
Trial registration	2	Name of trial registry, identifying number (with URL) and date of registration	P.24, lines 573-575
Protocol and statistical analysis plan	3	Where the trial protocol and statistical analysis plan can be accessed	P.26, lines 617-618
Data sharing	4	Where and how the individual de-identified participant data (including data dictionary), statistical code and any other materials can be accessed	P.24, lines 581-596
Funding and conflicts of interest	5a	Sources of funding and other support (eg, supply of drugs), and role of funders in the design, conduct, analysis and reporting of the trial	P.27, lines 659-663
	5b	Financial and other conflicts of interest of the manuscript authors	P.27, lines 659-663
Introduction			
Background and rationale	6	Scientific background and rationale	P.13, lines. 308-337
Objectives	7	Specific objectives related to benefits and harms	P.24, lines 581-601
Methods			
Patient and public involvement	8	Details of patient or public involvement in the design, conduct and reporting of the trial	N/A
Trial design	9	Description of trial design including type of trial (eg, parallel group, crossover), allocation ratio, and framework (eg, superiority, equivalence, non-inferiority, exploratory)	P.24, lines 572-618
Changes to trial protocol	10	Important changes to the trial after it commenced including any outcomes or analyses that were not prespecified, with reason	N/A
Trial setting	11	Settings (eg, community, hospital) and locations (eg, countries, sites) where the trial was conducted	P.24 lines 573-575
Eligibility criteria	12a	Eligibility criteria for participants	P.24 lines 576-580
	12b	If applicable, eligibility criteria for sites and for individuals delivering the interventions (eg, surgeons, physiotherapists)	N/A
Intervention and comparator	13	Intervention and comparator with sufficient details to allow replication. If relevant, where additional materials describing the intervention and comparator (eg, intervention manual) can be accessed	P.24, line 581-586
Outcomes	14	Prespecified primary and secondary outcomes, including the specific measurement variable (eg, systolic blood pressure), analysis metric (eg, change from baseline, final value, time to event), method of aggregation (eg, median, proportion), and time point for each outcome	P.25 lines 597-615
Harms	15	How harms were defined and assessed (eg, systematically, non-systematically)	P.24 lines 581-596
Sample size	16a	How sample size was determined, including all assumptions supporting the sample size calculation	Pilot and feasibility
	16b	Explanation of any interim analyses and stopping guidelines	P.24, lines 597-601
Randomisation:			
Sequence generation	17a	Who generated the random allocation sequence and the method used	N/A
	17b	Type of randomisation and details of any restriction (eg, stratification, blocking and block size)	N/A

			Reported on page no.
Allocation concealment mechanism	18	Mechanism used to implement the random allocation sequence (eg, central computer/telephone; sequentially numbered, opaque, sealed containers), describing any steps to conceal the sequence until interventions were assigned	N/A
Implementation	19	Whether the personnel who enrolled and those who assigned participants to the interventions had access to the random allocation sequence	N/A
Blinding	20	Who was blinded after assignment to interventions (eg, participants, care providers, outcome assessors, data analysts)	N/A
	20	If blinded, how blinding was achieved and description of the similarity of interventions	N/A
Statistical methods	21	Statistical methods used to compare groups for primary and secondary outcomes, including harms	P.24, lines 602-607
	21	Definition of who is included in each analysis (eg, all randomised participants), and in which group	N/A
	21c	How missing data were handled in the analysis	P.25, lines 613-615
	21	Methods for any additional analyses (eg, subgroup and sensitivity analyses), distinguishing prespecified from post hoc	N/A
Results			
Participant flow, including flow diagram	22	For each group, the numbers of participants who were randomly assigned, received intended intervention, and were analysed for the primary outcome	P.13, lines 309-337
	22	For each group, losses and exclusions after randomisation, together with reasons	N/A
Recruitment	23	Dates defining the periods of recruitment and follow-up for outcomes of benefits and harms	P.24, lines 581-596
	23	If relevant, why the trial ended or was stopped	N/A
Intervention and comparator delivery	24	Intervention and comparator as they were actually administered (eg, where appropriate, who delivered the intervention/comparator, how participants adhered, whether they were delivered as intended (fidelity))	P.24, line 581-586
	24	Concomitant care received during the trial for each group	P.24, lines 597-615
Baseline data	25	A table showing baseline demographic and clinical characteristics for each group	Supp Table 2
Numbers analysed, outcomes and estimation	26	For each primary and secondary outcome, by group: - the number of participants included in the analysis - the number of participants with available data at the outcome time point - result for each group, and the estimated effect size and its precision (such as 95% confidence interval) - for binary outcomes, presentation of both absolute and relative effect size	P.49 Fig 9 + Sup. Fig 6 Sup. Table 2
Harms	27	All harms or unintended events in each group	Supp Table 2
Ancillary analyses	28	Any other analyses performed, including subgroup and sensitivity analyses, distinguishing pre-specified from post hoc	N/A
Discussion			
Interpretation	29	Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence	P.13, lines 308-337
Limitations	30	Trial limitations, addressing sources of potential bias, imprecision, generalisability, and, if relevant, multiplicity of analyses	P.13, lines 308-337

Citation: Hopewell S, Chan AW, Collins GS, Hróbjartsson A, Moher D, Schulz KF, et al. CONSORT 2025 Statement: updated guideline for reporting randomised trials. BMJ. 2025; 388:e081123. <https://dx.doi.org/10.1136/bmj-2024-081123>
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Supplementary Table 3. Primer sequences used in gene expression and mutagenesis studies. **a**, Oligonucleotide sequences used for quantitative real-time PCR (qPCR) of target genes. **b**, Primer pairs designed for site-directed mutagenesis to generate AQP2 cDNAs and related constructs.

Supplementary Table 3a. Sequences for the primers used in real time PCR			
Genes	Species	Primers forward (5' – 3')	Primers reverse (5' – 3')
<i>Aqp2</i>	Mouse	TTGCCATGTCTCCTTCCTTC	GGTCAGGAAGAGCTCCACAG
<i>Avpr2 or V2r</i>	Mouse	TGACCGAGACCCGCTGTTA	CGACCCCGTCGTATTAGGG
<i>Glut9</i>	Mouse	CCACGCTACCTGCTCTTGGA	TTTCCCAAGAACGTTGGA
<i>Abcg2</i>	Mouse	TCGCAGAAGGAGATGTGTTGAG	CCAGAATAGCATTAAAGGCCAG
<i>Panx1</i>	Mouse	AGCCAGAGAGTGGAGTTCAAAGA	CATTAGCAGGACGGATTTCAGAA
<i>Nlrp3</i>	Mouse	TCACAACTCGCCCAAGGAGGAA	AAGAGACCACGGCAGAAGCTAG
<i>Slc14a2</i>	Mouse	CCGCATCTACTTCCTGACAGTG	CGATGTGGAACACACTCCTACG
<i>Prkaa1</i>	Mouse	GGTGTACGGAAGGCAAAATGGC	CAGGATTCTTCCTTCGTACACGC
<i>Prkaa2</i>	Mouse	CTGAAGCCAGAGAATGTGCTGC	GAGATGACCTCAGGTGCTGCAT
<i>Gapdh</i>	Mouse	TGAGCAAGAGAGGCCCTATC	AGGCCCTCCTGTTATTATG
Supplementary Table 3a 3b. Sequences for the primers used in point mutagenesis			
S256A <i>Aqp2</i>	Mouse	GGCGGCAGG <u>GC</u> AGTGGAGCTGCA CTCTCC	CAGCTCCACT <u>GC</u> CTGCCGCCGC CGCACT

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Supplementary Table 4. Commercial antibodies used for immunoblotting experiments. List of primary and secondary antibodies used in Western blotting, including target specificity, host species, vendor, catalog numbers, and working dilutions.

Target Protein	Antibody Source	Catalog Number	Dilution	Blocking Solution (5%)	Incubation time and temperature
AQP2	Santa Cruz Biotechnology	sc-515770	1:200	Skim Milk	16h at 4°C
AQP2	Abcam	ab199975	1:50	BSA	1h at 22°C
Vinculin	Santa Cruz Biotechnology	sc-73614	1:200	Skim Milk	16h at 4°C
GAPDH	Santa Cruz Biotechnology	sc-32233	1:200	Skim Milk	16h at 4°C
AQP2-pS256	Biorbyt	orb155711	1:1000	Skim Milk	16h at 4°C
PANX1	Abcam	ab124131	1:1000	Skim Milk	16h at 4°C
NLRP3	Abcam	ab263899	1:1000	Skim Milk	16h at 4°C
GLUT9	Thermo Fisher Scientific	PA5-22971	1:1000	Skim Milk	16h at 4°C
SLC14A2/UT-A	Bioss	BS-11801R	1:1000	Skim Milk	16h at 4°C
Phospho-AMPKα (Thr172)	Cell Signaling	2531S	1:1000	BSA	16h at 4°C
AMPKα	Proteintech	10929-2-AP	1:2000	Skim Milk	1.5h at 22°C
ABCG2	Proteintech	27286-1-AP	1:1000	1% BSA	1.5h at 22°C
EEA1	Cell signaling	2411	1:100	1% BSA	16h at 4°C
Rab7	Cell signaling	9367S	1:100	1% BSA	16h at 4°C
Rab11	Cell signaling	5589S	1:50	1% BSA	16h at 4°C
CD81	Cell signaling	10037	1:200	1% BSA	16h at 4°C
Goat anti-Rabbit, Alexa Fluor-594	Thermo Fisher	A-11012	2 µg/mL	1% BSA	1h at 22°C
ENVISION+/HRP Rabbit	Agilent	K400311-2	100 µl	1% BSA	1h at 22°C
Goat anti-Rabbit (HRP)	Thermo Fisher Scientific	31460	1:5000	Skim Milk	1h at 22°C
Goat anti-Mouse (HRP)	Thermo Fisher Scientific	31430	1:5000	Skim Milk	1h at 22°C

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