SUPPLEMENTAL MATERIAL

Data S1.

SUPPLEMENTAL METHODS

The detailed description of the reagents and resources can be found in the Major Resources Tables.

Animals and experimental PH model

Care of the animals and surgical procedures were performed according to the Directive 2010/63/EU of the European Parliament. Animals were housed in an environmentally-controlled pathogen-free animal facility for the duration of the experiment. All animals had *ad libitum* access to food and water and were under a 12h-12h light-dark cycle. $Pw1^{IRESnLacZ}$ transgenic mice ($Pw1^{nlacZ/+}$), in which a nuclear operon lactose gene is expressed under the control of the Pw1 gene locus were bred in our animal facility and maintained in a C57BL6/J background [3,18]. C57BL6/J mice were from Janvier (Orléans, France). RosaCRE-ERT2 (B6.129-Gt(ROSA)$26^{Sortm1(}$cre/ERT2)Tyj/J) and $PDGFR\alpha^{+/(S)K}$ (B6.129S4-Pdgfratm12Sor/J) transgenic mice were bought from Jackson Labs and are in a C57BL6/J background. The $PDGFR\alpha^{+/(S)K}$ strain presents an inducible heterozygous mutation (D842V) located in the kinase domain leading to constitutive PDGFRα activation after Cre-dependent recombination [19]. The strains were crossed together to produce $Pw1^{nlacZ/+}$xRosaCre-ERT2x$PDGFR\alpha^{+/(S)K}$ mice. Mice (littermates of 6-10 weeks) were exposed to room air (normoxia) or chronic normobaric hypoxia (10% O_2) in a ventilated chamber for 4 or 21 days with the same light-dark cycle. Hypoxia was obtained by flushing nitrogen into the hypoxic chamber. Levels of CO_2 and O_2 in the chamber were constantly monitored and maintained by flushing oxygen in the chamber and by absorbing CO_2 with soda lime granules. The chamber temperature was maintained at 22–24°C and excess humidity was prevented by desiccant. To confirm chronic hypoxia (CH) after 21 days, hemoglobin concentration was measured in the peripheral blood using HemoCue (Hb 20+). Male and female mice show similar vascular remodeling to CH, so both females and males were used for studies after four days of CH. Females are known to

display statistically lower RVSP than males, so we only studied males for long-term CH or after PDGF-AA treatment to obtain homogeneous groups. Cre recombination was induced by Tamoxifen injection (2 mg/day/mouse for five consecutive days). Tamoxifen (Sigma Aldrich, France) was prepared in 100% ethanol and diluted in corn oil for i.p. injection. Mice (males and females) were sacrificed two or five weeks after the last injection. Mice, in particular littermates, were randomly assigned to the different experimental groups.

Hemodynamic measurements and tissue collection

Hemodynamic parameters were measured blinded to the experimental treatment as already described [3]. Mice were anesthetized with a ketamine/Xylazine mix (100 and 10 mg/kg i.p.) and maintained at 37°C. After intubation, mice were connected to a Minivent Mouse ventilator (type 845, Harvard Apparatus, respiratory frequency 170 stroke/min and respiratory volume 200 µL). After incision of the abdomen and diaphragm, mouse right ventricular systolic pressure (RVSP) was measured by introducing a Millar pressure transducer (size 1.4 F, Millar Micro-tip catheter transducer, model SPR-671; Millar Instruments, Inc, Houston, TX, USA) into the right ventricle. After recording, the mice were killed by removal of the heart. Right ventricle (RV) was separated from the left ventricle+septum (LV+S) and both were weighed to calculate the Fulton index (RV/LV+S) measuring right ventricular hypertrophy.

Mice treatment

Pw1$^{nlacZ/+}$mice received a daily i.p. injection of imatinib (50 mg/kg/d, #SML1027, Sigma Aldrich, St. Quentin Fallavier, France, dissolved in DMSO and diluted in PBS prior to injection) or vehicle (2% DMSO in PBS) during the whole duration of CH. Specific PDGFRα blocking antibody was expressed and purified from hybridoma cell line APA5 [45,46] as previously described [47,48]. For the blocking antibody experiments, mice were injected i.p. with 500 µg of PDGFRα blocking antibody APA5 or isotype control (clone 2A3, InVivoMAb BE0089, BioXCell, USA) three days before starting CH, with additional 100 µg i.p. injections every three days during the course of CH. For proliferation measurements, mice were injected i.p. with

ethynyl-deoxyuridine (EdU, 1 mg/mouse in PBS) 24 h prior to sacrifice. For PDGF-AA treatment, mouse PDGF-AA (PeproTech, Neuilly-Sur-Seine, France) or vehicle (PBS with 0.03% mouse serum albumin, Sigma Aldrich) was injected i.p. 6 days/week (150 ng/mouse) for five weeks.

Flow cytometry isolation of PW1$^+$ progenitor cells

Lung single-cell suspensions were obtained as previously described with minor changes. Tissue was digested in HBSS (Gibco, Thermo Fisher Scientific, Villebon, France) supplemented with 3% decomplemented fetal bovine serum (FBS; Thermo Fisher Scientific) and 0.5 mg/mL of collagenases IA-S, II-S, IV-S (Sigma Aldrich) for 35 min at 37°C. The remaining tissue fragments were next disrupted mechanically between two superfrost+ slides (Thermo Fisher Scientific), to separate the progenitor cells from the extracellular matrix. Mouse lung cells were stained for 20 min on ice in the dark. Antibodies used were CD45 PECy7, CD34 Brilliant Violet421, and c-kit PE [3]. DDAO-G (Life Technologies) was used to detect β-gal activity. Dead cells were labeled using Zombie Green Fixable Viability (Biolegend, San Diego). Cells were sorted using a FACSAria (Becton Dickinson) with appropriate isotype matching controls. Fluorescence Minus One controls (FMO) were used to identify and gate cells. The FACS gating strategy is described in Figure S7. We have previously shown that mouse pulmonary CD45$^-$/CD34$^+$/β-gal$^+$/c-kit$^-$ cells are vascular progenitor cells expressing PDGFRα$^+$ and PW1$^+$ [3].

Cell culture

FACS-isolated pulmonary CD45$^-$/CD34$^+$/β-gal$^+$/c-kit$^-$ progenitor cells were used to test the effect of PDGFRα activation or inhibition on their spontaneous differentiation into smooth muscle cells in *in vitro* culture. Following FACS, cells were plated at a density of 2000 cells/cm^2 and were maintained in DMEM medium (Thermo Fisher Scientific) supplemented with 20% FBS and 1% penicillin/streptomycin (Thermo Fisher Scientific) for 24 hours. For PDGFRα activation experiments, cells were incubated in DMEM medium supplemented with 1% FBS and 1% PS and were stimulated or not with 20 ng/ml of PDGF ligands (AA; AB and BB, R&D Systems). For PDGFRα inhibition experiments, cells were incubated with DMEM containing 5% FBS, 1% PS, and APA5 blocking antibody or control antibody (150 µg/mL).

In all cases, medium was changed every other day and culture was stopped at day five for immunofluorescence analysis.

Immunofluorescence analysis

Mouse lungs were inflated by intratracheal injection of 1-2 ml Optimal Cutting Temperature (OCT) medium (Leica, 50% in PBS) and fixed by immersion in a 4% paraformaldehyde solution (PFA; Sigma) for 24-48 hours before paraffin embedding. Alternatively, lungs were immersed in isopentane, frozen in liquid nitrogen, embedded in OCT and stored at -80°C. Sections (3 µm) of paraffin-embedded tissue were deparaffinized, rehydrated, and citrate antigen retrieval (20 min; 95°C) was performed. Cryosections (12 µm) were fixed in 4% paraformaldehyde. After permeabilization with ice-cold acetone/methanol (1:1) (10min; -20°C), sections were blocked with 5% FBS (1h) prior to incubation with FBS+antibodies (Table S1). Immunolabeled sections were mounted with Dako fluorescent mounting medium (Dako) and examined under fluorescence (Nikon epifluorescence Ti microscope). Images were analyzed using NIH ImageJ. Control experiments were performed using secondary antibodies alone and showed no non-specific labeling.

Pulmonary vessel muscularization was analyzed by co-labeling with anti-α-SMA and anti-vWF antibodies. The percentages of fully-muscularized (SMC covering more than 90% of the vessel) and partially-muscularized (SMC covering more than 10% and less than 90% of the vessel) were determined on more than 100 vWF^+ vessels/animal. The number of $PW1^+$ progenitor-derived SMC was determined as the percentage of $\beta\text{-gal}^+/\alpha\text{-SMA}^+$ SMC relative to the number of $\alpha\text{-SMA}^+$ cells for each vessel (15 to 100 µm diameter) in the whole lung section (15-30 vessels/animal). Perivascular $\beta\text{-gal}^+/PDGFR\alpha^+$ cells were defined to be within 40µm from the vWF^+ vessels. Images were analyzed by an investigator blinded to the experimental status of all animals. Proliferating cells were detected using EdU (Thermo Fisher Scientific) incorporation (Click-iT EdU Alexa Fluor 555 Imaging Kits; Thermo Fisher Scientific) followed by PW1 immunodetection. The number of $EdU^+/PW1^+$ proliferative progenitor cells was measured relative to the total number of $PW1^+$ cells. Apoptotic cells were detected with the TUNEL

method according to the manufacturer's protocol (Fluorescein In Situ Cell Death Kit; Roche). The number of TUNEL$^+$ apoptotic cells was assessed as a percentage of the total number of cells analyzed (2000 to 8000 cells/animal).

For human patients, 5 µm lung sections were deparaffinized and incubated with the antigen retrieval buffer. Then, sections were saturated with blocking buffer and incubated overnight with specific antibodies, followed by addition of the corresponding secondary fluorescent-labeled antibodies (Thermo Fisher Scientific). Nuclei were labeled using DAPI (Thermo Fisher Scientific). Mounting was performed using ProLong Gold antifade reagent (Thermo Fisher Scientific). All images were taken using an LSM700 confocal microscope (Zeiss, Marly-le-Roi, France).

Cultured cells were fixed with 4% PFA (6min) and permeabilized using acetone/methanol (1:1) (10 min; -20°C) prior to incubation with antibodies (Table S1). The number of proliferative cells (positive for Ki67$^+$) or differentiated (positive for α-SMA) was determined relative to the total number of cells in the well.

Table S1. Antibodies used for immunofluorescence experiments. ON, overnight. RT, room temperature.

Target antigen	Provider, reference	Dilution, conditions	Secondary antibody	Provider, reference	Dilution, conditions
PW1 (human)	Novus Biologicals NBP2-46379	1/100 ON; 4°C	Alexa Fluor Donkey anti-mouse 647	Thermo Fisher Scientific A31571	1/200 1h ; RT
PDGFRα (human)	abcam ab61219	1/200 ON; 4°C	Alexa Fluor Donkey anti-rabbit 547	Thermo Fisher Scientific A10040	1/200 1h ; RT

PW1 (mouse)	Relaix 1996 ref [49]	1/5000 ON; 4°C	Goat anti-rabbit 488	Thermo Fisher Scientific A11034	1/500 1h; RT
β-galactosidase	Abcam ab9361	1/6000 ON; 4°C	Goat anti-chicken 594	Thermo Fisher Scientific A11042	1/500 1h; RT
α-SMA-FITC	Sigma F3777	1/500 1h30; RT			
α-SMA	ebioscience 14-9760-82	1/500 ON; 4°C	Goat anti-mouse 555	Abcam Ab150078	1/500 1h; RT
Von Willebrand Factor	DAKO A0082	1/300 ON; 4°C	Goat anti-rabbit 488	Thermo Fisher Scientific A11034	1/500 1h; RT
Ki67	Abcam ab15580	1/300 ON; 4°C	Goat anti rabbit-488	Thermo Fisher Scientific A11034	1/500 1h; RT
DAPI	Santa Cruz sc3598	1/1000 1h; RT			

RT-qPCR

Total lung RNA was extracted using Trizol reagent (Thermo Fisher Scientific) according to the manufacturer's instructions. Total RNA (0.5 µg) was reverse transcribed (M-MuLV kit, Thermo Fisher Scientific) per manufacturer's instructions and real time PCR was performed and analyzed using a Light Cycler (LightCycler 480, Roche) with the Brilliant III kit (**Agilent Technologies, Les Ulis, France**). Gene expression was determined relative to RPL13a using the $2^{-\Delta\Delta CT}$ method. The primers sequences are:

Table S2. Primer sequences for real-time PCR measurements of mRNA expression.

Gene	Forward sequence (5'-3')	Reverse sequence (5'-3')
PDGF-A	CATCCGCTCCTTTGATGATCTT	GTGCTCGGGTCATGTTCAAGT

PDGF-B	CATCCGCTCCTTTGATGATCTT	GTGCTCGGGTCATGTTCAAGT
PDGF-C	GCCAAAGAACGGGGACTCG	AGTGACAACTCTCTCATGCCG
PDGF-D	ATGGAACGGCTCGTTTTAGTC	CTTCCAGTTGACAGTTCCGCA
PDGFRα	GGACTTACCCTGGAGAAGTGAGAA	ACACCAGTTTGATGGATGGGA
PDGFRβ	GTGGTGAACTTCCAATGGACG	GTCTGTCACTGGCTCCACCAG
Col1a1	CTCAGGGTGCTCGTGGAT	CTTAGGACCAGCAGGACCAG
Col3a1	GATCTCCTGGTTCTCCTGGAT	TCGTCCAGGTCTTCCTGACT
TGFβ	CTGAACCAAGGAGACGGAAT	GGCTGATCCCGTTGATTTC
Rpl13a	GAGGAGGCGAAACAAGTCCA	GGGTGGCCAGCTTAAGTTCT

Picrosirius red staining

Lung sections (3µm) of paraffin-embedded tissue were deparaffinized, rehydrated and collagen fibers were stained using a sirius red (Direct red 80, CI35780; Sigma) and picric acid solution (1:1) (45 min). Slides were immersed in two picric acid baths (2 min), two absolute ethanol baths (3 min), and two xylene baths (3 min). For each animal, the light microscopy image (Nikon epifluorescence Ti microscope) of the whole pulmonary parenchyma was analyzed to measure the degree of pulmonary fibrosis (percentage of collagen-stained area per total area) with Histolab software (Excilone, Elancourt, France).

Image processing and quantification

All images were composed, edited, and modifications applied to the whole image. Immunofluorescence images were analyzed using ZEN LSM (Carl Zeiss) and ImageJ (NIH) software for human samples and using NIS-Elements (Nikon) and ImageJ (NIH) for mouse samples. Images of picrosirius red staining were quantified using Histolab software (Excilone).

Immunoblotting

Lung samples were frozen in liquid nitrogen and conserved at -80°C. Tissue samples were homogenized with the IKA T10 Ultra Turrax® for 30 seconds on ice, in 20 volumes of RIPA buffer (50 mM Tris HCl pH 7.4, 150 mM NaCl, 1% triton X-100, 0.5% sodium deoxycholate, 0.5% SDS , 1 mM EDTA, and 0.1% anti-protease Sigma P8340 added extra-temporally) per mg of tissue. Tissue lysis was performed by sonicating twice on ice for 10 seconds with the B15 Sonifier Cell disruptor (Proscience) then extracts

were slowly agitated for 1 h at +4°C, aliquoted, and stocked for further use at -80°C. Protein concentration was quantified using the Bradford method. For Western blotting, protein extracts were denatured in Laemmli buffer (BioRad ref 1610747) with 10% β-mercaptoethanol at 65°C for 20 minutes. Forty µg of protein/sample were run on Criterion Tris-glycine 4% to 15% (BioRad ref 6571095) at 80v. Gel transfer was performed using the Biorad TransBlot Turbo system on 0.45 µm nitrocellulose membrane which was blocked in Tris-buffered Saline, 0.1% Tween, 5% non-fat milk for two hours, then hybridized overnight at 4°C with primary antibodies (PDGFRα from R&D ref AF-1062, PDGFRβ clone APB5 from, GAPDH 14C10 from Cell Signaling) followed by HRP-linked secondary antibodies (anti-goat Jackson Lab ref 305-035-003, anti-rat ref NA935 from Cytiva, anti-rabbit from Abcam ref AB6721). Western blot imaging was performed using Western Clarity ECL (ref 170-0560; BioRad) and a LAS 4000 camera (GE-Healthcare). Quantification was performed using ImageJ (NIH) analysis.

Statistical analysis

All measures were performed by an experimenter blinded to animal status or cell treatment. The statistical analysis of the data was performed using XLStat 2013 (Addinsoft, New York, USA) or GraphPad Prism 6 (GraphPad software). Kruskall-Wallis followed by Dunn post-hoc tests or, when appropriate, 2-way ANOVA followed by Tuckey post-hoc tests were used for multiple comparisons and Mann-Whitney or Wilcoxon signed-rank tests for single comparisons. Sample size is indicated in the figures and in the figure legends and was calculated based on our previous experience. P values less than 0.05 were considered to be statistically significant. Graphs display all experimental values with bars representing the means and whiskers representing SD.

Study approval

Animal experiments for the project were approved by our institutional review board (APAFIS#3566bis-2016022617318129 v8).

Studies with patients complied with the Declaration of Helsinki and are part of the French Network on Pulmonary Hypertension, a program approved by our institutional Ethics Committee (2018-A01252-53). Written informed consent was received from participants prior to inclusion in the study.

Table S3. Patients' characteristics.

	Control patients					iPAH patients				
Case	Control 1	Control 2	Control 3	Control 4	Control 5	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age (y)	45	65	40	60	62	21	16	14	51	40
Sex	M	F	F	M	F	M	M	F	F	F
Pathology	Invasive adeno-carcinoma	Invasive adeno-carcinoma	Invasive adeno-carcinoma	Carcinoïd	Invasive adeno-carcinoma	iPAH	iPAH	iPAH	iPAH	iPAH
mPAP (mmHg)	x	x	x	x	x	86	110	75	64	49
PVR (Wood units)	x	x	x	x	x	17	12,89	10	9	7,5
capP (mmHg)	x	x	x	x	x	10	8	12	4	10
CI ($L.min^{-1}.m^{-2}$)	x	x	x	x	x	2.05	3.9	2.6	2.6	3,2
6MWD (m)	x	x	x	x	x	360	535	445	493	347
NYHA	x	x	x	x	x	IV	II	II	III	III

Five control patients and five iPAH patients were included. mPAP, mean pulmonary arterial pressure. PVR, pulmonary vascular resistance. CapP, capillary pressure. CI, cardiac index. 6MWD, 6 minutes walking distance test. NYHA, New York Heart Association Functional Classification.

Figure S1

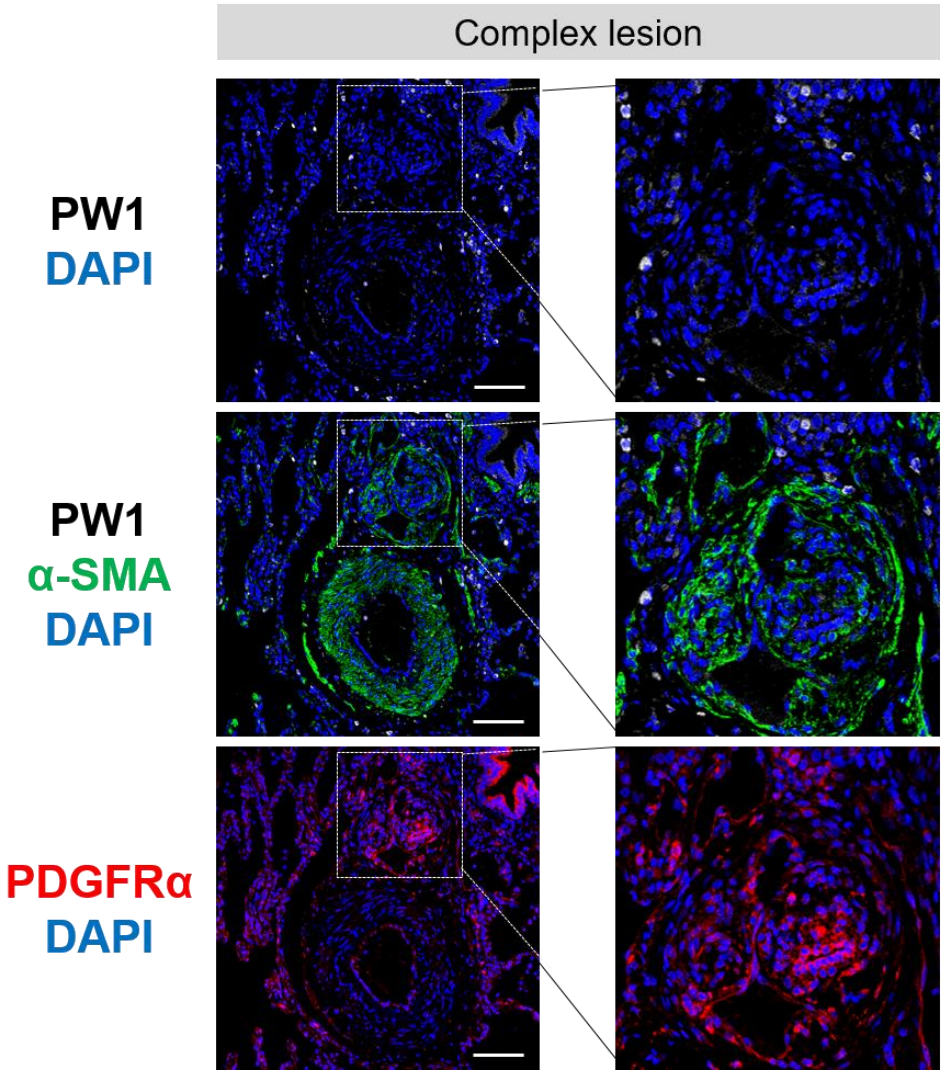

Figure S1. PW1^{+}/PDGFRα^{+} cells are not present within complex vascular lesions in iPAH patients. Lung sections from or iPAH patients (iPAH) were labelled for PW1 (white), PDGFRα (red) and α-SMA (green). Representative confocal images of a complex vascular lesion in iPAH patient lung. Scale bar = 100µm.

Figure S2

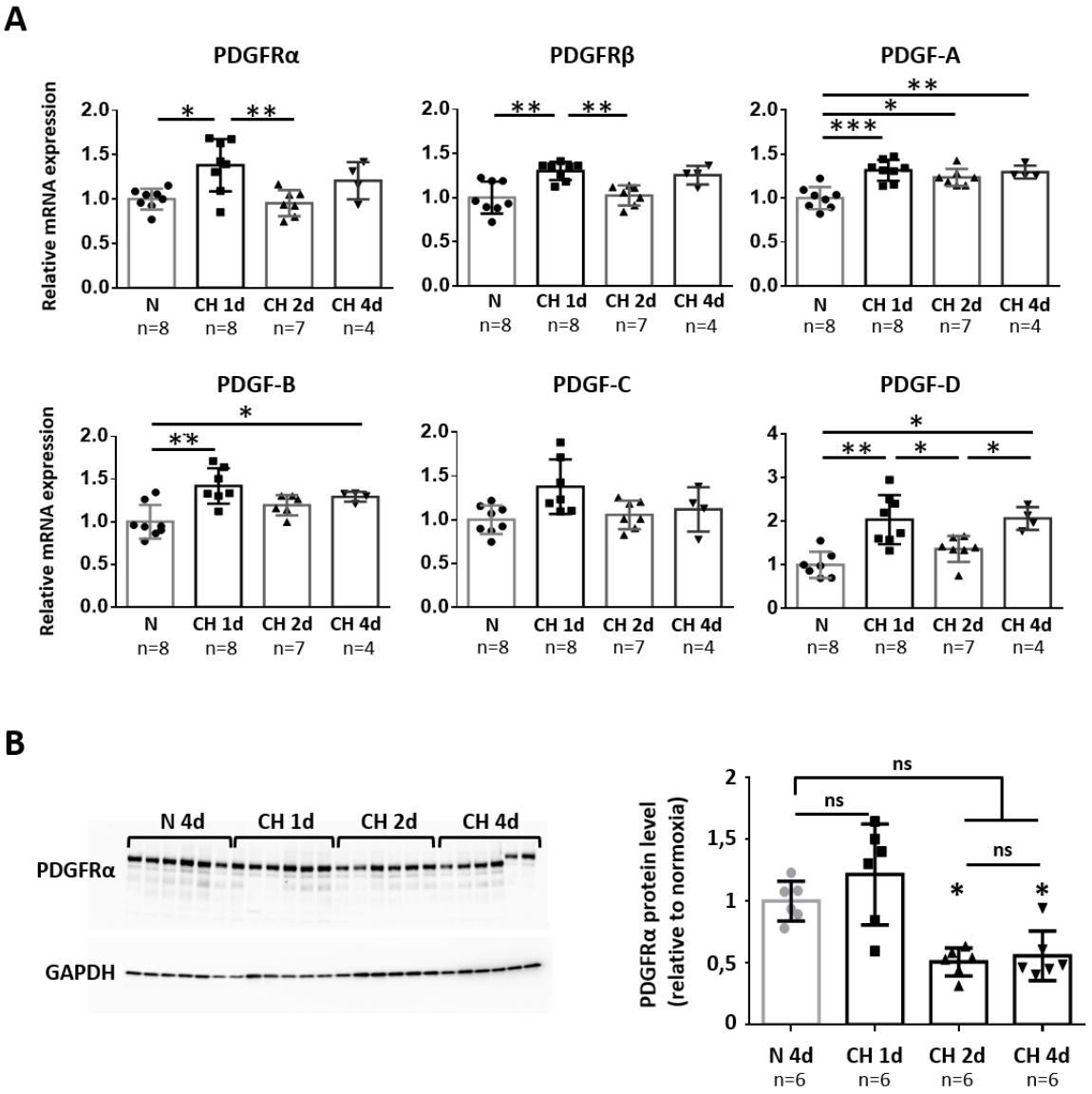

Figure S2. Expression of PDGF ligands and receptors is increased within the first day of chronic hypoxia. A, mRNAs for PDGFRα and β and PDGF-A, -B,- C, and -D were measured by real-time PCR on lung total RNA preparations from mice under normoxia (N) or after 1, 2, or 4 days of CH (CH 1d, CH 2d, and CH 4d respectively) (n=4-8). B, Western blot analysis of PDGFRα in lung extracts from control mice (N 4d) and from animals exposed to 1, 2, or 4 days of chronic hypoxia (CH 1-2-4d) (n=6/group). Quantification of PDGFRα normalized to GAPDH is shown in the bar graphs. Bars represent means and whiskers represent SD. *p<0.05, **p<0.01, ns=not significant (Kruskal-Wallis and Dunn).

Figure S3

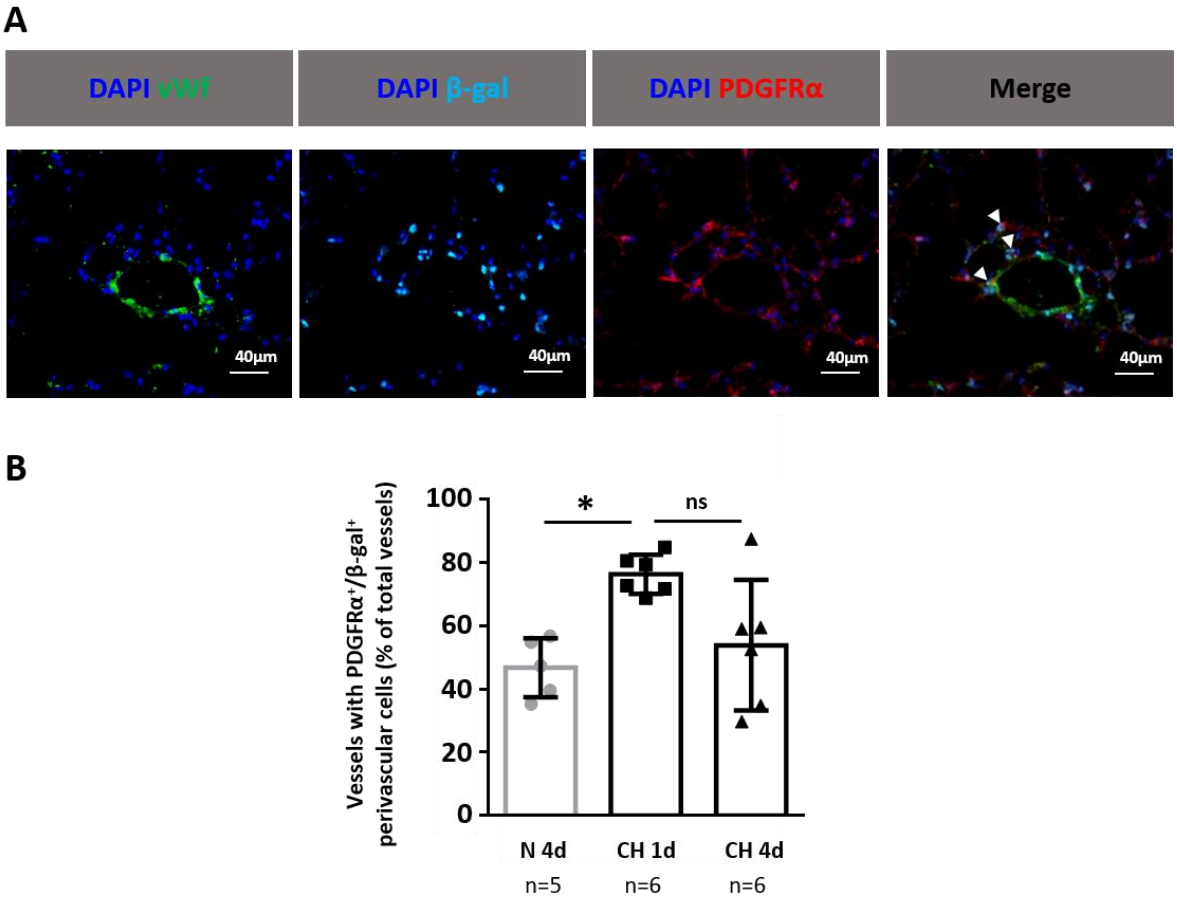

Figure S3. Pulmonary β-gal+/PDGFRα+ perivascular cells are increased within the first day of chronic hypoxia. PW1^{nLacz} mice were maintained under normoxia (N 4d) or chronic hypoxia for 1 or 4 days (CH 1d or 4d). **A**, Representative immunofluorescence image of pulmonary vessels labeled for von Willebrand factor (vWF, green), β-gal (cyan), PDGFRα (red), and DAPI (nuclei, dark blue). Perivascular β-gal$^+$/PDGFRα$^+$ cells are marked by arrowheads. **B,** Quantification of vessels surrounded by one or more β-gal$^+$/PDGFRα$^+$ perivascular cells under normoxia (N 4d) and after 1 or 4 days of chronic hypoxia (CH 1d or 4d) (n=5-6 mice/group). Bars represent means and whiskers represent SD. *p<0.05 vs N 4d, ns=not significant (Kruskal-Wallis and Dunn).

Figure S4

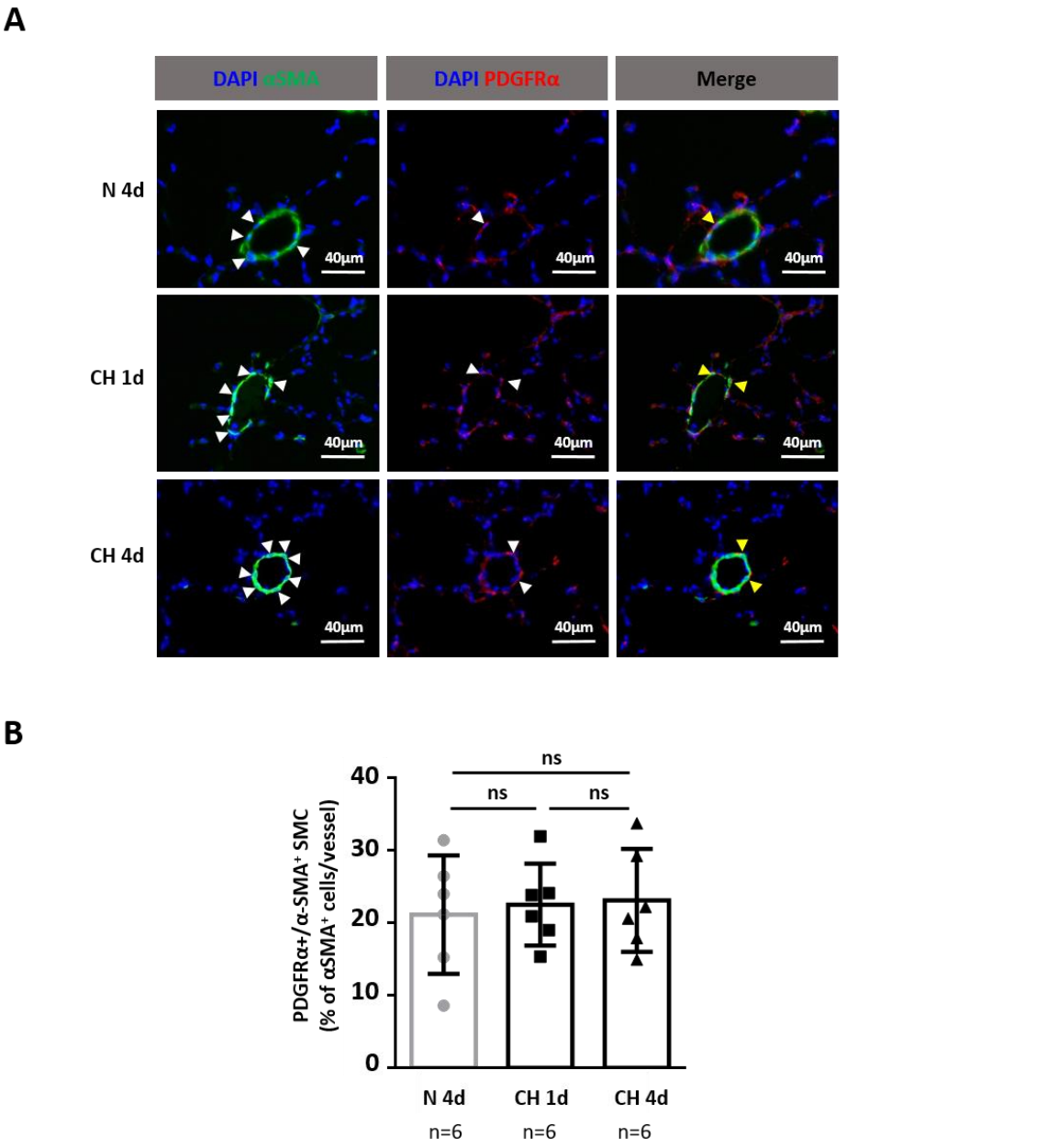

Figure S4. Chronic hypoxia does not increase the number of PDGFRα$^+$/α-SMA$^+$ SMC in pulmonary muscularized vessels. PW1^{nLacz} mice were maintained under normoxia (N 4d) or chronic hypoxia for 1 or 4 days (CH 1d or 4d). **A,** Representative image of pulmonary muscularized vessel labeled for αSMA (green) and PDGFRα (red) (double-positives are marked by yellow arrowheads). **B,** Quantification of lung PDGFRα$^+$/α-SMA$^+$ SMC in muscularized vessels (n=6 mice/group). Bars represent means and whiskers represent SD. ns=not significant (Kruskal-Wallis and Dunn).

Figure S5

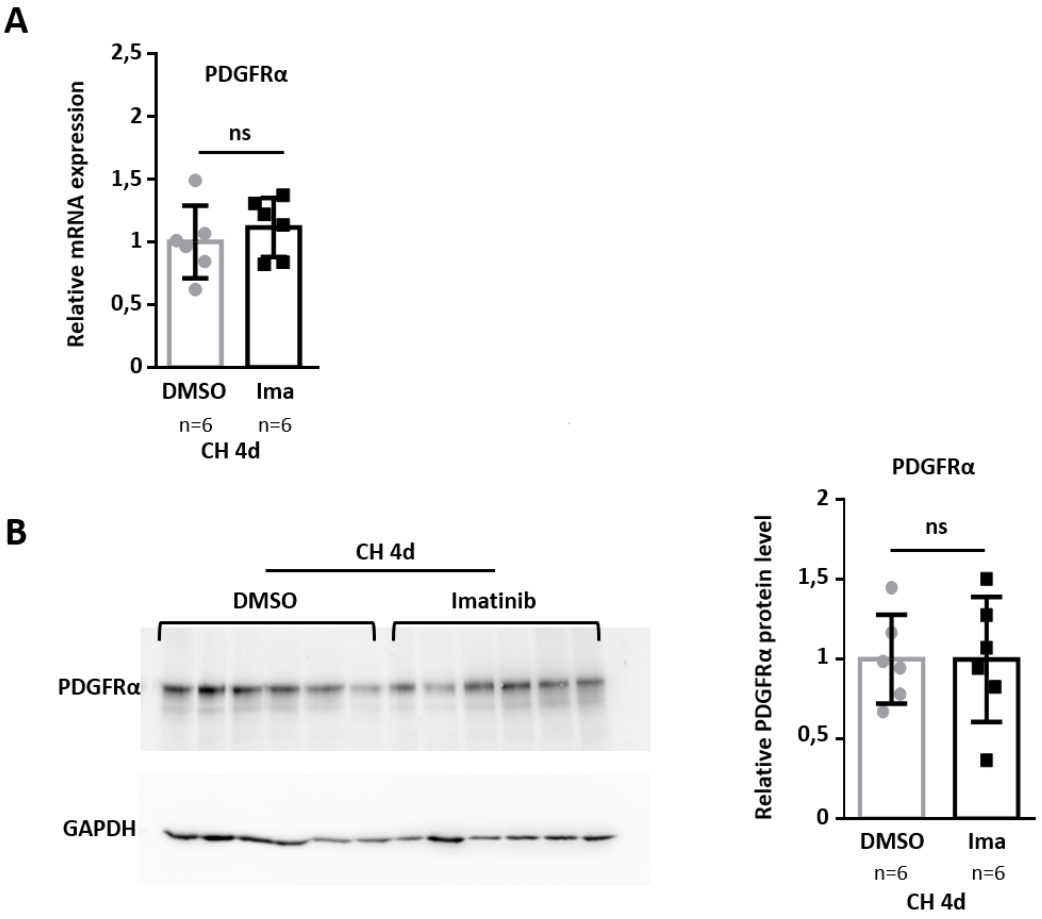

Figure S5. Imatinib treatment does not modify PDGFRα mRNA and protein levels during chronic hypoxia. Mice were maintained under chronic hypoxia for 4 days (CH 4d) and treated daily with DMSO or imatinib (Ima) **A,** PDGFRα mRNA was measured by real-time PCR on lung total RNA preparations from mice exposed to four days of chronic hypoxia (CH 4d) and treated with DMSO (vehicle) or imatinib (n=6 mice/group). **B,** Western blot analysis of PDGFRα in lungs from vehicle-treated mice (DMSO) or Imatinib-treated (Ima) (n=6 mice/group). Quantification of PDGFRα normalized to GAPDH is shown in the bar graphs. Bars represent means and whiskers represent SD. ns=not significant (Kruskal-Wallis and Dunn).

Figure S6

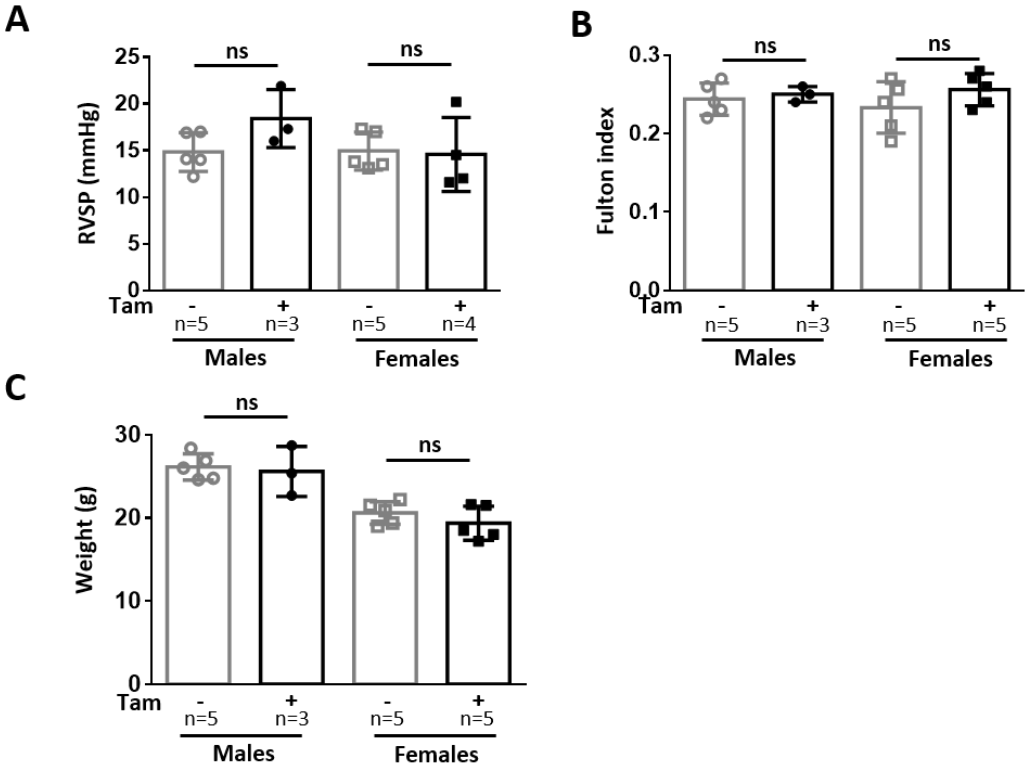

Figure S6: Right systolic ventricular pressure (RSVP, A), Fulton index (B), and total heart weight (C), were not modified after two weeks of constitutive PDGFRα activation. Constitutive PDGFRα activation was induced by Tamoxifen administration in PW1nLacz^{+}/PDGFRα$^{+/(S)K}$/Rosa-CRE^{+} mice. Induced (Tam +) and non-induced (Tam -) mice were analyzed two weeks after the final Tamoxifen administration, n=3-5/group. Bars represent means and whiskers represent SD. ns, not significant, 2-tailed Mann-Whitney.

Figure S7

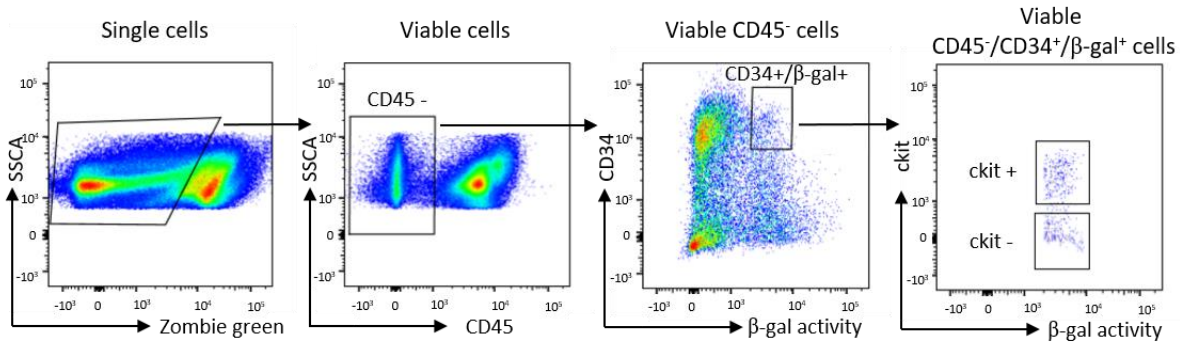

Figure S7: Mouse pulmonary PW1^{+} progenitor cells sorting strategy. Representative FACS profile of total mouse lung cells indicating the gating strategy to sort the PDGFRα^{+}/PW1^{+} populations based on Zombie green (viability marker), DDAO-G (β-galactosidase activity), and CD45, CD34, and c-kit cell surface expression. Viable CD45^{-}/CD34^{+}/c-kit^{+} and c-kit^{-} cells are PDGFRα^{+} [3] and were sorted for cell culture.

Figure S8

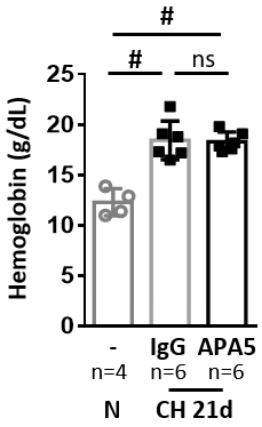

Figure S8. Chronic hypoxia for 21 days increases blood hemoglobin concentration. Blood hemoglobin concentration (g/dL) was measured in untreated normoxic (N, n=4) or in IgG- and APA5-treated PW1^{nLacz} mice after 21 days of CH (CH 21d, n=6) . Bars represent means and whiskers represent SD. # p<0.05 vs normoxic, ns = not significant, Kruskal-Wallis and Dunn.

Figure S9

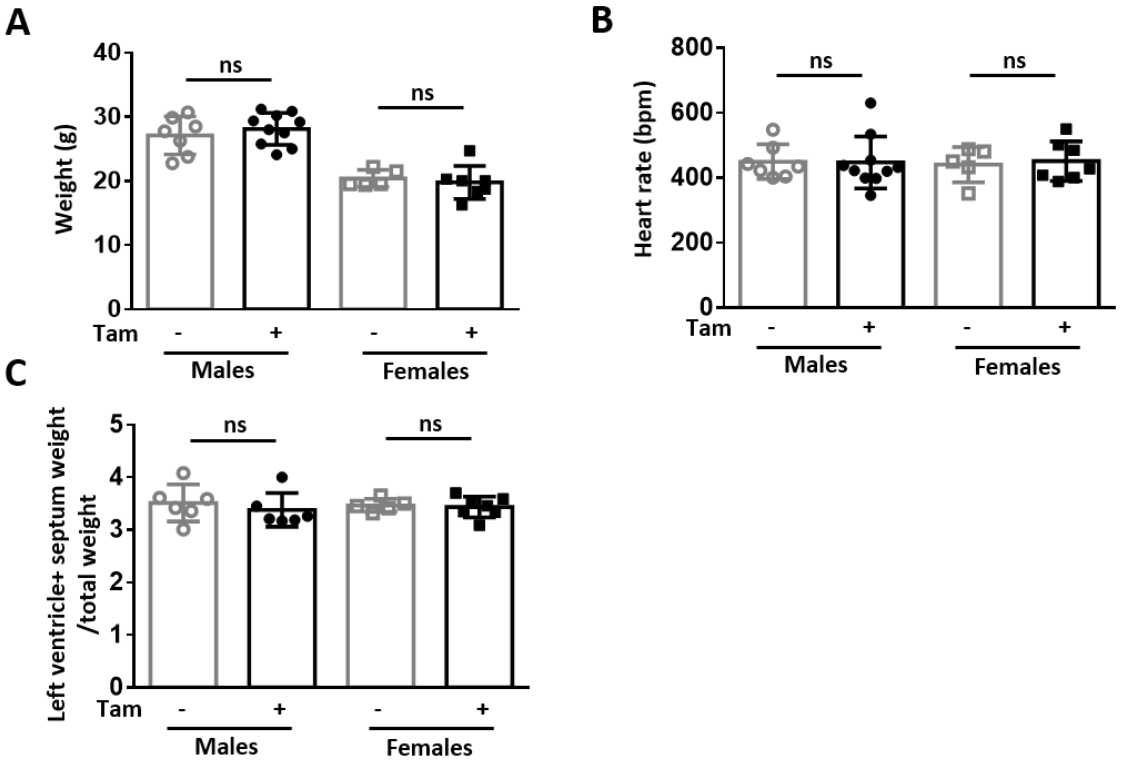

Figure S9: Total heart weight (A), heart rate (B), and left ventricle+septum weight (C) were not modified five weeks after constitutive PDGFRα activation. Constitutive PDGFRα activation was induced by Tamoxifen administration in PW1$^{nLacz/+}$/PDGFRα$^{+/(S)K}$/Rosa-CRE^{+} mice. Induced (Tam +) and non-induced (Tam -) mice were analyzed 5-6 weeks after the final Tamoxifen administration, n=6-7/group. Bars represent means and whiskers represent SD. ns, not significant, 2-tailed Mann-Whitney.

Figure S10

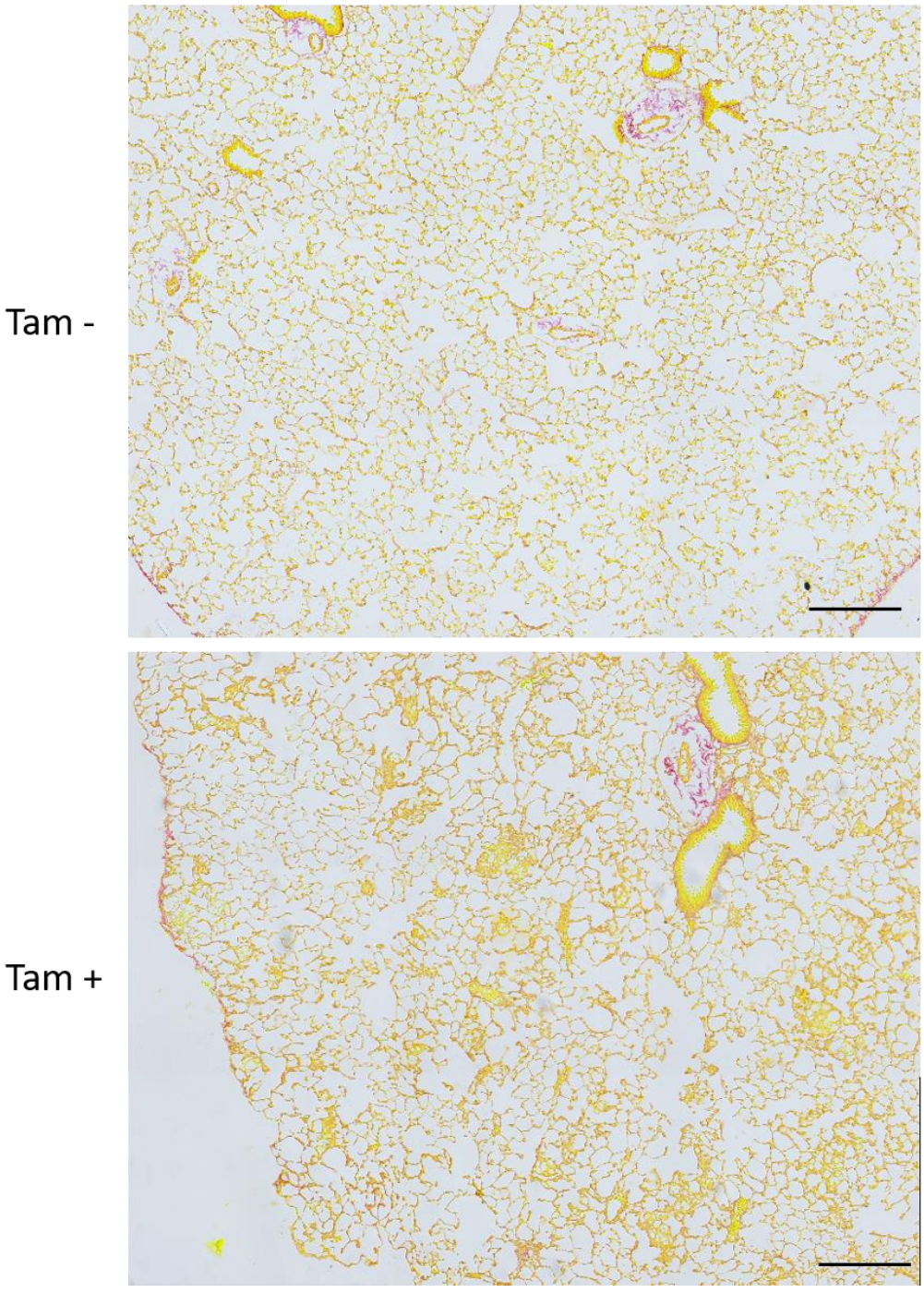

Figure S10. Lung fibrosis is not induced by PDGFRα. Representative images of picrosirius red staining for collagen (red) in pulmonary parenchyma of non-induced (Tam -) or tamoxifen-induced (Tam +) $PW1^{nLacz/+}$/$PDGFR\alpha^{+/(S)K}$/Rosa-CRE^{+} mice five weeks after Tamoxifen induction. Scale bar 200 µm.

Figure S11

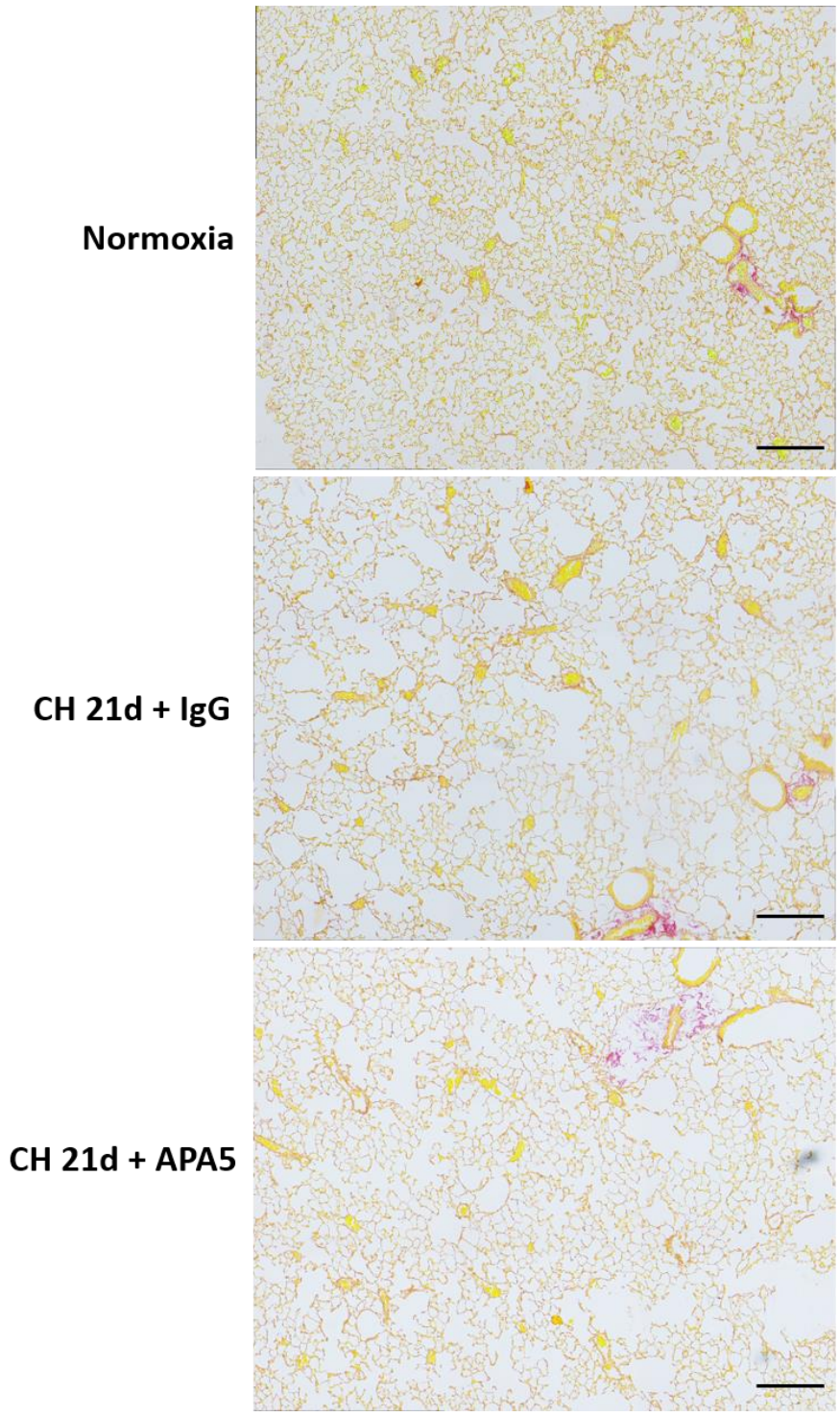

Figure S11. Lung fibrosis is not reduced by PDGFRα inhibition during CH. Representative images of picrosirius red staining for collagen (red) in pulmonary parenchyma of untreated mice under normoxia or IgG- and APA5-treated mice after 21 days of CH. Scale bar 200 µm.