Table, Supplemental Digital Content 2. Summary of included animal studies.

Credits: The authors.

References	Study population (n, species, sex, age, groups)	PIV measurement details (pressure stimulus: static/progressive, ramp, duration, temperature, location of measurement)	Results (max PIV, decrease of blood flow in other group at point of maximum PIV of controls, physiological results, miscellaneous	Conclusions of the authors
Begey 2018[30]	n: between 4-9 for each group Sex: Male Sp: C57Bl/6 mice Age: 10–14 weeks G: Healthy controls vs high-salt diet	PS: progressive, 2.2 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +23 SEM 6% at 0.7 kPa BF at 0.7 kPa after 10-12 day high-salt diet: -4 SEM 4%, p<0.01	No conclusions regarding cutaneous PIV
Danigo 2015[31]	n: 20 for each group Sex: Male Sp: Swiss mice Age: 5-6 weeks G: STZ-induced diabetes of 8 weeks with/without 2 week Candesartan treatment; healthy controls with/without 2 week Candesartan treatment	PS: progressive, 2.2 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +35 SEM 8% at 0.4 kPa BF at 0.4 kPa after 8 weeks of STZ-induced-DM: -17 SEM 7%, p < 0.001 Max PIV, I: +27 SEM 11%, p < 0.05 Candesartan increased resilience against pressure ulcer formation induction testing	Candesartan decreases vulnerability to pressure-induced ulcerations and restores PIV in mice with STZ-induced diabetes
Demiot 2006[32]	n: 7-10 for every group Sex: Male Sp: Swiss mice Age: 12 weeks G: STZ-induced diabetes of 1 week; STZ-induced diabetes of 1 week with daily Sorbinil; Alagebrium; or α-LPA treatment; healthy control mice (5 groups)	PS: progressive, 2.2 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +42 SEM 8% at unknown pressure (NR) After 1 week of STZ-induced DM: No PIV but a decrease, p < 0.001, values NR Sorbinil: Did not prevent PIV alteration, p < 0.01, values NR Alagebrium: Did not prevent PIV alteration, p < 0.01, values NR α-LPA: preserved PIV, p < 0.05, values NR	α-LPA treatment preserved the PIV response in diabetic mice
Demiot 2006[33]	n: 10 in each group Sex: Male Sp: Swiss mice Age: NR G: STZ-induced diabetes of 8 weeks with/without 2 week Sorbinil or Alagebrium treatment; healthy controls	PS: progressive, 2.2 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +55% SEM 6% at 0.4 kPa BF at 0.4 kPa after 8 weeks of STZ-induced diabetes: -33% SEM 8%, p < 0.001 Max PIV, Sorbinil: +43% SEM 7% at 0.4 kPa, p < 0.001 BF at 0.4 kPa, Alagebrium: -1% SEM 6%, p < 0.001	PIV needs intact vascular and C fiber functions

	with/without 2 week Sorbinil or Alagebrium treatment			
Demiot 2011[34]	n: 10 for every group Sex: Male Sp: Swiss mice Age: 5-6 weeks G: STZ-induced diabetes of 8 weeks with/without 2 week Erythropoietin treatment	PS: progressive, 2.2 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +35 SEM 9% at 0.4 kPa BF at 0.4 kPa after 8 weeks of STZ-induced diabetes: decreased (NR, only in figures) BF at 0.4 kPa, EPO: NR, but PIV was restored, $p < 0.01$	EPO increases resilience against pressure ulcer formation
Fizanne 2003[35]	n: 7-9 for every group Sex: Male Sp: Wistar rats Age: NR G: low doses of isoflurane anesthetic, high dose, high dose with gelofusine for blood pressure correction	PS: progressive, 11.1 Pa/s-1 Temp: 30 °C incubator Location: femur	Max PIV, low dose isoflurane: +37 SEM 10% at 2.0 kPa BF at 2.0 kPa, high dose isoflurane: -20 SEM 5%, $p < 0.001$ BF at 2.0 kPa, high dose isoflurane with blood pressure correction: -20 SEM 10%, $p < 0.001$	PIV is eliminated with high-dose anesthetics
Fizanne 2004[36]	n: 5-10 for every group Sex: Male (rats), mice NR Sp: Swiss mice & Wistar rats Age: NR Physiological targets: VPAC1/VPAC2, VPAC1, VPAC2/PAC1, PAC1	PS, mice: progressive, 2.2 Pa/s-1 PS, rats: progressive, 11.1 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C mice: +60 SEM 15% Max PIV, C rats: +60 SEM 15%	VPAC1/VPAC2 receptors are necessary for a full PIV response
Fromy 2000[37]	n: 9-20 for every group Sex: NR Sp: Wistar rats Age: NR Physiological targets: NK1, NK2&NK3, CGRP, Prostaglandins, endothelial NO, neuronal NO, capsaicin-sensitive nerve fibers (C fibers)	PS: progressive, 11.1 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +25 SEM 9% CGRP released by C fibers and endothelial NO are essential PIV mediators, amplified by prostaglandins	PIV depends on C fibers, CGRP and NO, and is amplified by prostaglandins
Fromy 2007[38]	n: 5-13 for every group Sex: Male Sp: Wistar rats Age: NR G: controls, acute pain with or w/o morphine, sympathectomy with or w/o	PS: progressive, 11.1 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +40 SEM 7% at 1.3 kPa BF at 1.3 kPa, pain: -31 SEM 6%, $p < 0.01$ BF at 1.3 kPa, morphine: +43 SEM 10%	PIV is eliminated during pain, but can be restored with pain management

	pain, with or w/o noradrenaline			
Fromy 2012[25]	n: 5-10 for every group Sex: Male Sp: ASIC3 KO +/+ and KO mice, Wistar rats Age: NR Physiological target: ASIC3 (by KO + pharmacological blockade with ASIC3-antagonists)	PS, mice: progressive, 2.2 Pa/s-1 PS, rats: progressive, 11.1 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: NR (only in figures) Elimination of PIV increased pressure ulcer formation during induction tests KO of ASIC3 was also associated with decreased post-pressure hyperemia, $p < 0.01$	ASIC3 is an essential mechanosensor for PIV to occur and protects against pressure ulcer formation Amiloride & Diclofenac eliminate PIV
Garry 2005[39]	n: 10 for every group Sex: NR Sp: Wistar rats Age: NR Physiological targets: BK_{Ca}, SK_{Ca}, K_v, K_{ATP}, channels	PS, rats: progressive, 11.1 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: values NR (only figures) K_{ATP} channels necessary for full response BK_{Ca} channels necessary for vascular smooth muscle relaxation in response to nitric oxide	PIV involves K_{ATP} and BK_{Ca} channels
Garry 2007[40]	n: 4-11 for every group Sex: NR Sp: TREK-1 +/+ and KO mice Age: NR Physiological target: TREK-1 channel	PS: progressive, 2.2 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +41 SEM 6% TREK-1 channels necessary for full PIV response	PIV involves TREK-1 channels
Gaubert 2007[41]	n: 5-13 for every group Sex: Male Sp: C57BL/6 mice Age: 12 weeks G: young adult (6–7 months) vs old (22–25 months) Physiological targets: NO, PGs and EDHF	PS: progressive, 2.2 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +39 SEM 6% at 0.39 kPa	PIV is dependent on EDHF instead of NO in old mice
Herrman 1999[42]	n: 6-7 for every group Sex: Male Sp: fuzzy rats Age: 12-14 months G: before and after 5 hours of pressure-induced ischemia	PS: progressive, 14.5 Pa/s-1 Temp: 28.6 SEM 0.27 (skin temperature) Location: trochanter	Max PIV, C: values NR outside of figure, but a significant increase is reported	After pressure-induced ischemia, no PIV could be induced
Pelletier 2012[43]	n: 10 for every group	PS: progressive, 11.1 Pa/s-1	Max PIV, C: +70 SEM 7%, corresponding pressure level NR	PIV was impaired following nerve compression and

	Sex: Male Sp: Wistar rats Age: NR G: Controls, compression neuropathy of 1 or 6 months, both tested again 1 month after nerve decompression	Temp: 30 °C incubator Location: hind limb	Max PIV, 1 month CN: +25 SEM 8%, $p < 0.001$ Max PIV, 6 months CN: values NR, but no PIV could be induced, $p < 0.001$ Max PIV, 1 month after release of 1 month of CN: +74 SEM 12%, $p < 0.001$ Max PIV, 6 month after release of 1 month of CN: +31 SEM 7%, $p < 0.01$	restored following nerve release
Sigaudo-Roussel 2004[44]	n: 9-12 for every group Sex: Male Sp: Swiss mice Age: NR G: STZ-induced diabetes of 1 week duration, controls	PS: progressive, 2.2 Pa/s-1 Temp: 30 °C incubator Location: cranial	Max PIV, C: +34 SEM 13% at 0.2 kPa BF at 0.2 kPa, 1 week diabetic mice: -3 SEM 7%, $p < 0.05$	Early endothelial impairment during diabetes is sufficient to impair PIV

ASIC3, acid-sensing ion channel 3; BF, blood flow; BK_{Ca}, SK_{Ca}, K_v, K_{ATP}, certain potassium channel types; C57BL/6 mice, regular type of lab mouse; CGRP, calcitonin gene-related peptide; CN, compression neuropathy; DM, diabetes mellitus; EDHF, endothelium-derived hyperpolarizing factor; EPO, erythropoietin; G:, groups; KO, indicates knock-out mouse (mouse genetically devoid of a certain gene); kPa, kilopascal; Max PIV, maximum pressure-induced vasodilatory capacity expressed in percentages in comparison to baseline blood flow; NK1, NK2 & NK3 receptors, neurokinin receptors for tachykinin peptides; NO, nitric oxide; NR, not reported; PIV, pressure-induced vasodilation; PS:, pressure stimulus characteristics; SEM, standard error of the mean; Sp:, species; STZ, Streptozotocin (method to induce diabetes in lab animals);Temp:, temperature; TREK-1, certain type of potassium channel; VPAC1/VPAC2, VPAC1, VPAC2/PAC1, PAC1, receptors for pituitary adenylate cyclase-activating polypeptide; w/o, without; α-LPA, alpha-lipoic acid.