Table, Supplemental Digital Content 3. Summary of included human studies.

Credits: The authors.

References	Study population (n, sex, age, groups with impairing/improving factor)	PIV measurement details (pressure stimulus: static/progressive, ramp, duration, temperature, location of measurement)	Results (max PIV, decrease at point of maximum PIV, physiological results, miscellaneous	Conclusions of the authors
Abraham 2001[22]	n: 8 Sex: 4/4 Age: 23 ± 1 G: Healthy volunteers subjected to several pressure regimens	PS: progressive, 4.4, 5.6, 11.1, or 16.7 Pa/s-1 + progressive, 16.7 Pa/s-1, then static for several minutes Temp: 23 ± 1°C (room) Location: finger	Max PIV, 4.4 Pa/s-1: +1 SEM 8% Max PIV, 5.6 Pa/s-1: +6 SEM 6% Max PIV, 11.1 Pa/s-1: +23 SEM 13% BF during 16.7 Pa/s-1: No PIV	PIV is a slow-responding phenomenon that is initiated by a wide arrange of pressure changes below painful stimulation
Bergstrand 2014[18]	G: <65 y subjects (n = 42); ≥65 y subjects (n = 38); ≥65 y patients* (n = 35) Sex: <65y subjects: 20m/22f; ≥65 y subjects: 16m/22f; ≥65y patients 23m/12f Age: <65y subjects: 41.7 ±12.9; ≥65 y subjects: 75.4 ± 7.8; ≥65y patients: 79.8 ± 7.9	PS: 10 minutes of static loading with bodyweight resulting from supine position Temp: 22.2 ± 2°C (room) Location: sacral	PIV, <65: +52 SEM 39% PIV, ≥65: +36 SEM 26% PIV, patients≥65: +53 SEM 36% PIV could also be measured at 2 mm and 10 mm depth	PIV can be observed at different tissue depths
Fromy 1998[12]	n: 10 Sex: NR Age: NR G: healthy volunteers before treatment, and after topical Capsaicin and local anesthesia	PS: progressive, 11.1 Pa/s-1 Temp: 23 ± 1°C (room) Location: finger	Max PIV, C: +53 SEM 10% at 4.9 kPa BF at 4.9 kPa, anesthesia: -55 SEM 18%, $p < 0.05$ BF at 4.9 kPa, Capsaicin: values NR (but similar to anesthesia), $p < 0.05$	PIV is mediated by C fibers
Fromy 2002[23]	G: Healthy controls, non-neuropathic diabetics, subclinical neuropathic diabetics, neuropathic diabetics (n =15 for every group) Sex: Healthy controls: 7m/8f; non-neuropathic diabetics: 9m/6f; subclinical neuropathic diabetics: 8m/7f; neuropathic diabetics: 8m/7f Age: Healthy controls: 47 ± 2; non-neuropathic diabetics: 40 ± 3; subclinical neuropathic diabetics: 58 ± 4; neuropathic diabetics: 50 ± 3	PS: progressive, 11.1 Pa/s-1 Temp: 25.6 ± 0.1°C (room) Location: medial malleolus	Max PIV, C: PIV was not observed Blood flow decreased significantly faster in diabetics, even in those without clinical or subclinical neuropathy, $p < 0.05$	PIV is impaired in diabetics, even those without subclinical or clinical neuropathy
Fromy 2010[24]	G: young (n = 12); non-neuropathic (n = 12); neuropathic (n = 10)	PS: progressive, 11.1 Pa/s-1	Max PIV, C: +62 SEM 4% at 4.2 kPa	PIV is impaired by aging and eliminated by peripheral neuropathy

	Sex: young: 8m/4f; non-neuropathic: 6m/6f; neuropathic: 4m/6f Age: young: 26 ± 1; non-neuropathic: 64 ± 1; neuropathic: 68 ± 2	Temp: 34 °C (skin, room NR) Location: finger	BF at 4.2 kPa, non-neuropathic: +12 SEM 7%, $p < 0.001$ BF at 4.2 kPa, neuropathic: -31 SEM 10%, $p < 0.001$	
Fromy 2012[25]	n: 20 Sex: 10/10 Age: 24 ± 1 G: injection with Amiloride vs placebo (saline, Diclofenac gel vs placebo gel (participants received every treatment)	PS: progressive, 11.1 Pa/s-1 Temp: range: 34 – 35 °C Location: dorsum of dig II or III	Max PIV, C: NR, only in figures Diclofenac and Amiloride eliminated PIV, $p < 0.01$	Diclofenac and Amiloride eliminate PIV
Hagblad 2012[17]	n: 10 Sex: 5/5 Age: mean 46 (25-76) G: healthy volunteers	PS: 60 minutes of static loading with bodyweight resulting from supine position Temp: 33.3 ± 0.9 °C (skin) Location: sacral	PIV: only range was provided: +3-262%, other values NR. Figure shows progressive increase during loading in all subjects. PIV could also be measured at 2 mm and 10 mm depth	The supine position results in a progressive increase of sacral blood flow at multiple depths in healthy subjects
Hsiu 2014[26]	n: 15 Sex: Male Age: 23-37 G: healthy volunteers subjected to several amounts of pressure	PS: static loading with 0 kPa; 2.7 kPa; 8 kPa; 13.3 kPa; or 21.3 kPa for 20 minutes Temp: mean temp between 31.8-32.1 °C (skin) Location: dorsum of first hand web	PIV: significant increases during 8 kPa; 13.3 kPa; 21.3 kPa of pressure stimulation, exact values not provided, (figures only)	Different amounts of pressure result in different PIV responses
Källman 2016[15]	n: 25 Sex: 16 male 9 female Age: 86 ± 7.6 G: nursing home residents >65 years	PS: static loading with bodyweight resulting from supine position in 0° or 30° for 60 minutes Temp: 34 °C (skin), room temp NR Location: sacral	mean max PIV NR 13/25 participants developed PIV at 1 mm depth PIV could also be measured at 2 mm and 10 mm depth	PIV occurs at multiple depths and remains present during one hour of loading
Källman 2015[16]	n: 25 Sex: 13 male; 12 female Age: 85 ± 7.3 G: nursing home residents >65 year subjected to several lying positions	PS: static loading with bodyweight resulting from supine position in 0° or 30°; or lateral position in 30° or 90° for 60 minutes Temp: 23.7 ± 0.9 °C (room) Location: sacral or trochanter major	Mean max PIV NR During loading, mean blood flow progressively increased in all positions and at 1, 2 and 10 mm depth at both locations	The 30° supine tilt position allows greater tissue perfusion
Koïtka 2004[11]	n: 24 Sex: NR	PS: progressive, 11.1 Pa/s-1	Max PIV, C: +44 SEM 14% at 3 kPa	PIV exists at the foot level in normal subjects, whereas in

	Age: DM1: 22 ± 1; controls 23 ± 1 G: 12 healthy controls; 12 subjects with DM1	Temp: 29.5 ± 0.2 °C (room) Location: head of metatarsus I	BF at 3 kPa, DM1: -1 SEM 28%, p < 0.001	young DM1 diabetics it does not because of endothelial dysfunction
Koïtka 2004[27]	n: 10 Sex: 6 male; 4 female Age: 25 ± 1 G: healthy volunteers subjected to different ambient temperatures	PS: progressive, 11.1 Pa/s-1 Temp: Low (29.0 ± 0.3°C); intermediate (32.6 ± 0.1°C); high (33.9 ± 0.1°C); very high (36.0 ± 0.1°C) (skin temperatures) Location: head of metatarsus I	BF at 4 kPa, low temp: -40 SEM 10% BF at 4 kPa, intermediate temp: -32 SEM 8% Max PIV, high temp: +36 SEM 17% Max PIV, very high temp: +48 SEM 18%	PIV does not occur during low temperatures
Sae-Sia 2007[28]	n: healthy controls: 47; SCI: 20; other trauma: 35 Sex: SCI: 20 male 0 female; other trauma 34/1; healthy 38/9 Age: SCI: 38.7 ± 4.3; other trauma 39.4 ± 3.0; healthy 33.8 ±1.8 G: healthy controls; SCI patients; other trauma patients	PS: static loading with bodyweight resulting from supine position in 0° for two hours Temp: 29.0 ± 0.1°C Location: sacral	BF during loading, controls: increased in comparison to baseline at every time point, p < 0.01 BF during loading, SCI: Decreased in comparison to baseline at every time point, p < 0.01 BF during loading, trauma: increased in comparison to baseline at every time point, p < 0.01	PIV is eliminated immediately after SCI
Tzen 2013[29]	n: 14 SCI; 14 controls Sex: SCI 13 male / 1 female; controls 7/7 Age: SCI 33.79 ± 11.12; controls 37.79 ± 10.96 G: SCI & healthy controls; subjected to fast (-4°C/min) or slow (-0.33°C/min) cooling of the skin to 25 °C	PS: constant, 8 kPa for 30 minutes Temp: 22 ± 1 °C (room) Location: sacral (while prone)	No PIV could be detected: BF decreased in both controls and SCI patients during loading with no differences in BF between both fast and slow cooling	No relevant conclusions regarding PIV

BF, blood flow; dig, digit; DM1, diabetes mellitus type 1; G:, groups; kPa, kilopascal; Max PIV, maximum pressure-induced vasodilatory capacity expressed in percentages in comparison to baseline blood flow; mm, millimeters; NR, not reported; Pa, Pascal; PIV, pressure-induced vasodilation; PS, pressure stimulus characteristics; SCI, spinal cord injury; SEM, standard error of the mean; Temp, temperature. *'Patients were recruited from the Departments of Neurology, Acute Internal Medicine, Hand Surgery, Plastic Surgery and Burns, and Geriatric Medicine'.