2.1: KNOCK-OUT OF MATRIX METALLOPROTEINASE-12 EXACERBATES COMPROMISED MECHANICAL HOMEOSTASIS IN ARTERIAL AGING

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brachial artery with a clinically validated automatic sphygmomanometer (OMRON 705IT) and an appropriately sized cuff. Gender-specific percentiles were used for the definition of the individual BP phenotype. Carotid-femoral PWV was measured to all participants at the third clinical evaluation, with the Compilior SP device, complying with the methodological recommendations. All participants were evaluated by the same experienced clinician.

**Results:** Mean PWV was 6.20 ± 0.95 m/s and was higher in males compared with females (6.31 ± 0.97 m/s vs 6.02 ± 0.89, respectively; p < 0.0001). Gender-specific percentile tables, accounting for age, were obtained from the normotensive participants (n = 758), as depicted in Figure 1. The determinants of PWV were assessed through linear regression. In a multivariable model, age, gender, blood pressure and family history of cardiovascular disease were significantly associated with PWV.

**Conclusion:** In children and adolescents, aortic PWV is strongly influenced by age, gender, BP and genetics, in line with the available evidences in adult populations. Further studies are needed towards a thorough understanding of the arterial dynamics at these ages.

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**References**


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**Oral Session II — Young Investigator Award**

### 2.1 KNOCK-OUT OF MATRIX METALLOPROTEINASE-12 EXACERBATES COMPROMISED MECHANICAL HOMEOSTASIS IN ARTERIAL AGING

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**Background:** Matrix metalloproteinase-12 (MMP12) may modulate arterial stiffening with age [1]. We aimed to study the effect of aging on biaxial arterial stiffening in wild-type (WT) and MMP12 knock-out (MMP12-/-) mice.

**Methods and Results:** After euthanasia, descending thoracic (DTA) and suprarenal abdominal (SAA) aortas of young and old, WT (ages 21 ± 0 and 103 ± 1 weeks; mean ± SE) and MMP12-/- (13 ± 0 and 52 ± 0 weeks) male mice were dissected and cannulated on glass pipettes in a computer-controlled biaxial testing device. Pressure-diameter tests were performed at 95%/100%/105% of estimated in vivo stretch; axial force-length tests were comparable (133 ± 88 vs. 126 ± 93 mmHg; SBP/DBP; telemetry); WT aging did not influence blood pressure [3]. All metrics are therefore presented at a common pressure (figure). At first sight, MMP12-/- aging resembles WT aging: increased blood pressure [3]. All metrics are comparable at 95%/100%/105% of estimated in vivo stretch; axial force-length tests were comparable (133 ± 88 vs. 126 ± 93 mmHg; SBP/DBP; telemetry); WT aging did not influence blood pressure [3]. All metrics are therefore presented at a common pressure (figure). At first sight, MMP12-/- aging resembles WT aging: increased blood pressure [3]. All metrics are comparable at 95%/100%/105% of estimated in vivo stretch; axial force-length tests were comparable (133 ± 88 vs. 126 ± 93 mmHg; SBP/DBP; telemetry); WT aging did not influence blood pressure [3]. All metrics are therefore presented at a common pressure (figure). At first sight, MMP12-/- aging resembles WT aging: increased blood pressure [3].

**Discussion:** Our findings suggest that in both WT and MMP12-/-, mechanical homeostasis with aging was compromised, a finding that was exacerbated with MMP12-/-. MMP12-/- was previously reported to reduce age-associated stiffening [1]. This contradictory finding may be explained by the use of atomic force microscopy in [1] (measuring compressive stiffness) versus our use of tensile biaxial testing.

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**References**


