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Estimating normal and reference pulse wave velocity in New Zealand population

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ABSTRACT

This study aimed to establish normal and reference pulse wave velocity (PWV) values and assess their relationship with the established cardiovascular risk factors in NZ. An observational cross-sectional study of 120 adults above 18 years was conducted at Auckland University of Technology within three age groups (18-30, 31-60, and >60 years) and four blood pressure (BP) categories (normal, elevated, stage 1 and stage 2) before being clustered as Normal Value Population (NVP) and Reference Value Population (RVP) based on BP and presence of other CVD risk. PWV was measured using Doppler ultrasound via the direct carotid-femoral method (distance × 0.8). Mean PWV for the whole study population was 5.88 ± 1.49 m/s and increased with age ($p < 0.001$) and with a more profound rise in the RVP group ($p < 0.001$, z = -4.98). Overall PWV values were lower than international reference standards, which may reflect population-specific physiological or methodological factors. A significant difference in mean PWV in the European and non-European populations ($p = 0.004$) indicated that ethnicity was an important part of the assessment. Age and mean blood pressure were the strongest predictors of PWV, accounting for 54.5% of the variability.

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KEYWORDS
Pulse wave velocity (PWV); arterial stiffness; cardiovascular risk assessment; New Zealand population; blood pressure

Introduction

Arterial stiffness, characterised by the loss of elasticity and distensibility, is a hallmark of vascular ageing and a key predictor of cardiovascular disease (CVD) events (Cecelja and Chowienczyk 2010; Cecelja and Chowienczyk 2012; Cecelja and Chowienczyk 2012; Palombo and Kozakova 2016). It has gained clinical prominence amongst other biomarkers and associated measurement modalities as a non-invasive biomarker that offers predictive value beyond traditional cardiovascular risk factors, particularly in individuals with diabetes, obesity and hypertension (Kingwell and Gatzka 2002; McLean et al. 2013; Upadhyay 2015; Weber et al. 2017; Lyngbakken et al. 2019; Kalra and Lowe 2020;

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Ojji et al. 2020; Anand et al. 2021; Dahiya et al. 2024). Among various methods available, carotid–femoral pulse wave velocity (cf-PWV) remains the gold standard for evaluating central arterial stiffness, reflecting the velocity of pressure wave propagation along the aortic and central arteries (Cavalcante et al. 2011).

Pulse wave velocity (PWV) measurement protocols vary considerably across studies, with approaches ranging from local site-specific assessments to regional segmental measurements. These methodological differences – particularly in estimating arterial path length, pulse transit time, device-specific protocols and participant inclusion criteria – can significantly affect the comparability of PWV values. To address these inconsistencies, expert consensus guidelines have established the cf-PWV as the gold-standard method for non-invasive evaluation of central arterial stiffness (Laurent et al. 2006; Van Bortel et al. 2012). This approach standardises the measurement of superficial distance and transit time between the common carotid and femoral arteries and has demonstrated independent prognostic value for cardiovascular risk beyond traditional factors (Laurent et al. 2006; Cavalcante et al. 2011; Van Bortel et al. 2012; Milan et al. 2019). In alignment with these international recommendations, the present study employed the cf-PWV method to allow comparison with global reference standards.

In the context of New Zealand (NZ), establishing normative PWV values is especially important due to the country's aging and ethnically diverse population. National data show that Māori, the Indigenous people of NZ, experience disproportionately higher rates of CVD compared to other ethnic groups (Cameron et al. 2012; Grey et al. 2018; Mason et al. 2019; Gilheany-Black and Davis 2021; Brewer et al. 2022; Statistics New Zealand 2024). According to the Global Burden of Disease (GBD) study, CVD and stroke remain among the leading causes of mortality and morbidity in NZ (Institute for Health Metrics and Evaluation (IHME) 2024). The economic burden is also significant; each stroke is estimated to cost between $NZ 60,000 and $99,000 over five years, contributing to a national cost exceeding $NZ 880 million annually (Hogan and Siddharth 2018). These realities underscore the need for early, accurate risk identification strategies – including vascular biomarkers like PWV – to inform targeted prevention and reduce long-term healthcare costs.

While international guidelines such as those from the European Society of Cardiology (ESC), the European Society of Hypertension (ESH) and the American Heart Association (AHA) have established reference thresholds for PWV of 10 m/s, these are based on population data that may not apply to NZ due to demographic differences (Laurent et al. 2006; Mancia et al. 2007; Townsend 2015). The 2018 New Zealand CVD Risk Assessment and Management Guidelines rely on five-year risk equations from the PREDICT study, extended the Framingham risk score – a nationally integrated, web-based tool that accounts for ethnic variability and incorporates real-world clinical data (Wells et al. 2017; Ministry of Health 2018; Pylypchuk et al. 2018; Tawfiq et al. 2023; Lin et al. 2024). However, this tool currently lacks normative PWV parameters relevant to NZ's population.

Given NZ's multicultural population and wide ethnic diversity, reference values from other published guidelines cannot be directly applied to the NZ population without consideration. Due to limited local data, the current study aimed to (1) establish normative and reference cf-PWV values in a sample of the NZ population and (2) examine associations between PWV and traditional cardiovascular risk factors, including ethnicity.

These findings are intended to support the integration of PWV into routine cardiovascular risk assessments in NZ's diverse primary care settings.

Methods

Study design

This cross-sectional observational study was conducted at the Auckland University of Technology (AUT). The study was part of a doctoral project funded by the Vice Chancellor Scholarship. We aimed to estimate normal and reference values for aortic pulse wave velocity (PWV) in a New Zealand adult population and examine its association with cardiovascular risk factors. Participants were categorised into two subgroups – Normal Value Population (NVP) and Reference Value Population (RVP) – to reflect differing cardiovascular risk profiles. The first group, NVP, had normal BP with no additional cardiovascular risk factors. The second group, RVP, expanded the normal value sub-set to include people with cardiovascular risk factors without independent influence on PWV values.

Inclusion and exclusion criteria

Inclusion Criteria: Participants aged 18 years and of both sexes with all ranges of BP were selected. The selected participants were categorised according to the 2017 AHA guidelines (Whelton et al. 2018) for their blood pressure (BP) categories:

1. Normal: < 120/80 mmHg,
2. Elevated: systolic 120–129 mmHg and diastolic <80 mmHg,
3. Stage 1 Hypertension: systolic 130–139 mmHg or diastolic 80–89 mmHg,
4. Stage 2 Hypertension: systolic at least 140 or higher or diastolic at least 90 mmHg or higher

Exclusion Criteria: Individuals with known CVD, diabetes, renal disease, chronic inflammatory conditions, or taking lipid-lowering or antidiabetic medications, pregnant individuals, those with irregular cardiac rhythms (e.g. atrial fibrillation), or those with poor-quality vascular recordings were excluded. It is known that PWV is dependent on systolic blood pressure, and all anti-hypertensive drugs lower the PWV value (Liu et al. 2013); therefore, participants on any BP-lowering drugs were also excluded.

Participant screening and recruitment

A total of 120 adult participants (aged ≥18 years) were recruited via public advertisements, flyers and word-of-mouth. Interested individuals underwent initial verbal prescreening to determine eligibility. This study was approved by the New Zealand Health and Disability Ethics Committee (HDEC; Reference: 18/STH/45). Eligible participants were invited for a 30-minute study session where they received a detailed Participant Information Sheet. The study aims, procedures and expectations were explained, and participants were given the opportunity to ask questions. All participants provided

written informed consent. Personal identifiers were removed, and participants were assigned anonymised codes to ensure confidentiality and data security.

Outcome measures

Demographic data (age, sex, ethnicity, employment status), clinical history (smoking, alcohol use, diabetes, cholesterol, medications) and anthropometrics (height, weight, BMI) were recorded. Cardiovascular measurements included peripheral and central blood pressure, mean arterial pressure (MAP), pulse rate (PR), augmentation index (AIx) measured using Uscom BP+ automated BP measurement device, and carotid–femoral PWV with ACUSON Sequoia c512 Echocardiography Ultrasound System.

Measurement procedure

BP measurement: BP was measured in a seated position. After a 5-minute rest, three readings were taken at 2-minute intervals. If values differed by more than ±10 mmHg, additional readings were taken. The average of two consistent readings was recorded. The device calculated the AIx automatically.

PWV measurement: PWV was measured using Doppler ultrasound with a 6L3 linear probe and ECG leads. Participants were placed supine, and ECG electrodes were attached to the wrists and right ankle. Pulse transit time was measured from the ECG's R-wave to the pulse wave's foot at the carotid and femoral arteries across three cardiac cycles (Figures 1 and 2). Arterial path length was measured using a measuring tape (direct distance method), and PWV was calculated using the formula: $\boldsymbol{PWV = (Distance \times 0.8) / Transit\ Time}$, in line with European reference protocols to ensure comparability with established norms (Van Bortel et al. 2012).

Statistical analysis

All analyses were conducted using SPSS. Descriptive statistics were presented as means ± SD or medians (IQR), depending on data distribution. Group comparisons used t-tests or ANOVA for normally distributed data and Mann–Whitney U or Kruskal–Wallis tests for non-parametric data. The correlation between PWV and cardiovascular risk factors was assessed using Pearson or Spearman correlation coefficients. Effect sizes were interpreted using standard thresholds (small = 0.10, medium = 0.30, large = 0.50). ANCOVA was used to adjust for covariates such as age and MAP, and linear regression models were developed to predict PWV based on cardiovascular risk profiles.

Results

Out of 195 initial responses, 120 participants met the eligibility criteria and were categorised into Normal Value Population (NVP, n = 77) and Reference Value Population (RVP, n = 43) based on the presence or absence of cardiovascular risk factors. Table 1 summarises the demographic and clinical characteristics across both groups. The average age of the total population was 45.6 years, with a mean age of 39.7 years in the NVP and 56.5 years in the RVP. The overall population had a balanced

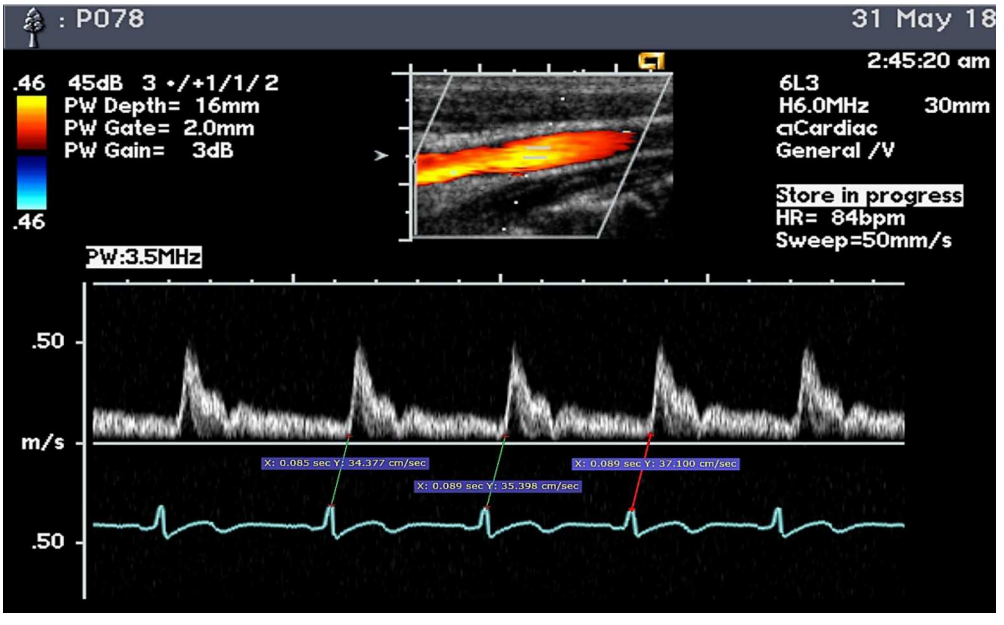

Figure 1. Doppler ultrasound of the pulse wave at the common carotid artery.

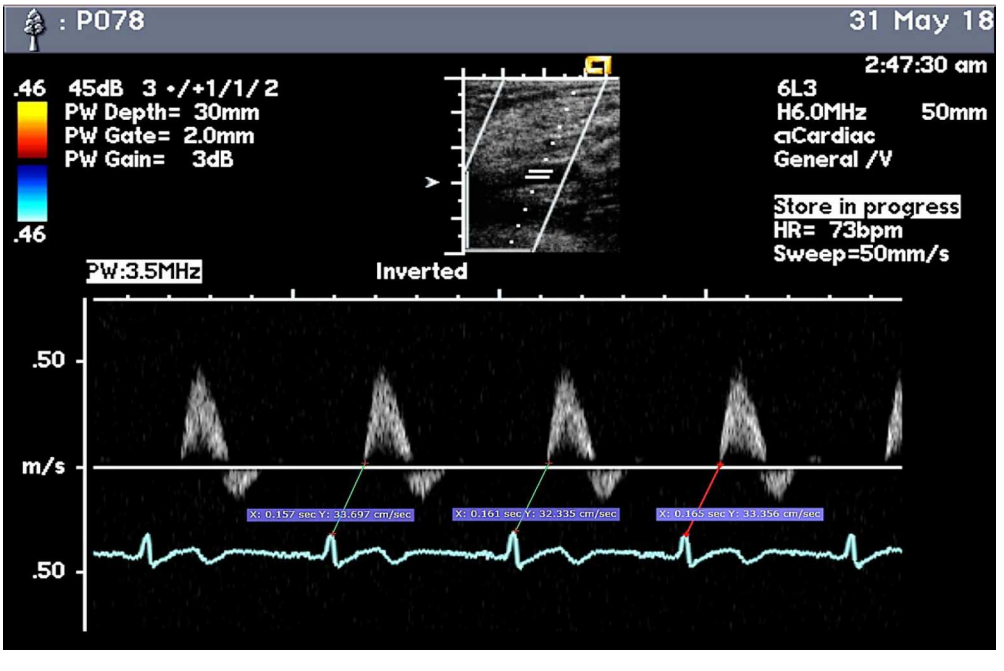

Figure 2. Doppler ultrasound of the pulse wave at the common femoral artery.

sex distribution (54 males, 66 females) and included 54.2% of participants of European descent. Significant differences were observed in blood pressure parameters between the groups. The mean systolic BP (SBP) in the RVP group was 138 mmHg compared to 111 mmHg in the NVP.

Table 1. Demographic and clinical characteristics of NVP and RVP study population.

Parameters	Normal value population	Reference value population	Whole population
N	77	43	120
Age [years, mean (± SD)]	39.69 (16.95)	56.52 (18.0)	45.58 (19.04)
Age range (years)	19–88	21–85	19–88
Sex (M/F)	37/40	17/26	54/66
Ethnicity (E/NE)	36/41	17/26	65/55
Height [m, mean (± SD)]	1.69 (0.11)	1.66 (0.10)	1.68 (0.10)
Weight [kg, mean (± SD)]	71.77 (14.46)	71.69 (16.06)	71.74 (14.99)
BMI [kg/m^2, mean (± SD)]	24.99 (4.31)	25.85 (4.70)	25.30 (4.45)
SBP [mmHg, mean (± SD)]	111 (10)	138 (16)	121 (18)
DBP [mmHg, mean (± SD)]	72 (9)	86 (10)	77 (11)
CSBP [mmHg, mean (± SD)]	105 (10)	131 (15)	114 (17)
CDBP [mmHg, mean (± SD)]	73 (9)	86 (10)	78 (11)
MBP [mmHg, mean (± SD)]	88 (8)	107 (11)	95 (13)
PR [bpm, mean (± SD)]	76 (17)	71 (15)	74 (17)
AIx % [mean (± SD)]	63.09 (40.65)	106.48 (64.84)	78.63 (54.55)
Smoking- Yes [n (%)]	13 (17)	5 (12)	18 (15)
Alcohol consumption [Yes, n (%)]	47 (61)	32 (74)	76 (63)
Diabetes [Yes, n (%)]	-	0	0
Dyslipidaemia [Yes, n (%)]	-	3 (2.5)	3 (2.5)
Hypertension [Yes, n (%)]	-	30 (25)	30 (25)
Mean PWV [m/s, mean (± SD)]	5.40 (1.21)	6.73 (1.56)	5.88 (1.49)

SD: Standard Deviation, M: Male, F: Female, E: European population, NE: non-European population, BMI: Body Mass Index, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, CSBP: Central Systolic Blood Pressure, CDBP: Central Diastolic Blood Pressure, MBP: Mean Blood Pressure, PR: Pulse Rate, AIx: Augmentation Index, PWV: Pulse Wave Velocity, n: number of participants.

The mean PWV of the whole study population (n = 120) was 5.88 ± 1.49 m/s. Participants with normal BP constituting the NVP group had the lowest mean PWV value at 5.40 ± 1.21 m/s, while in the RVP group with cardiovascular risk factors, the mean PWV was 6.74 ± 1.56 m/s. The mean PWV values of the NVP and RVP groups were significantly different ($p < 0.001$, $z = -4.986$) when compared using the non-parametric test (Mann–Whitney Test).

PWV across age groups

Participants were stratified into age categories (18–30, 31–60 and >60 years). The mean PWV values for each group in different age groups are shown in Table 2. A significant difference in PWV was observed between the NVP and RVP within the 31–60 age group ($p = 0.02$), while no statistically significant differences were found in the youngest and oldest age groups. The mean PWV for participants over 60 years was 7.08 m/s, the highest among all age groups.

The mean and median PWV values, along with the 25th and 75th percentile with age across different age groups, are shown in Figure 3. The mean PWV values compared within the three age categories were significantly different, the highest being the over 60 years of age group with 7.08 m/s [18-30 vs. 31–60 ($p < 0.001$, $z = -3.26$), 18–30 vs. > 60 ($p < 0.001$, $z = -6.40$), 31–60 vs. > 60 ($p < 0.001$, $z = -4.16$)].

PWV differences by sex and ethnicity

When analysed by sex using a non-parametric Mann–Whitney test, no significant differences were found in PWV within individual age categories. However, the mean PWV for

Table 2. Mean pulse wave velocity in the normal and reference groups as per the age category.

	Mean PWV (± SD) (m/s)				
Age category (years)	NVP	n	RVP	n	*P*-value
18–30	4.75 (0.75)	34	5.10 (0.22)	6	0.063
31–60	5.48 (0.97)	28	6.39 (1.32)	12	0.020*
> 60	6.72 (1.41)	15	7.30 (1.55)	25	0.171
All	5.40 (1.21)	77	6.74 (1.56)	43	0.001***

PWV: Pulse Wave Velocity, SD: Standard Deviation, NVP: Normal Value Population, RVP: Reference Value Population, *: $p < 0.05$, **: $p < 0.01$, ***: $p < 0.001$.

females across the entire population was significantly higher than for males (6.15 vs. 5.56 m/s, $p = 0.03$; Table 3). A significant difference in PWV was also observed between European (n = 65) and non-European (n = 55) participants (6.20 vs. 5.50 m/s, $p = 0.004$), suggesting ethnicity may play a role in arterial stiffness.

Correlation between PWV and cardiovascular risk factors

Correlation analysis revealed strong positive relationships between PWV and age (r = 0.71), mean blood pressure (MBP; r = 0.49), BP categories (r = 0.50), and population group (NVP/RVP; r = 0.46), all statistically significant at $p < 0.01$. BMI showed a moderate positive correlation (r = 0.21), while sex showed a small effect (r = 0.17, $p = 0.02$). Height and weight were not significantly correlated with PWV. Table 4 summarises the correlation of cardiovascular risk factors with PWV. The association between the mean PWV and BP categories as per the age groups is depicted in Figure 4. For each age group, the elevated BP group had lower PWV scores compared to other BP

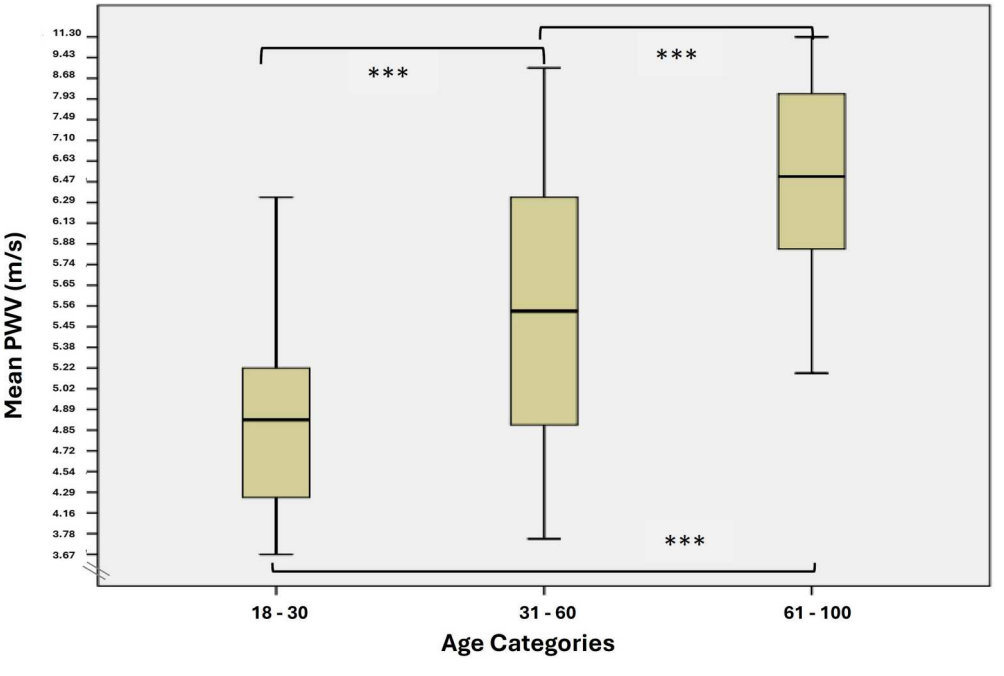

Figure 3. Box plot distribution of the mean pulse wave velocity (PWV) in different age groups (***: $p < 0.001$ indicates a statistically significant difference of the respective age groups).

Table 3. Mean pulse wave velocity by sex and age category.

	Mean PWV (± SD) (m/s)				
Age category (years)	Male	n	Female	n	*P*-value
18–30	4.91 (0.75)	23	4.67 (0.63)	17	0.35
31–60	5.48 (0.82)	19	6.00 (1.35)	21	0.54
> 60	6.92 (1.80)	12	7.15 (1.40)	28	0.44
All	5.56 (1.2)	54	6.15 (1.57)	66	0.03*

PWV: Pulse Wave Velocity, SD: Standard Deviation, NVP: Normal Value Population, RVP: Reference Value Population, *: $p < 0.05$.

categories. Notably, PWV values in the elevated BP categories were unexpectedly lower than those in the normal BP group, particularly in younger participants.

Regression analysis: predictors of PWV

Linear regression was conducted to determine the association of the outcome variable or dependent variable, i.e. mean PWV from a predictor or independent variable. As a cardiovascular risk factor, age accounts for 48.8% of the variability in mean PWV [$R^2 = 0.49$, $F(1, 118) = 112.32$, $p < 0.001$]. It was evident that age significantly predicted mean PWV ($\beta = 0.55$, $p < 0.001$). The regression equation having age as a predictor for mean PWV was as follows:

$$PWV = 3.391 + 0.05 \times Age$$

The graph in Figure 5 demonstrates the regression equation and the correlation between age and mean PWV. The addition of MBP as a predictor variable into the age-centred regression model influenced mean PWV significantly ($p < 0.001$). The enhanced prediction by MBP accounted for 54.5% variability in an age-centred mean PWV [$R^2 = 0.54$, $F(2,117) = 70.17$, $p < 0.001$]. The adjusted regression model equation was expressed as:

$$PWV = 0.908 + 0.04 \times Age + 0.03 \times MBP$$

A multivariate model including age, BMI, BP category and population group status (NVP/RVP) explained 57% of the total variance in PWV ($p < 0.001$). A stepwise backward method was used to enter the predictors in the model. Residual plots confirmed the normality and validity of the regression model (Figures 6 and 7). A horizontal regression line at $R^2 = 0$ affirms the hypothesis. The regression equation was:

$$PWV = 2.708 + 0.04 \times Age + 0.04 \times BMI + 0.69 \times BP\ Category - 0.81 \times NVP\ or\ RVP$$

Table 4. Correlation of cardiovascular risk factors with pulse wave velocity (PWV).

Cardiovascular Risk Factors	Correlation Coefficient (r)	*p*-value	Effect size
Age	0.71	<0.01*	Large
MBP	0.49	<0.01*	Large
BP categories	0.50	<0.01*	Large
NVP or RVP group	0.46	<0.01*	Large
BMI	0.21	<0.01*	Medium
Sex	0.17	0.02*	Small

BP: Blood Pressure, MBP: Mean Blood Pressure, NVP: Normal Value Population, RVP: Reference Value Population, BMI: Body Mass Index, *: $p < 0.05$.

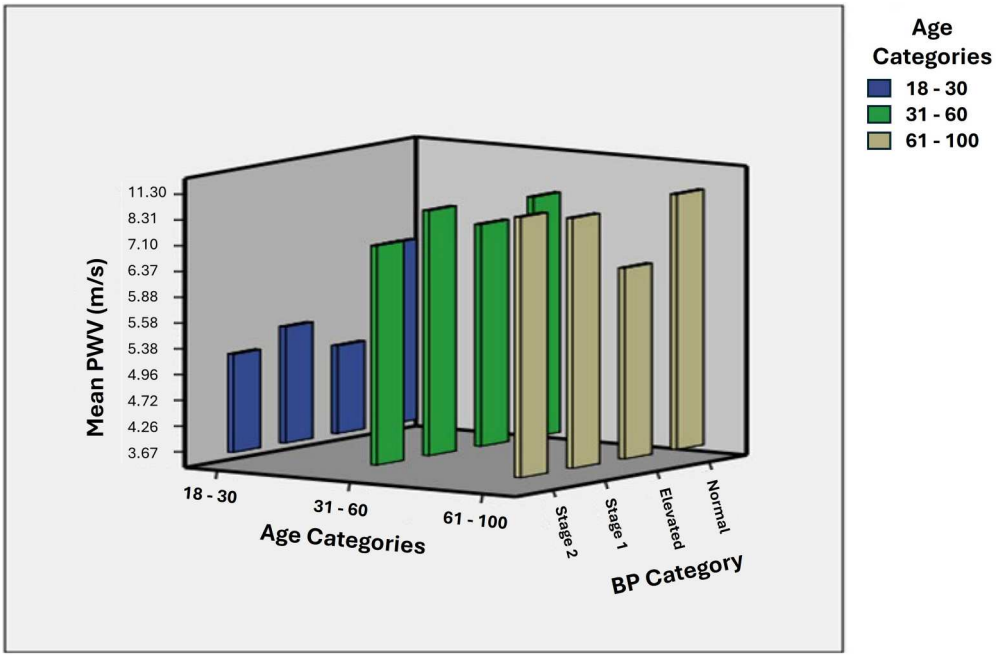

Figure 4. Mean pulse wave velocity (PWV) values according to the age and blood pressure (BP) categories.

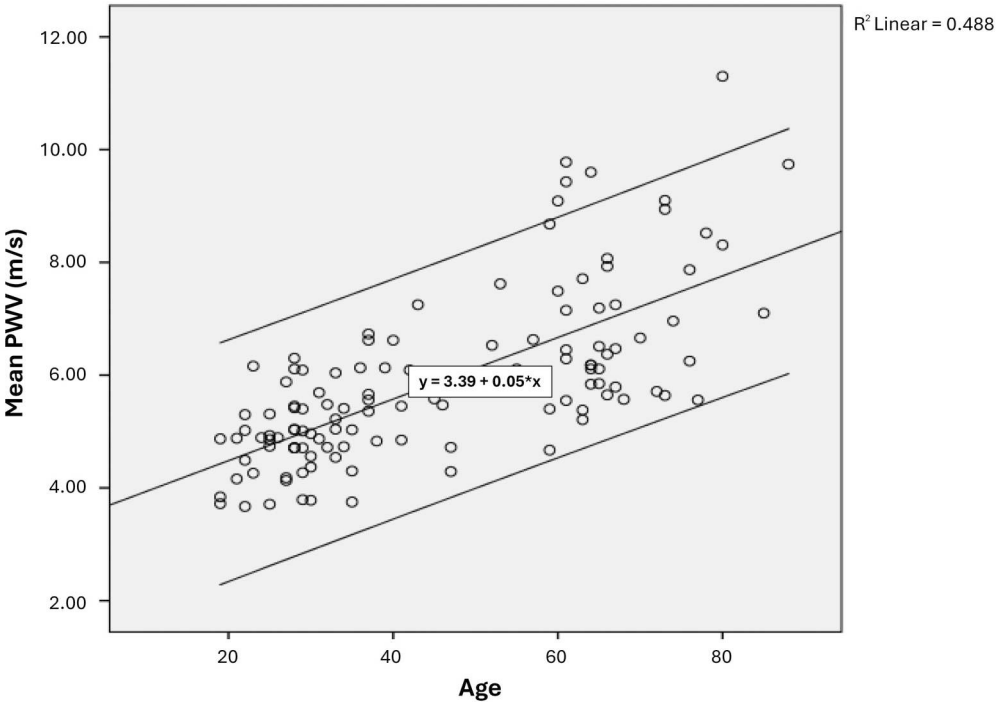

Figure 5. Mean pulse wave velocity (PWV) and age linear regression model.

Table 5 and Figure 8 show a comparative analysis of multiple regression analysis from the present study and data published by other international studies with similar age ranges but varied sample sizes. Although coefficients varied across populations, age and BP consistently emerged as primary predictors of PWV. The present study's regression model aligned most closely with findings from African and European populations with similar age ranges.

Dummy regression by BP category

To further examine the influence of BP, dummy variables for BP categories were entered into a regression model. The results showed significant increases in PWV for Stage 1 ($\beta = 1.27$, $p < 0.001$) and Stage 2 hypertension ($\beta = 2.42$, $p < 0.001$) compared to normal BP, accounting for 30.6% of the variability. The elevated BP category, however, did not significantly affect PWV ($\beta = 0.33$, $p = 0.40$), highlighting a non-linear association between BP and arterial stiffness in this cohort. The regression of mean PWV on these dummy variables resulted in the following model:

$$\begin{aligned} \textit{Mean PWV} = {} & 5.406 + 2.42 \times (\textit{Normal vs Stage 2}) + 1.27 \times (\textit{Normal vs Stage 1}) \\ & + 0.33 \times (\textit{Normal vs Elevated}) \end{aligned}$$

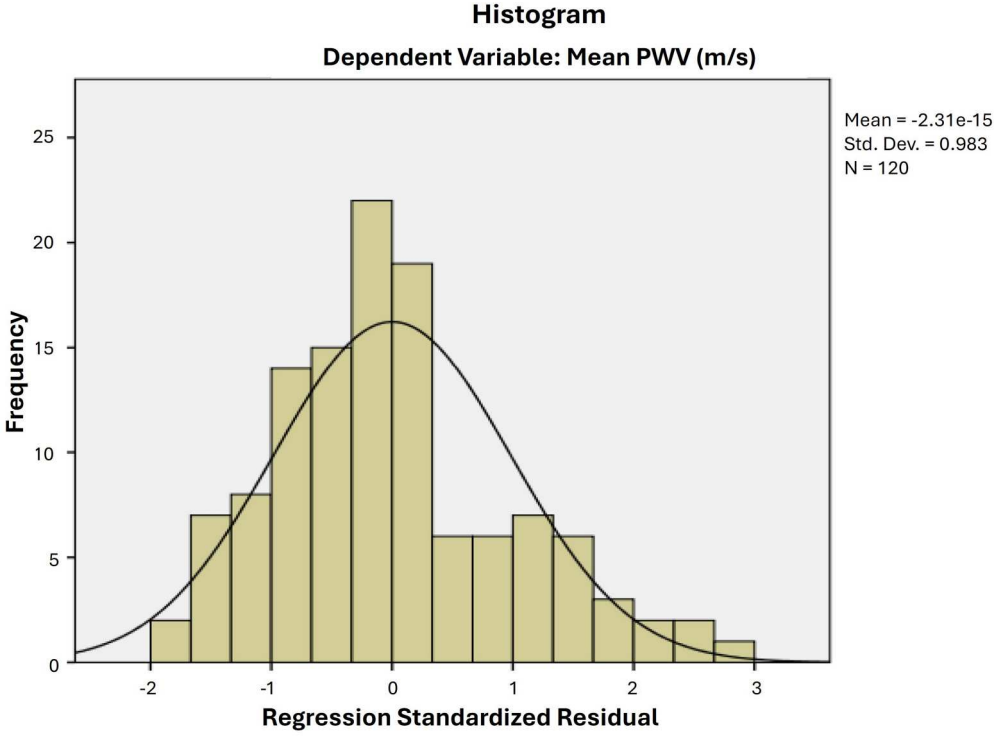

Figure 6. A histogram of the regression standardised residual.

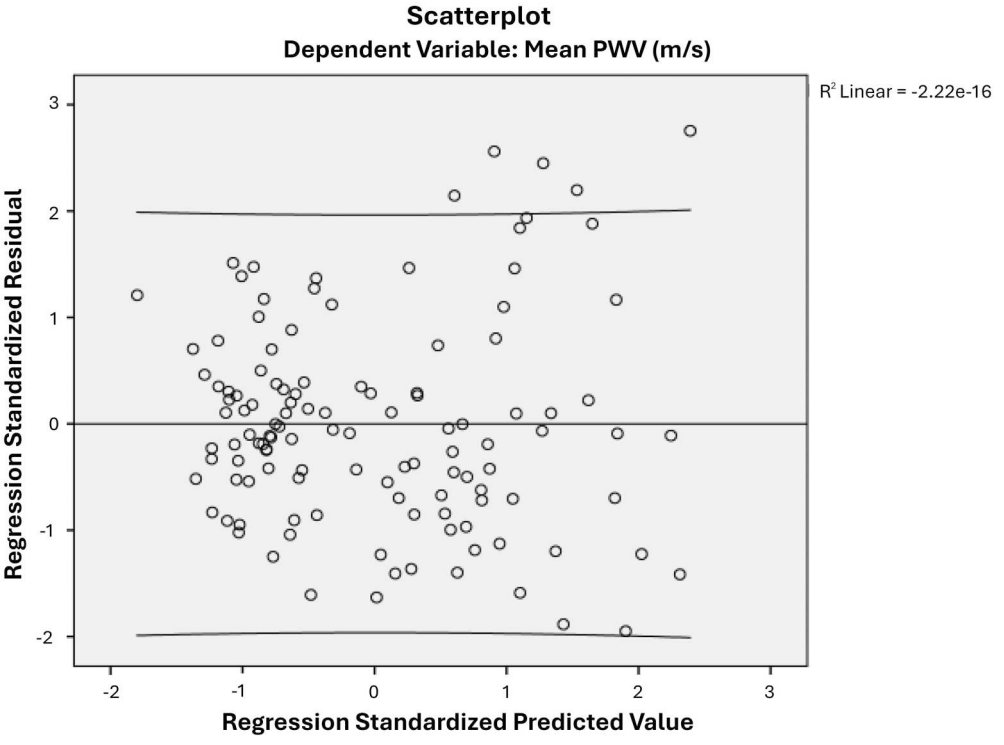

Figure 7. A scatterplot of the regression standardised residuals and predicted mean pulse wave velocity (PWV) values.

Table 5. Comparative analysis of regression analysis of the present study with other published literature.

Reference	Unstandardised coefficient	Study sample description
Ferreira et al. 1999	PWV = 4.078 + 0.042 x age + 0.024 x SBP	120, 19–50 years, African and European
Nguyen et al. 2008	For white race, R^2 = 17.5 MA*P* = 0.28, age = 0.22, smoking = 0.13, HR = 0.08, adiponectin = –0.006 For black race, R^2 = 23.1 MA*P* = 0.32, age = 0.18, smoking = 0.14, HR = 0.12, adiponectin = – 0.13	991, 24–44 years, white and black American
Elias 2011	PWV = 10.059 + 1.069 x age group + 0.447 x BP diagnostic category + 0.081 x age group x BP R^2 = 0.38	626, 40–93 years, American
Santos et al. 2011	Ethnicity = 0.246, gender = 0.217, age = 0.071, MB*P* = 0.046, HR = 0.004, BMI = −0.041	1427, 25–64 years, Brazil
McEniery et al. 2005	Age = 0.078, MB*P* = 0.034, HR = 0.016, gender (female) = – 0.266 Adjusted R^2 = 0.65	4001, 18–90 years, American
Reusz et al. 2010	PWV = 1.129 + 0.049 x age + 0.008 x height + 0.024 x MAP	450, European, healthy school children and adolescents
Magalhães et al. 2013	PWV = 1.899 + 0.065 x age + 0.057 x MBP – 0.504 x gender (female) R^2 = 0.34	301, 22–72 years, African
Present study	PWV = 2.708 + 0.04 × Age + 0.04 × BMI + 0.69 × BP Category - 0.81 × PopuNR	120, 19–88 years, New Zealanders

PWV: Pulse Wave Velocity, SBP: Systolic Blood Pressure, MAP: Mean Arterial Pressure, HR: Heart Rate, MBP: Mean Blood Pressure, BMI: Body Mass Index, PopuNR: being in the normal or reference value population.

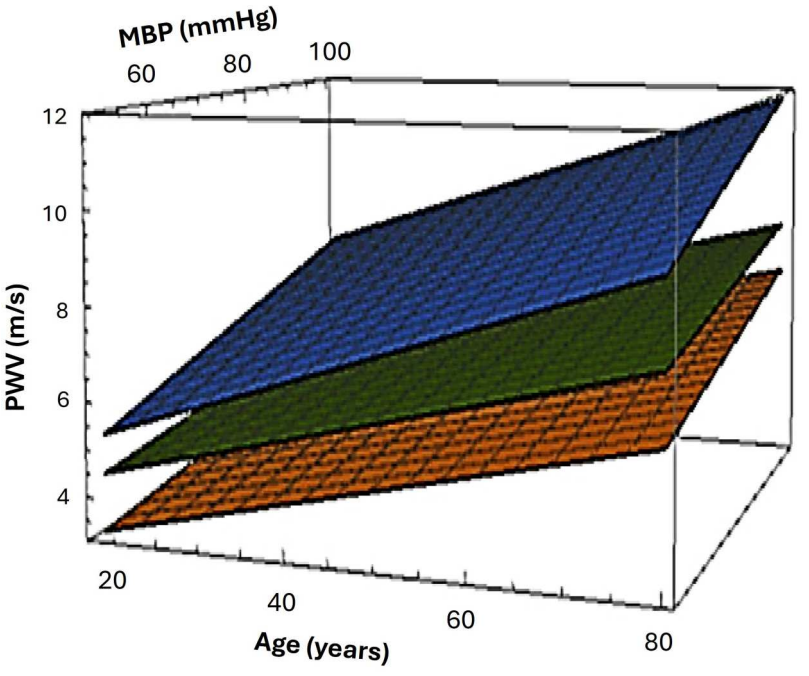

Figure 8. Graphical representation of regression equations involving age and mean blood pressure (MBP), Regression equations from the studies with African (Magalhães et al. 2013 ■) and European (Reusz et al. 2010 ■) populations plotted against the present study (■) multivariate regression model.

Discussion

This study provides the first known attempt to estimate normative and reference aortic PWV values in an NZ population using Doppler ultrasound, alongside analysis of cardiovascular risk factors. Participants were stratified across the three age groups and four BP categories and further classified into Normal Value Population (NVP) or Reference Value Population (RVP) groups based on the presence or absence of CVD risk factors. The NVP group had a mean age of 40 years, while the RVP group averaged 56.5 years, aligning reasonably with European reference data (39.7 vs. 33 years in NVP; 56.5 vs. 50 years in RVP) (The Reference Values for Arterial Stiffness' Collaboration 2010). This demographic comparability enhances the external validity of our findings.

The overall mean PWV of the study population was 5.88 ± 1.49 m/s. As expected, the RVP group had significantly higher mean PWV (6.74 m/s) than the NVP group (5.40 m/s), consistent with the established relationship between arterial stiffness and cardiovascular risk factors (Cecelja and Chowienczyk 2012; Ben-Shlomo et al. 2014). However, our mean PWV values were notably lower than those reported in European and international reference datasets, where elderly European participants typically show PWV values above 8.0 m/s (Koivistoinen et al. 2007; Alecu et al. 2008). Interestingly, our findings were more comparable to those from African and Arab populations (6.6–6.7 m/s) (Magalhães et al. 2013; Al-Hashmi et al. 2014), suggesting potential ethnic and methodological influences.

The lower PWV values observed may reflect differences in arterial anatomy or physiology across populations, the use of Doppler ultrasound (as opposed to other commercial

devices like SphygmoCor or PulsePen), and the study's healthy, treatment-naïve cohort (Alecu et al. 2008; Al-Hashmi et al. 2014). Additionally, the direct distance method (multiplied by 0.8) was intentionally chosen to align with the methodology used in the European reference study for PWV value (Upadhyay 2015), thereby enabling meaningful comparisons with existing normative data. Moreover, the literature notes that each distance definition yields different PWV values, contributing to variability and confusion in interpretation across studies (Anand et al. 2021; Dahiya et al. 2024). While the 12 m/s threshold cited in European Society guidelines corresponds to the full direct carotid–femoral distance, the more recent reference values are based on 80% of the direct carotid–femoral distance, which we applied to maintain consistency (Upadhyay 2015). While this approach enables comparability, we acknowledge that it may contribute to lower PWV values systematically.

Several cardiovascular risk factors correlated positively with PWV, including age, mean blood pressure (MBP), BP category and group classification (NVP vs. RVP). Consistent with the literature, age alone accounted for 48% of PWV variability, which improved to 54.5% when MBP was included. Seated BP measurements may also have influenced MBP estimates, as prior research shows that seated BP tends to yield slightly higher MBP values than supine measurements (Finucane et al., 2020), potentially affecting PWV outcomes.

Notably, sex was found to be positively correlated with PWV, though the effect size was small ($r = 0.17$, $p = 0.02$). As reported in prior studies, men typically present with higher PWV than women, particularly in younger age groups, likely due to structural and hormonal differences affecting arterial compliance. This difference diminishes post-menopause due to estrogen decline and vascular changes (Cecelja & Chowienczyk, 2009). The modest effect in our cohort may reflect age balancing or the influence of confounding variables like BP. This result should be interpreted cautiously since the study was not powered specifically to explore sex-based differences. Nonetheless, these findings support the inclusion of sex as a relevant, though modest, factor in vascular health assessment.

Although smoking is a well-established modifiable risk factor for CVD, its treatment in this study had inherent limitations. Smoking status was recorded as a binary variable (Yes/No), without distinguishing between current and ex-smokers. Consequently, individuals who had previously smoked but responded 'No' may have been misclassified as having no smoking-related risk, particularly in the NVP and vice-versa. This binary classification likely underrepresents the cumulative vascular impact of past smoking and may confound the interpretation of PWV values in the NVP group. Additionally, the statistical analysis considered smoking as a general risk factor across the entire cohort rather than exploring subgroup-specific effects between the NVP and RVP. We acknowledge this limitation as this approach may have obscured differences in PWV associated with smoking exposure within these subgroups.

Ethnic diversity in our sample, while reflective of Auckland's demographic composition, did not mirror the broader NZ population. Ethnic groups did not stratify the study population; participants were classified as either European (54.2% of the total population) or non-European, including Māori, Chinese, Indian and others from regions like Iran, Sri Lanka, and the Middle East. Auckland has a higher percentage of Pacific Islanders (14.6%) and Asians (23.1%), influencing the recruitment results. Since

ethnicity influences vascular properties, the observed mean PWV in the European subgroup (6.2 ± 1.5 m/s) being lower than reported values in the literature (8.7 ± 2.3 m/s) (Alecu et al. 2008) may reflect such population-specific differences.

Even though previous literature suggests that BP and age share a linear relationship with PWV, the findings in the present study have shown a more nuanced relationship. This anomaly could be attributed to the smaller sample size in this subgroup or to ethnic composition, where elevated BP participants were primarily of European and Asian descent. In younger participants (18–30 years), higher BP categories were associated with lower PWV, contrary to typical expectations. These observations suggest that in younger adults, particularly those of Asian or European ethnicity, the relationship between BP and PWV may not follow the same patterns observed in older populations.

Notwithstanding that MBP is a significant, positive predictor variable in linear regression, low PWV values at high BP range in the younger age group and generally lower PWV values at elevated BP compared with stage 1 and stage 2 hypertension in all age groups were study-specific findings. Because of these differences, BP categories were included in a regression model as dummy variables. Explaining 30.6% of the variation in mean PWV, stage 1 and stage 2 hypertension significantly increase mean PWV values compared with a normal BP.

Regression analysis showed that group classification (NVP vs. RVP), age, BMI and BP categories together explained 57% of the variance in mean PWV. The findings of our study were consistent with the literature regarding age and BP categories being significantly correlated. However, cardiovascular risk factors such as height and weight showed no significant correlation, whereas BMI and sex had a minimal effect. The differences observed in our study could be due to the exclusion of some clinical parameters, such as diabetes and dyslipidaemia, that are directly associated with high BMI scores at the time of diagnosis (Balkau et al. 2006; Tirosh et al. 2011). Although BP and age are traditionally viewed as having a linear relationship with PWV, our findings suggest this relationship may be more complex in younger populations or mixed ethnic groups. Dummy variable regression revealed that stage 1 and 2 hypertension contributed significantly to PWV increases compared to the normal BP category, accounting for 30.6% of the PWV variance.

Limitations and future work

This study represents a foundational step in establishing normative PWV values in a NZ population. However, the limited sample size and ethnic imbalance – particularly the underrepresentation of Māori and Pacific individuals – restrict generalizability. Exclusion of participants with diabetes, dyslipidaemia, or anti-hypertensive treatment also reduced ethnic diversity in the RVP group and may have biased results towards healthier individuals.

The observed low PWV values, even in older age groups, may reflect these selection criteria and the recruitment of a predominantly healthy population. For instance, the closest mean PWV value to international recommendations was found in the 60 + age group (7.08 m/s), still below the expected 10 m/s threshold. This suggests caution against universal PWV thresholds and underscores the need for population-specific reference values.

Future studies should include larger, more ethnically representative samples and participants with varying levels of cardiovascular risk. Incorporating individuals with diabetes, high cholesterol and treated hypertension would provide greater insight into PWV variation and enhance applicability to clinical practice. A nationwide, multicentre study stratified by ethnicity and cardiovascular risk levels could help establish robust normative PWV values and inform NZ-specific CVD prevention and management guidelines.

Conclusion

This study first estimates normal and reference aortic pulse wave velocity (PWV) values in a New Zealand population stratified by age and blood pressure categories. The findings confirm that age and mean blood pressure are the most consistent and significant predictors of PWV, while other factors such as sex, BMI and ethnicity showed more minor or inconsistent effects. Notably, the mean PWV values observed were generally lower than published international reference values, which may reflect differences in ethnicity, measurement methodology, or the healthy profile of the study population.

Despite limitations such as a modest sample size and underrepresentation of some ethnic groups, this study contributes valuable local data to inform cardiovascular risk assessment in New Zealand. The observed variability in PWV, particularly among younger individuals with elevated BP, underscores the importance of considering age, ethnicity and other contextual factors in vascular health evaluation. Future research with a larger, ethnically diverse cohort is warranted to refine normative PWV ranges and better inform clinical guidelines for cardiovascular risk management in the New Zealand context.

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No potential conflict of interest was reported by the author(s).

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