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# Utility of different blood pressure measurement components in childhood to predict adult carotid intima media thickness. The i3C Consortium Study.

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## Abstract

Childhood blood pressure levels predict adult subclinical atherosclerosis. However, the best childhood blood pressure component for prediction has not been determined. This study comprised 5,925 participants aged 3–18 years from six cohorts who were followed into adulthood (mean follow-up 25.8±6.2 years). Childhood blood pressure was measured by using a standard mercury sphygmomanometer in all cohorts. Study-specific carotid intima-media thickness ≥90$^{th}$ percentile was used to define subclinical atherosclerosis. Per standard deviation change in the predictor, childhood systolic blood pressure (age- and sex-adjusted Odds Ratio [95% confidence interval] 1.24[1.13–1.37]), mean arterial pressure (1.10[1.07–1.13]) and pulse pressure (1.15 [1.05–1.27]) were associated with increased adulthood intima-media thickness. In age-and sex-adjusted analyses, area under the receiver-operating characteristic curves for systolic blood pressure (c-value [95% confidence interval] 0.677[0.657–0.704]) showed significantly improved prediction compared to diastolic blood pressure (0.669[0.646–0.693], P=0.006), or mean arterial pressure (0.674[0.653–0.699] P=0.01). Pulse pressure provided a c-value that was not different from systolic blood pressure (0.676[0.653–0.699] P=0.16). Combining different blood pressure components did not improve prediction over systolic blood pressure measurement alone. Based on the associations with adult carotid intima-media thickness, cut-points for elevated systolic blood pressure were 105 mmHg for 3–6 year-old boys, 108 mmHg for 3–6 year-old girls, 108 mmHg for 7–12 year-old boys, 106 mmHg for 7–12 year-old girls, 123 mmHg for 13–18 year-old boys and 115 mmHg for 13–18 year-old girls. Our analyses suggest that several childhood blood pressure measurement components are related to adulthood carotid intima-media thickness. Of these, systolic blood pressure provided the best predictive ability.

### Keywords

blood pressure; atherosclerosis; intima-media thickness; epidemiology; pediatrics

## INTRODUCTION

Among adults, elevated carotid artery intima-media thickness (cIMT) is associated with cardiovascular disease (CVD) and stroke[1]. Data from the International Childhood Cardiovascular Cohort (i3C) Consortium have shown childhood blood pressure (BP) levels to associate with adult cIMT measured over 20 years later. Among the 4,210 participants from four cohort studies, elevated blood pressure (either systolic or diastolic ≥ 90$^{th}$ percentile for age, sex and height) that persisted from childhood into adulthood was associated with a nearly 2-fold higher risk of developing high cIMT compared to those who had normal BP levels at both time points[2].

BP is a well-established risk factor in childhood for future pre-clinical atherosclerosis, but uncertainty exists regarding the relative importance of various BP components in predicting risk[3,4]. Systolic BP (SBP) is influenced by cardiac contractility, while diastolic BP (DBP) by cardiac relaxation. Pulse pressure (PP), the difference between SBP and DBP, represents the force that the heart generates each time it contracts and may also reflect arterial stiffness[5].

Mean arterial pressure (MAP) represents an average arterial pressure during a single cardiac cycle and is estimated from SBP and DBP readings. In the Framingham Heart Study, combining PP with MAP, and SBP with DBP, produced models that were superior to single BP components for predicting subsequent CVD[3].

In this study, we used data from 5,925 individuals in six prospective cohort studies that have followed participants from childhood into adulthood. The objective of the study was to examine the utility of various BP components and their combinations in childhood for predicting future high cIMT.

## METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request.

### Study sample

Detailed study characteristics and examination methods for the six cohorts have been previously described[6–12]. Although loss to follow-up varied by cohort, previous analyses have suggested the representativeness of the cohorts has largely been maintained[6,8,13,14]. In total, 5,925 participants who had available blood pressure data from childhood (ages 3–18) and ultrasound data from adulthood (ages 19–51) were included in this study. Mean follow-up period was 25 years. Locally appointed ethics committees reviewed and approved the individual cohort studies that we analyzed, and participants in those studies (or their legal guardians) provided written informed consent. The present analysis conformed to the Declaration of Helsinki.

### Blood pressure measurements

BP at baseline was measured by using a standard mercury sphygmomanometer in all cohorts. Summary of the methods in different cohorts is shown in Supplemental Table S1. Pulse pressure (PP), was defined as follows: SBP-DBP. Mean arterial pressure (MAP) was defined as follows: (SBP+(DBP×2))/3. Diastolic IV was used to calculate PP IV and diastolic V to calculate PP V. Diastolic IV and V were used to calculate MAP IV and MAP V, respectively.

### Ultrasound measurements

B-mode ultrasound studies of the left carotid artery were performed at follow-up examinations using standardized protocols in each study. Details of the ultrasound data and protocols have been described elsewhere[6,12,15,16]. In the Young Finns Study (YFS), to assess intra-individual reproducibility of ultrasound measurements, 57 subjects were re-examined three months after the initial visit. The average absolute difference and standard deviation between measurements was 0.05±0.04 mm. In Bogalusa Heart Study (BHS), 75 participants underwent repeat ultrasound examinations 10–12 days after their initial visit to determine intra-individual reproducibility. The average absolute difference and standard deviation between measurements for all cIMT segments was 0.05±0.03 mm. In the Insulin Study, reproducibility of the cIMT showed a mean difference (±SD) of 0.02 ± 0.03 for analysis

separated by one week. In the Muscatine Study, a 4.4% random sample underwent repeat cIMT studies during a second visit, a mean of 107 days later, to assess intra-individual reproducibility. The mean absolute difference for all cIMT segments was 0.06 mm. In Childhood Determinants of Adult Health study (CDAH), intra-individual reproducibility for replicate maximum cIMT measurements was assessed in a random sample of 30 participants. The average absolute difference and standard deviation between measurements was 0.02±0.04 mm. In the Kaunas Study, 50 measurements of cIMT were assessed by a second investigator with between-observer coefficient of variation 4 %.

### Definition of high cIMT in adulthood

cIMT values are strongly related with age and sex[9]. In addition, methods to measure cIMT differed between the cohorts. Therefore, to take into account these issues, we first calculated age- and sex-specific percentile points for each individual's cIMT values separately in each cohort. In line with our prior reports, high cIMT in adulthood was then defined as ≥ 90$^{th}$ percentile based on these age-, sex- and study-specific percentile points[2]. In sensitivity analyses, similar results were found using cut-points corresponding to the 80$^{th}$ cIMT percentiles (data not shown).

### Statistical methods

The normality assumption of the residuals was assessed by examining histograms of the residuals and normal probability plots. The residuals were normally distributed. No significant interaction effects were observed between sex and blood pressure measures with continuous ultrasound variables, indicating that the associations of risk markers and ultrasound variables were similar between sexes. Additionally, due to differences in the range of risk factor levels among the cohorts, and changes reflecting secular trends, the analyses were performed by calculating age-, sex- and study-specific z-scores for each childhood blood pressure measurement and for adulthood high cIMT. Therefore, data from males and females were combined in all models. Differences between study groups were assessed by fitting ANOVA models. Age- and sex-adjusted logistic regression models were used to examine the associations for the blood pressure measurements with adult preclinical atherosclerosis (high cIMT). The ability of individual and combined blood pressure measures to predict high cIMT in adulthood was assessed using area under the receiver-operating characteristic curves (AUC), category-free net reclassification improvement (NRI) and integrated discrimination improvement (IDI). Calibration was assessed by using the Hosmer-Lemeshow (H-L$\chi$2) goodness of fit test. Differences in AUC between age- and sex-adjusted models were estimated with the use of the DeLong algorithm[17]. IDI represents an averaged measure with reduced variability and thus a more conservative significance level of $P \leq 0.01$ was used in the present study[18]. In addition, NRI is designed to quantify improvement in performance and hence its magnitude is more important than statistical significance[19]. Thus, caution in interpreting statistical significance levels for IDI and NRI is recommended. The optimal cut-points were defined from the ROC curve by calculating sensitivity and specificity and deriving the distance from the "perfect" point at the upper-left corner of the ROC plot where 1-Specificity=0 and Sensitivity=1. Optimal cutoff i.e. distance to (0,1) was defined by the equation: $\sqrt{(1\text{-Sensitivity})^2+(1\text{-Specificity})^2}$. Sensitivity describes the probability of correctly classifying the participant as having high cIMT (≥ 90th

percentile); specificity describes the probability of correctly classifying the participant as having normal cIMT (< 90th percentile). Except for the IDI analyses mentioned above, statistical significance was inferred as a *P* value of ≤0.05. Statistical analyses were performed with SAS 9.4.

## RESULTS

### Clinical characteristics

Table 1 shows clinical characteristics of the 5,925 study participants at baseline in childhood. Age, SBP, DBP, MAP and PP were different among study cohorts. No sex differences were observed between study cohorts. Absolute cIMT values at different percentiles from each cohort are shown in Supplemental Table S2.

### Association of childhood blood pressure components with high cIMT in adulthood

SBP, MAP IV and PP (IV and V) were associated with subsequent high cIMT. Age- and sex-adjusted results are shown in Table 2. When the models were further adjusted for childhood BMI, the associations remained statistically significant for SBP (OR for 1-SD change [95% CI] 1.17 [1.05–1.29], P-value 0.003), PP V (1.14 [1.04–1.25] P-value 0.004) and PP IV (1.11 [1.01–1.23] P-value 0.03) but not for MAP V (1.04 [0.94–1.14] P-value 0.50). Additionally, adjusting for length of follow-up, all results remained essentially similar (data not shown).

In age-stratified analyses (Supplemental Table S3) among participants who were 3–11 years-old at baseline, SBP (1.20 [1.01–1.41]), PP V (1.21 [1.04–1.41]) and PP IV (1.20 [1.02–1.40]) were associated with high cIMT in adulthood. Among participants who were 12–18 years-old, SBP (1.28 [1.14–1.44]), MAP V (1.14 [1.02–1.27]), MAP IV (1.19 [1.06–1.34]), PP V (1.17 [1.05–1.29]) and PP IV (1.17 [1.05–1.31]) were associated with high cIMT in adulthood.

### Differences in individual and combined blood pressure measurements in childhood predicting high cIMT in adulthood.

Table 3 shows c-statistics for individual blood pressure measures in predicting high cIMT compared to the c-statistic for SBP, which outperformed all individual blood pressure measures except for PP V and PP IV. Detailed analysis between SBP and PP V, and between SBP and PP IV showed no statistical differences in NRI-values (0.041, P-value 0.37; and 0.089, P-value 0.06, respectively) or in IDI-values (0.001, P-value 0.04; and 0.002, P=0.02, respectively). When compared to other main cardiovascular risk factors in childhood, the predictive ability of SBP did not significantly differ from that of BMI (AUCs (95% CI) 0.677 [0.657–0.704] vs. 0.681 [0.659–0.703], P=0.37). SBP provided better predictive value (0.677 [0.657–0.704]) compared to total cholesterol (0.668 [0.645–0.692], P for difference 0.02).

In age-stratified analyses (Supplemental Table S4) among participants who were 3–11 years-old no differences between blood pressure measures were detected in predicting cIMT in adulthood. Among participants who were 12–18 years-old, SBP outperformed DBP IV, DBP

V, MAP V and MAP IV ($P < 0.04$). No differences were observed between SBP and PP V and PP IV.

Next, we assessed the differences between individual and combined blood pressure measurements in models to predict high cIMT in adulthood (Table 4). No difference was observed between DBP IV and DBP V measures, or between SBP and SBP + DBP V. Similar results were observed when DBP IV was used instead of DBP V (data not shown). PP V and PP V + MAP V outperformed MAP V alone in predicting high cIMT. No difference was observed between MAP V + PP V and SBP + DBP V in predicting high cIMT. Similar results were obtained when MAP V was replaced with MAP IV, and PP V was replaced with PP IV (data not shown). No difference was observed between SBP and MAP V + PP V ($P$ always > 0.31 for AUC difference, IDI and NRI-values). Goodness-of-fit indicated by the Hosmer-Lemeshow $\chi 2$ was acceptable (always < 10) for all models examined.

We also examined the predictive value of childhood SBP for high adult cIMT stratified by study cohort (Figure 1). For YFS, AUC (95 % CI) was 0.740 (0.708–0.772), for BHS 0.649 (0.603–0.696), for CDAH 0.650 (0.593–0.701), for the Muscatine Study 0.648 (0.579–0.717), for the Insulin Study 0.574 (0.479–0.669) and for the Kaunas study 0.646 (0.568–0.723). Finally, we created cut-points for elevated systolic blood pressure values in different age and gender groups based on the associations with adult cIMT (Table 5).

## DISCUSSION

The findings from a large longitudinal six-cohort international study show that a single measurement of SBP during childhood/adolescence, was better, or an equally accurate alternative, to other BP components or their combinations in predicting the development of subclinical atherosclerosis, as represented by high cIMT, a mean of 25 years later. In addition, this study confirms the value of a simple measurement of SBP in the clinical setting.

Although it is known that BP in childhood is predictive of BP in adulthood, data relating BP levels in childhood to later cardiovascular events is currently lacking. In a cohort study of Swedish male conscripts BP at the age of 18 was associated with CVD mortality later in adulthood[20]. In addition, childhood BP has been associated with different markers of subclinical atherosclerosis, such as cIMT and arterial pulse-wave velocity[21,22]. The present study expands on these data by comparing the utility of different childhood BP components and their combinations in predicting future cIMT, a marker of subclinical atherosclerosis.

Questions remain regarding the utility of single versus combined BP measurement components in predicting cardiovascular health. Most pediatric patients with elevated BP are found to have isolated systolic hypertension[23]. Then with increasing age there is a gradual shift to DBP as the major predictor of risk, then to SBP and to PP. PP, an indicator of arterial stiffness[5], has been shown to be useful in predicting CVD events in the elderly[24]. A follow-up of participants in the Multiple Risk Factor Intervention Trial concluded that CVD risk assessment was improved in 35–57-year-old males by considering both SBP and DBP

jointly compared with SBP, DBP, or PP separately[25]. Results from the Framingham Heart Study confirmed the importance of combining BP components, such as SBP and DBP, or PP and MAP, to improve stratification of CVD risk in adult populations[3].

BP tracks from childhood into adulthood, with persons with elevated childhood BP having a higher probability of adult hypertension than those with normal BP[26]. In the present study, we found that prediction of cIMT was significantly better with SBP than with DBP. This is consistent with results from the YFS and CDAH studies showing that child-to-adult tracking correlations were higher for SBP compared with DBP[26–28]. Measurement of DBP is complicated by the presence of two diastolic Korotkoff phases (fourth phase and fifth phase) in many children and adults. The recommendations for the assessment of DBP among children have varied. Previously, the fourth Korotkoff phase (muffling of sound) was a recommended method in assessing DBP[4]. Although the fourth Korotkoff phase has been shown to have less inter-observer variability and a stronger correlation with adult hypertension[28], the most recent recommendations (since the 1996 Working Group report[29]) have supported the use of the fifth Korotkoff phase (disappearance of sound) for all children and adolescents[30]. The present study supports the present recommendations by showing no significant differences between the fourth and fifth Korotkoff phases in predicting future cIMT.

Concerning clinical practice implications of the present study, we provided cut-points for elevated childhood blood pressure levels in different age and sex groups, based on the prediction of adult cIMT (Table 5). Sensitivity (42–80%) and specificity (27–73%) were not very high for these childhood cut-offs. However, they are comparable with NCEP and NHANES cut-points for elevated LDL-cholesterol in childhood predicting subsequent high cIMT[31].

The main strength of this study is the large data base from all six studies that included similar lifestyle and biological risk factors in childhood and followed the cohorts into adulthood. The study also has certain limitations. First, there were no replicate measures that might have modified our results, with BP measurements taken only at a single time point in childhood and in adulthood. However, in the YFS, single childhood measurements were nearly as informative as repeated measurements in determining associations between childhood and adult measurements[6]. Additionally, when individual SBP and DBP were studied, SBP was found to be associated with cIMT, but the correlations between DBP and cIMT in adulthood were weaker, and a higher number of DBP measurements did not improve prediction of cIMT[32]. In the present study, BP was measured 2 – 6 times during one visit. It has been reported that BP tracking was slightly higher with multiple BP measurements per visit, but the tracking correlations were not improved with more than two measurements[28]. Second, because the study cohorts are comprised of young adults at follow-up, we are not able to study associations between risk factors and definite cardiovascular events. Instead, we have used cIMT as a surrogate end-point. Therefore, the risk stratification groupings are not based on absolute risk of CVD events (as in adult risk score systems), but on high cIMT ($\geq 90^{th}$ percentile). We observed that childhood BP levels predict cIMT in young adulthood. In older adults cIMT has been shown to predict subsequent CVD events[1], but there is paucity of knowledge concerning the predictive utility

of young adulthood cIMT measurements for future CVD with the exception of data from the Carotid Atherosclerosis Progression Study indicating an equivalent or higher relative risk of a combined end-point (myocardial infarction, stroke, or death) among younger (<50 years) vs. older (≥50 years) adults[32].

Our data, based on six large population-based prospective cohorts, confirm that youth with elevated SBP are at increased risk of adult high cIMT measured, on average, 25-years later. Moreover, prediction using SBP alone is at least equivalent to any other individual or combined BP measurement, suggesting that in the pediatric setting, the prediction of future subclinical atherosclerosis can be best achieved utilizing childhood SBP measurements.

### Perspectives

This finding is clinically important because prehypertension and hypertension are substantially under diagnosed in the pediatric setting. Additionally, uncertainty exists regarding the relative importance of various blood pressure components in predicting risk. The findings from the present study address these important areas and confirm the value of a simple measurement of systolic blood pressure in the clinical setting.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## ACKNOWLEDGEMENTS

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## NOVELTY AND SIGNIFICANCE

### What is new?

In the present international, multicenter study, we assessed and compared the predictive ability of childhood blood pressure components (systolic, diastolic, mean arterial and pulse pressure) in predicting carotid intima-media thickness in adulthood. In addition, we provided age- and gender-specific cut-points for clinical practice.

### What is relevant?

We found that children with elevated systolic blood pressure are at increased risk of adult subclinical atherosclerosis 25-years later. Moreover, prediction using systolic blood pressure alone is at least equivalent to any other individual or combined blood pressure measurements.

### Summary

Systolic blood pressure alone in childhood could provide a simple way to predict future subclinical atherosclerosis.

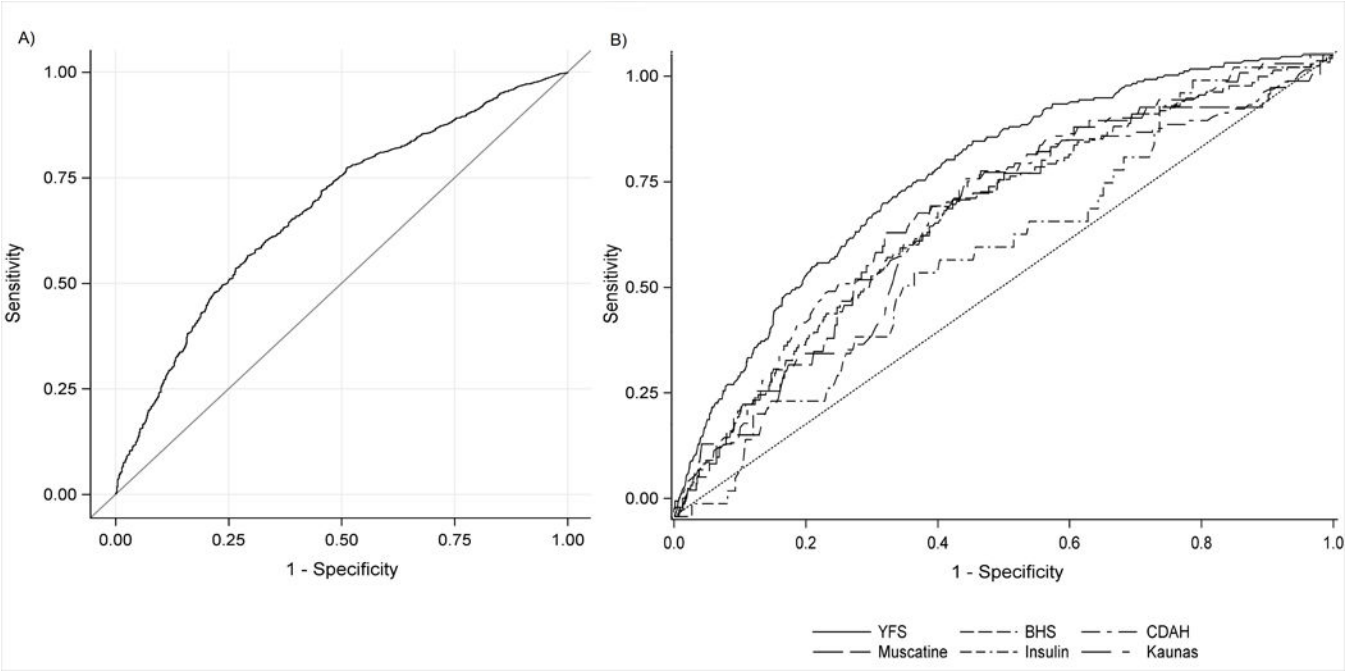


**Figure 1.**

Receiver operating characteristic curves for childhood systolic blood pressure in predicting adult high carotid intima-media thickness, A) combined in all cohorts, B) by cohort (YFS: Young Finns study, BHS: Bogalusa Heart study, CDAH: Childhood Determinants of Adult Health study).

**Table 1.**

Baseline characteristics in the six study cohorts

| Variable | YFS (N=2,554) Mean±SD | BHS (N=1,300) Mean±SD | CDAH (N=695) Mean±SD | Muscatine (N=721) Mean±SD | Insulin (N=294) Mean±SD | Kaunas (N=361) Mean±SD | P-value† | All (N=5,925) |
|---|---|---|---|---|---|---|---|---|
| Age (years) | 11±4 | 10±3 | 13±2 | 13±3 | 14±2 | 13±0.3 | <0.0001 | 12±4 |
| Sex (male %) | 54 | 57 | 54 | 52 | 47 | 54 | 0.44 | 54 |
| Systolic blood pressure (mmHg) | 112±11 | 100±10 | 109±13 | 115±14 | 108±9 | 114±12 | <0.0001 | 109±13 |
| Diastolic IV blood pressure (mmHg) | 75±10 | 62±8 | 78±12 | 75±9.3 | - | - | <0.0001 | 72±11 |
| Diastolic V blood pressure (mmHg) | 68±10 | 45±12 | 66±12 | 68±10 | 56±13 | 55±11 | <0.0001 | 62±15 |
| Mean arterial pressure-IV (mmHg) | 88±9 | 75±8 | 81±10 | 89±10 | - | - | <0.0001 | |
| Mean arterial pressure-V (mmHg) | 83±9 | 63±10 | 81±10 | 83±10 | 73±10 | 75±9 | <0.0001 | 76±12 |
| Pulse pressure-IV (mmHg) | 38±11 | 55±13 | 43±13 | 40±11 | - | - | 0.0003 | |
| Pulse pressure-V (mmHg) | 45±11 | 54±13 | 42±13 | 48±13 | 51±13 | 59±14 | <0.0001 | 48±13 |
| Body mass index (kg/m$^2$) | 17.8±3.0 | 17.7±3.6 | 18.6±2.9 | 19.8±3.5 | 22.7±5.1 | 18.9±3.1 | <0.0001 | 18.4±3.6 |

*In the Insulin- and Kaunas studies diastolic IV blood pressure was not measured.

†Group comparisons among study cohorts

**Table 2.**

Associations of childhood blood pressure measures (age-, sex-, and study specific) with high cIMT in adulthood (age-, sex-, and study-specific ≥ 90$^{th}$ percentile).

| Blood pressure measurement | OR (95% CI) | P-value |
|---|---|---|
| Systolic blood pressure | 1.24 (1.13–1.37) | <0.0001 |
| Diastolic IV | 1.07 (0.97–1.17) | 0.16 |
| Diastolic V | 1.01 (0.92–1.10) | 0.88 |
| MAP IV * | 1.10 (1.07–1.13) | 0.006 |
| MAP V † | 1.08 (0.98–1.19) | 0.11 |
| PP IV * | 1.15 (1.05–1.27) | 0.0027 |
| PP V † | 1.11 (1.08–1.13) | 0.0004 |

Age- and sex-adjusted univariate logistic regression models were performed for each blood pressure measure.

Odds Ratios (OR) and 95 % Confidence Intervals (CI) are for a 1-SD increase

* Diastolic IV was used in the MAP IV and PP IV models

† Diastolic V was used in the MAP V and PP V models

**Table 3.**

Utility of single measures of childhood blood pressure (age-, sex- and study-specific) to predict high adult cIMT.

| Blood pressure measurement | AUC (95% CI) | P-value for difference |
|---|---|---|
| Systolic blood pressure | 0.677 (0.657–0.704) | REFERENCE |
| Diastolic IV | 0.670 (0.647–0.694) | 0.004 |
| Diastolic V | 0.669 (0.646–0.693) | 0.006 |
| MAP IV * | 0.674 (0.653–0.699) | 0.01 |
| MAP V † | 0.672 (0.648–0.648) | 0.003 |
| PP IV * | 0.676 (0.653–0.699) | 0.16 |
| PP V † | 0.673 (0.651–0.694) | 0.21 |

* Diastolic IV was used in the MAP IV and PP IV models

† Diastolic V was used in the MAP V and PP V models

**Table 4.**

Utility of different blood pressure measurement components and their combinations in childhood to predict adult high cIMT (age-, sex- and study-specific ≥ 90$^{th}$ percentile).

| **Method for prediction** | **Diastolic V** | **Diastolic IV** | **P for difference** |
|---|---|---|---|
| AUC (95%CI) | 0.669 (0.646–0.693) | 0.670 (0.647–0.694) | 0.37 |
| IDI | | 0.0012 | 0.002 |
| NRI | | 0.05 | 0.25 |
| | Systolic | Systolic + Diastolic V | P for difference |
| AUC (95%CI) | 0.677 (0.657–0.704) | 0.678 (0.655–0.700) | 0.32 |
| IDI | | 0.0001 | 0.78 |
| NRI | | 0.007 | 0.12 |
| | Systolic | Systolic + PP V* | P for difference |
| AUC (95%CI) | 0.677 (0.657–0.704) | 0.678 (0.655–0.700) | 0.40 |
| IDI | | 0.0001 | 0.70 |
| NRI | | 0.072 | 0.10 |
| | PP V* | PP V* + MAP V* | P for difference |
| AUC (95%CI) | 0.673 (0.651–0.694) | 0.677 (0.655–0.699) | 0.09 |
| IDI | | 0.002 | 0.005 |
| NRI | | 0.066 | 0.15 |
| | MAP V* | PP V* + MAP V* | P for difference |
| AUC (95%CI) | 0.667 (0.645–0.689) | 0.677 (0.655–0.699) | 0.005 |
| IDI | | 0.035 | 0.001 |
| NRI | | 0.15 | 0.011 |
| | PP V* + MAP V* | Systolic + Diastolic V | P for difference |
| AUC (95%CI) | 0.677 (0.655–0.699) | 0.678 (0.656–0.700) | 0.25 |
| IDI | | 0.0003 | 0.23 |
| NRI | | 0.014 | 0.76 |

* Diastolic V was used in the MAP V and PP V models

AUC: Area under the curve, IDI: integrated discrimination index, NRI: net reclassification index; MAP: mean arterial pressure, PP: pulse pressure, cIMT: carotid intima-media thickness

**Table 5.**

Optimal cut-points for childhood systolic blood pressure predicting high cIMT in adulthood, including sensitivity and specificity.

| Sex | Age (y) | N | Optimal cutoff for systolic BP* (mmHg) | Sensitivity (%) | Specificity (%) |
|---|---|---|---|---|---|
| Males | | | | | |
| | 3–6 | 657 | 105 | 53 | 50 |
| | 7–12 | 1,152 | 108 | 45 | 66 |
| | 13–18 | 888 | 120 | 53 | 56 |
| Females | | | | | |
| | 3–6 | 833 | 108 | 52 | 60 |
| | 7–12 | 1,371 | 106 | 50 | 50 |
| | 13–18 | 1,024 | 115 | 38 | 63 |

*Optimal distance to (0,1) was defined by the equation: $\sqrt{(1\text{-Sensitivity})^2+(1\text{-Specificity})^2}$

BP: blood pressure.