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Central Blood Pressure and Augmentation Index in Older Adults With Intellectual Disabilities

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Keywords: ageing | augmentation index | cardiovascular risk | central blood pressure | down syndrome | intellectual disability

ABSTRACT

Background: Older adults with intellectual disabilities are at a higher cardiovascular risk than their peers in the general population. Investigating central blood pressure and augmentation index is necessary to better understand the risk of cardiovascular disease, to better identify those individuals at risk and to potentially change pharmacological treatment regimens. We therefore aim to investigate central blood pressure and augmentation index in two large cohorts (total $N = 237$) of older adults with intellectual disabilities, across different age ranges and sexes. Additionally, we will explore the cross-sectional relationships of central blood pressure and augmentation index with other cardiovascular risk factors and the presence of cardiovascular disease across a broad age range.

Method: Collected data of two cohorts of older adults with intellectual disabilities were included: $n = 121$ individuals with intellectual disabilities of ≥ 60 years from the Healthy Ageing and Intellectual Disabilities (HA-ID) study, and $n = 115$ individuals with intellectual disabilities ≥ 40 years from The Intellectual Disability Supplement to the Irish Longitudinal Study on Ageing (IDS-TILDA) study. The Mobil-O-Graph was used to measure central blood pressure and augmentation index. The distribution of haemodynamic measures across different sex and age groups was reported, and bivariate correlations were calculated to explore associations between haemodynamic measures, cardiovascular risk factors and history of CVD.

Results: Mean brachial pressures for the HA-ID cohort (mean age 71 ± 6 years) was 133/81 mmHg. The slightly younger IDS-TILDA cohort (mean age 60 ± 9 years) had a median brachial blood pressure of 127/81. Mean central SBP (cSBP) in the older HA-ID cohort was 122 mmHg versus 120 mmHg in the younger IDS-TILDA cohort, with a central DBP (cDBP) of 82 mmHg

in both cohorts, and a central pulse pressure (cPP, cSBP-cDBP) of 40 mmHg for the HA-ID cohort and 38 mmHg for the IDS-TILDA cohort.

Conclusions: Females with intellectual disabilities had higher central blood pressures, augmentation pressure and augmentation index than males, and females showed an age-related increase in central blood pressures.

1 | Introduction

Older adults with intellectual disabilities are at a higher cardiovascular risk than their peers in the general population. Similar to the general population, prevalence rates of cardiovascular disease (CVD) in older adults with intellectual disabilities vary greatly and are largely based on electronic health records, which tend to underestimate prevalence particularly in this population due to underdiagnosis (Jansen et al. 2013; Haveman et al. 2011). Higher prevalence rates in older adults with intellectual disabilities have been found for heart failure (de Winter et al. 2016; de Leeuw et al. 2023; Pemmasani and Yandrapalli 2021), cerebrovascular disease (van den Bemd et al. 2022; Cooper 1998) and stroke (de Leeuw et al. 2023; Hussain et al. 2020). CVD risk factors have been studied extensively in this population, both similar risk factors to the general population, as well as population-specific risk factors. High prevalence rates have been shown for metabolic syndrome (de Winter et al. 2011), being overweight or obese (de Winter et al. 2012b; Flygare Wallen et al. 2018; O'Brien et al. 2021; O'Brien et al. 2023), being physically inactive (Hilgenkamp et al. 2012; O'Brien et al. 2021), and for hypertension, diabetes and hypercholesterolaemia (de Winter et al. 2012a; Flygare Wallen et al. 2018; van den Bemd et al. 2022; O'Brien et al. 2021). Population-specific risk factors such as the frequent use of atypical antipsychotics (Newcomer 2007) and the presence of specific genetic syndromes (Nordstrøm et al. 2016) may further increase the cardiovascular risk as they age. A longitudinal study investigated risk factors for CVD and mortality 3 years after baseline in adults with intellectual disabilities of 50 years and over (de Winter et al. 2016), finding that age, hypertension, obesity, chronic kidney disease and a history of coronary artery disease, stroke or heart failure were significant risk factors (de Winter et al. 2016). Based on this high risk for CVD and the broad range of risk factors, it is important to prioritise monitoring and early detection in this population using accurate yet feasible methods or strategies.

In the Global Burden of Disease Report from 2020, hypertension was identified as the leading risk factor for mortality and disability worldwide (Global Burden of Disease Risk Factors Collaborators 2020). Decades of randomised controlled trials demonstrate that lowering blood pressure reduces the risk of cardiovascular disease and events substantially (Lewington et al. 2002; Turnbull and Blood Pressure Lowering Treatment Trialists 2003), which makes this a critical modifiable target for interventions. Systolic blood pressure and pulse pressure (arterial pulsatility) typically increase with age and are higher in overweight or obese individuals (Hall et al. 2015). In turn, hypertension causes damage to vulnerable vascular beds like the brain, the heart and kidneys, and results in structural remodelling of the heart and vascular system, increasing the risk of coronary artery disease, stroke and heart failure (Turnbull and Blood Pressure Lowering Treatment Trialists 2003). In a previous study on risk factors for mortality in older adults with intellectual disability, all significant risk factors were related to hypertension, yet hypertension did not fully explain cardiovascular risk on its own (de Winter et al. 2016). This may partially be explained by using brachial blood pressure as the method of measurement.

Recent reports have shown that central (or aortic) blood pressure measurements are more strongly related to cardiovascular outcomes than the traditional brachial blood pressure measurement (Roman et al. 2007; Sharman et al. 2017; Cheng et al. 2022). Arterial pressure varies considerably across the arterial tree, with vital organs like the heart exposed to central pressure rather than to brachial pressure (Kostapanos et al. 2016; Battistoni et al. 2020). As individuals age or develop disease, their arteries stiffen, leading to a faster propagation of the forward pulse waves, but also faster reflected waves going back to the heart. This results in augmented aortic pressure, which increases the work the heart must perform for every stroke, subsequently causing remodelling of the heart and increasing stiffness of the arteries over time (Townsend et al. 2016). Increased amplitude of the wave reflection and an increased late systolic load have been shown to cause myocardial hypertrophy (Westerhof and O'Rourke 1995), and systolic and diastolic myocardial dysfunction (Kawaguchi et al. 2003; Weber et al. 2008; Canepa et al. 2014). Measures of central blood pressure and the augmentation index (Aix) capture this increased load and are, therefore, more strongly related to cardiovascular disease risk (McEniery et al. 2014; Chirinos et al. 2012; Vlachopoulos et al. 2010) and all-cause 15-year mortality (Wang et al. 2010). Additionally, antihypertensive medication has a differential effect on central versus brachial blood pressure, underscoring the need for accurate detection of individuals at risk for damage to end organs based on central blood pressure (Kostapanos et al. 2016; McEniery et al. 2014; Cheng et al. 2013).

Central blood pressure and augmentation index have not yet been explored in older adults with intellectual disabilities, despite evidence suggesting damage to the vascular tree in this population (de Winter et al. 2013). Investigating central blood pressure and augmentation index is necessary to better understand the risk of cardiovascular disease, to better identify those individuals at risk, and to potentially change pharmacological treatment regimens based on this new information. As a first step, we therefore aim to investigate central blood pressure and augmentation index in two large cohorts (total $N = 237$) of older individuals with intellectual disabilities, each with different age ranges and explore the cross-sectional relationships of central blood pressure and augmentation index with other cardiovascular risk factors and the presence of cardiovascular disease across a broad age range.

2 | Methods

2.1 | Design and Participants

This study was a retrospective cross-sectional secondary analysis, including two separate cohorts of older adults with intellectual disabilities. The first cohort from the Healthy Ageing and Intellectual Disabilities (HA-ID) study (Erasmus MC, University Medical Center Rotterdam, the Netherlands) consisted of 121 individuals with intellectual disabilities of 60 years and over. The data were collected between October 2020 and December July 2023, as part of the 10-year follow-up data collection wave, which for the first time included the vascular outcomes described in this paper. Further details on design, recruitment and data collection are published elsewhere (de Leeuw et al. 2022). The second cohort from The Intellectual Disability Supplement to the Irish Longitudinal Study on Ageing (IDS-TILDA) study (Trinity College Dublin, Ireland) consisted of 115 individuals with intellectual disabilities of 40 years and over. The data included in this paper were collected between September 2019 and March 2020, as part of Wave 4 of IDS-TILDA, the closest available timepoint for comparison with HA-ID. Further details on design, recruitment and data collection have previously been reported (Burke et al. 2020; McCarron et al. 2022). Participants were eligible for inclusion in Wave 4 of IDS-TILDA if they completed self or informant outcome measures at interview and objective measures at health assessment as detailed below. The final samples from both cohorts only included participants with a good quality Mobil-O-Graph measurement, as determined by scoring 1 (*very good*) or 2 (*good*) on the signal quality variable from the Mobil-O-Graph software. Due to different institutional and national data sharing policies, the raw data of the two studies could not be combined and were therefore analysed in parallel for this paper.

2.2 | Ethics

Ethical approval was granted by the ethics committees of both universities and from each of the participating service providers (HA-ID: MEC-2019-0562, IDS-TILDA: 181210). Accessible, easy-read material with full explanation was provided to ensure informed consent. For eligible participants who could understand the study information and were capable of providing informed consent themselves, informed consent was obtained directly from them. If participants were not capable of providing informed consent, informed consent was obtained from their legal representative.

2.3 | Outcome Measures

2.3.1 | Haemodynamic Outcome Measures Including Central Blood Pressure and Augmentation Index

The Mobil-O-Graph was used to measure central blood pressure and augmentation index (Wassertheurer et al. 2010). The Mobil-O-Graph is a non-invasive, oscillometric blood pressure device that estimates central blood pressure from brachial blood pressure by using the ARCSolver algorithm as the generalised transfer function (Weiss et al. 2012). Comparison with either direct intra-arterial pressure measurement (Nakagomi et al. 2017; Gotzmann et al. 2020) or with other validated devices commonly used for the noninvasive estimation of central blood pressure (Sphygmocor or Complior) has demonstrated the validity of the Mobil-O-Graph for measuring aortic pressure in normotensive and hypertensive middle-aged and older adults as well as patients with coronary artery disease (Weiss et al. 2012; Weber et al. 2011; Gotzmann et al. 2020). The accuracy of the estimation is greatly enhanced by calibrating the aortic wave form with the mean and diastolic peripheral pressure (C2 calibration) versus using systolic and diastolic peripheral pressure (C1 calibration), and by measuring the peripheral mean arterial pressure instead of calculating this based on diastolic and/or systolic pressure values (Nakagomi et al. 2017). The Mobil-O-Graph measures brachial systolic, diastolic and mean pressure directly and calculates the following haemodynamic outcome measures:

I. Brachial pulse pressure (PP) was calculated from peripheral systolic and diastolic BP as PP = systolic BP − diastolic BP.

II. Central (aortic) systolic and diastolic blood pressures (cSBP and cDBP) were calculated from the peripheral (brachial) blood pressure wave recordings by a generalised transfer function with the ARCSolver software (Austrian Institute of Technology, Vienna, Austria) (Wassertheurer et al. 2010).

III. Central Pulse Pressure (cPP) was calculated as cPP = cSBP–cDBP.

IV. Augmentation pressure (AP) was calculated as the difference between the late (P2) and early (P1) systolic peaks of the aortic waveform: $AP = P_2 - P_1$ (Chirinos et al. 2005).

V. AIx, a measure of global wave reflections, was defined as the percentage of AP to central PP: AIx = ([P2 − P1]/cPP*100) (Chirinos et al. 2005). Due to the influence of heart rate on AIx, it was also normalised to a heart rate of 75 bpm (AIx@75).

VI. Pf and Pb refer to the amplitude of the forward and reflected (backward) travelling pressure waves in the arterial system. The forward waves are the result of the heart's pumping action, while the reflected waves are the waves that travel back to the heart at points of arterial resistance. With stiffer arteries, the reflected waves arrive at the heart earlier, increasing the pressure the heart needs to work against (Nichols et al. 2008; Nichols and Edwards 2001) (Weber et al. 2011) (Walser et al. 2023).

2.3.2 | Other Cardiovascular Risk Factors

In addition to blood pressure, other cardiovascular risk factors included in this study were: sex, age, level of intellectual disability (borderline/mild, moderate and severe/profound), Down syndrome (yes/no/unknown), living arrangements (residential/24-h care, community-based living and with family/independent), mobility (independent, walking aids and wheelchair), smoker (yes/no), use of atypical antipsychotics (yes/no) and cardiovascular medication (anti-hypertensives, diuretics,

beta blockers, calcium channel blockers, ACE inhibitors, lipid-modifying agents, diabetes medications, yes/no), presence of diabetes mellitus type 2 (yes/no), hypertension (yes/no), hypercholesteremia (yes/no) and chronic kidney disease (yes/no). Past or present cardiovascular disease (CVD), from here on are referred to as history of CVD (including coronary artery disease, stroke, heart failure [de Winter et al. 2016], revascularisation, arrhythmia, angina pectoris, aneurysm aortae, peripheral arterial disease, thrombosis, myocardial infarction, TIA, pacemaker), was extracted from the medical files.

Objective measures obtained through health assessment have been previously described (de Leeuw et al. 2022), and included weight, height, waist circumference, hip circumference, BMI and physical fitness (2 min step test). Physical activity was measured using the International Physical Activity Questionnaire (IPAQ) and categorised as inactive, minimally active, 'Health Enhancing Physical Activity' active according to the instrument's instructions (Lee et al. 2011). Total Cholesterol, HDL, LDL and triglycerides were measured using blood sample (HA-ID) and blood drop (IDS-TILDA).

2.3.3 | Statistical Analyses

Both cohorts were analysed separately. Descriptive statistics were used to report the distribution of haemodynamic measures including central blood pressure and augmentation index, across different sex and age groups. Normality was checked with Kolmogorov–Smirnov test and Shapiro Wilk test. Outcomes are presented as means with standard deviations if normally distributed, or as medians with interquartile range (IQR) if not normally distributed. Effects of sex, age and the interaction effect of sex*age was calculated with two-way independent ANOVAs.

Next, bivariate correlations were calculated to explore associations between haemodynamic measures including central blood pressure and augmentation index outcomes and cardiovascular risk factors, as well as with history of CVD. Pearson correlation coefficient was used for normally distributed, continuous variables, Spearman correlation coefficient was used for non-normally distributed continuous variables or ordinal variables, point-biserial correlation coefficient was used for the correlation between categorical and dichotomous variables, Phi coefficient was used for correlation between two binary variables, and for variables with multiple nominal categories (living circumstances), we used an ANOVA.

For each one of the haemodynamic variables individually, exploratory multivariate logistic regression analyses were performed with history of CVD as the dependent variable. Sex, age and level of intellectual disability, together with other cardiovascular risk factors that demonstrated significant bivariate correlations with CVD were entered to the model, along with one of the haemodynamic variables (cSBP, cDBP, cPP, AP or Aix@75) in each individual model. In case of independent variables with significant correlations between each other (such as weight, BMI and/or waist circumference) we only included the variable with the strongest correlation with history of CVD after controlling for sex, age and level of ID.

3 | Results

3.1 | Demographic Profile and Prevalence of Cardiovascular Risk Factors and Disease

The demographic profiles of the samples from the Netherlands (HA-ID: $N=121$) and Ireland (IDS-TILDA; $N=115$) are presented in Table 1. The mean age of participants was 70.81 years (SD = 5.74) in the HA-ID cohort and 59.81 years (SD = 8.94) in IDS-TILDA cohort. Females constituted slightly over half of the sample in both cohorts. Most participants resided in community group homes or residential care facilities. Most participants had mild or moderate intellectual disabilities. Regarding cardiovascular risk factors, the mean BMI was in the overweight category (BMI between 25 and 29.9). A large proportion of participants were inactive or minimally active.

Approximately one quarter of HA-ID participants had hypercholesteraemia and one quarter had hypertension. Additionally, 10.7% had type 2 diabetes and no participant had chronic kidney disease. In the IDS-TILDA cohort, one third had hypercholesteraemia (and one fifth had hypertension). Furthermore, 8% had type 2 diabetes and 3.5% had chronic kidney disease. A minority of participants were smokers in both groups.

The most commonly reported medication used in both cohorts was lipid modifying agents, but when we combined all five classes of medications used in the treatment of hypertension (antihypertensives, diuretics, beta blockers, calcium channel blockers and ACE inhibitors), hypertension medication was used even more frequently.

3.2 | Haemodynamic Measures Including Central Blood Pressure and Augmentation Index

Table 2 presents the average or median haemodynamic measures for both cohorts, including central blood pressures, augmentation pressure, augmentation index and forward and reflected wave separation data. Table 3 and Figures 1–4 present cSBP, cDBP, AP and Aix@75 in both cohorts by age and sex.

In the HA-ID cohort, females had significantly higher cSBP ($p=0.008$), AP ($p=0.023$) and Aix@75 ($p<0.001$) than males, while there was a significant sex–age effect for cSBP ($p=0.030$) and cDBP ($p=0.027$), driven mostly by higher pressures in females in the 80–89 years category. The effect of age was non-significant for all four outcome variables.

In the IDS-TILDA cohort, no sex or age main or interaction effects were observed for any of the haemodynamic variables ($p>0.05$).

3.3 | Cross-Sectional Relationships

Table 4 presents the bivariate correlation coefficients between potential cardiovascular risk factors and presence of cardiovascular disease with the haemodynamic variables. For the HA-ID cohort, the haemodynamic variables with a significant positive correlation with female sex were cPP, AP and Aix@75, whereas age was only correlated with cPP. Level of ID was negatively

TABLE 1 | Descriptives participants with a valid Mobil-O-Graph measurement.

	HA-ID ($N=121$)	IDS-TILDA ($N=115$)
Sex		
Females, *n* (%)	61 (50.4%)	67 (58.3%)
Males, *n* (%)	60 (49.6%)	48 (41.7%)
Age (mean, SD, range)	Mean: 70.8, SD: 5.7	Mean: 59.8, SD: 8.9
40–49 years (*n*, %)	0 (0%)	12 (10.4%)
50–59 years (*n*, %)	0 (0%)	42 (36.5%)
60–69 years (*n*, %)	59 (48.8%)	45 (39.1%)
70–79 years (*n*, %)	50 (41.3%)	14 (12.2%)
80–89 years (*n*, %)	12 (9.9%)	2 (1.7%)
Level of intellectual disability		
Borderline/mild (*n*, %)	32 (26.4%)	25 (21.7%)
Moderate (*n*, %)	67 (55.4%)	60 (52.2%)
Severe/profound (*n*, %)	21 (17.3%)	20 (17.4%)
Unknown/missing (*n*, %)	1 (0.8%)	10 (8.7%)
Down syndrome		
Yes (*n*, %)	3 (2.5%)	19 (16.5%)
No (*n*, %)	115 (95.0%)	96 (83.5%)
Unknown/missing (*n*, %)	3 (2.5%)	
Living circumstances/setting		
Residential/24 h care (*n*, %)	56 (46.3%)	44 (38.2%)
Community-based living (*n*, %)	61 (50.4%)	51 (44.3%)
With family/independent (*n*, %)	1 (0.8%)	18 (15.7%)
Unknown/missing (*n*, %)	3 (2.5%)	2 (1.7%)
Mobility/level of support		
Independent (*n*, %)	45 (37.2%)	78 (67.8%)
Walking aids (*n*, %)	29 (24.0%)	15 (13%)
Wheelchair (*n*, %)	9 (7.4%)	21 (18.3%)
Unknown/missing (*n*, %)	13 (10.7%)	1 (0.9%)
Body mass index (mean, SD)	27.6 (5.1)	29.0 (6.1)
Missing (*n*, %)	$n=7$	$n=3$
Waist circumference, cm (mean, SD)	97.0 (11.9)	103.7 (13.1)
Missing (*n*, %)	$n=8$	$n=31$
Hip circumference, cm (mean, SD)	102.6 (13.1)	107.44 (11.8)
Missing (*n*, %)	$n=18$	$n=31$
Smoking		
Yes (*n*, %)	12 (9.9%)	3 (2.6%)
No (*n*, %)	58 (47.9%)	110 (95.7%)
Missing (*n*, %)	51 (42.1%)	$n=2$ (1.7%)

(Continues)

TABLE 1 | (Continued)

	HA-ID ($N=121$)	IDS-TILDA ($N=115$)
Physical activity level (IPAQ categories)		
Inactive (*n*, %)	35 (28.9%)	72 (62.6%)
Minimally active (*n*, %)	27 (22.3%)	35 (30.4%)
HEPA active (*n*, %)	7 (5.8%)	8 (7%)
Missing (*n*, %)	52 (43.0%)	
Physical fitness (2 min step test) (mean, SD)	42.2 (21.6)	66.3 (34.5)
Missing (*n*, %)	50 (41.3%)	69 (60%)
Medications with ATC code	$N=103$	$N=113$
Atypical antipsychotics (*n*, %)	6 (5.8%)	38 (33.6%)
Lipid-modifying agents, that is, statins (C10) (*n*, %)	33 (32.0%)	41 (36.3%)
Diabetes medications (A10) (*n*, %)	5 (4.9%)	9 (7.8%)
Treatment for hypertension		
Any antihypertensive medication (*n*, %)	27 (22.3%)	33 (28.7%)
Antihypertensives (C02) (*n*, %)	0 (0%)	0 (0)
Diuretics (C03) (*n*, %)	12 (11.7%)	13 (11.5%)
Beta blockers (C07) (*n*, %)	8 (7.8%)	8 (7.1%)
Calcium channel blockers (C08) (*n*, %)	8 (7.8%)	9 (8%)
ACE inhibitors (C09) (*n*, %)	16 (15.5%)	15 (13.3%)
Diagnosis in medical records		$N=113$
Diabetes mellitus type 2 (*n*, %)	13 (10.7%)	9 (8.0%)
Hypercholesterolaemia (*n*, %)	31 (25.6%)	39 (33.9%)
Hypertension (*n*, %)	30 (24.8%)	24 (20.9%)
Chronic kidney disease (*n*, %)	0 (0.0%)	4 (3.5%)
Lipid levels available (*n*, %)	98 (80.2%)	67 (62%)
Total cholesterol (mean, SD)	4.68 (1.14)	4.46 (0.80) ($n=55$)
High-density lipoprotein (mean, SD)	1.31 (0.38)	1.36 (0.34) ($n=56$)
Low-density lipoprotein (mean, SD)	Mean: 2.69, SD: 0.93	Mean: 2.30, SD: 0.67 ($n=54$)
Triglycerides (mean, SD)	Not available	Mean: 1.78, SD: 0.97 ($n=55$)
History of cardiovascular disease (*n*, %)	28 (23.1%)	12 (10.4%)
Coronary artery disease	N/A	N/A
Stroke	9 (7.4%)	1 (0.86%)
Heart failure	6 (5.0%)	1 (0.86%)
Revascularisation	3 (2.5%)	0 (0.0%)
Arrhythmia	7 (5.8%)	8 (7.9%)
Angina pectoris	3 (2.5%)	0 (0.0%)
Aneurysm aortae	0 (0.0%)	0 (0.0%)
Peripheral arterial disease	2 (1.7%)	0 (0.0%)
Thrombosis	3 (2.5%)	0 (0.0%)

(Continues)

TABLE 1 | (Continued)

	HA-ID ($N=121$)	IDS-TILDA ($N=115$)
Myocardial infarction	4 (3.3%)	1 (0.86%)
Transient ischaemic attack (TIA)	4 (3.3%)	3 (2.6%)
Pacemaker	1 (0.8%)	0 (0.0%)
Missing	12 (9.9%)	0 (0.0%)

Abbreviations: HA-ID: Healthy Ageing and Intellectual Disability; IDS-TILDA: Intellectual Disability Supplement to the Irish Longitudinal Study on Ageing; SD: standard deviation.

TABLE 2 | Hemodynamic outcomes form the Mobil-O-Graph measurement for participants with a valid Mobil-O-Graph measurement.

	HA-ID ($N=121$) Mean (SD)[a]	IDS-TILDA ($N=115$) Mean (SD)
Brachial SBP (mmHg)	133.3 (21.6)	127 (114–134)[a,b]
Brachial DBP (mmHg)	80.7 (12.0)	80.9 (12.2)[b]
Brachial MAP (mmHg)	104.8 (15.0)	102 (91–110)[a,b]
Brachial PP (mmHg)	52.6 (16.8)	45.7 (12.9)[b]
Heart rate (bpm)	74.0 (65.0–85.5)[a]	71.8 (12.6)[b]
Central SBP (mmHg)	122.4 (20.2)	119.9 (17.2)
Central DBP (mmHg)	82.4 (12.2)	80 (72–89)[a]
Central PP (mmHg)	38.0 (30.0–49.0)[a]	38.3 (12.4)
Augmentation Pressure (AP)	10.0 (4.5–17.0)[a]	10 (7–16)[a] $n=80$
Augmentation index (AIx)	25.3 (11.2)	22.5 (13.0–36.0)[a] $n=64$
Augmentation index normalised for HR (AIx@75)	28.0 (18.0–35.0)[a]	23.9 (11.9)[b]
Pf	32.6 (26.3–38.6)[a]	28.6 (23.4–33.4)[a,b]
Pb	20.20 (14.9–25.7)[a]	18.1 (14.8–22.4)[a,b]

Abbreviations: DBP: diastolic blood pressure; HA-ID: Healthy Ageing and Intellectual Disability; HR: heart rate; IDS-TILDA: Intellectual Disability Supplement to the Irish Longitudinal Study on Ageing; MAP: mean arterial pressure; Pb: backward pressure wave amplitude; Pf: forward pressure wave amplitude; PP: pulse pressure; SBP: systolic blood pressure; SD: standard deviation.
[a]In case of non-normally distributed outcomes, median and interquartile range are reported.
[b]IDS-TILDA data available for $n=98$.

correlated with all haemodynamic outcomes except Aix@75. Having Down syndrome was negatively correlated with cSBP and cDBP, meaning that individuals with Down syndrome had lower cSBP and cDBP than individuals without DS. Other cardiovascular risk factors that correlated with one or more haemodynamic variables were hypertension (cSBP, Aix@75), total cholesterol (cSBP, cDBP), LDL (cDBP), physical fitness (cDBP), smoking (AiX@75), physical activity level (AiX@75) and height (AiX@75). Although several cardiovascular risk factors correlated with the history of cardiovascular disease (age, level of intellectual disability, weight, BMI, waist circumference, statins use, diabetes, hypertension, anti-hypertensive medication, total cholesterol, high-density lipoprotein and low-density lipoprotein), none of the haemodynamic variables correlated with the history of cardiovascular disease.

For the IDS-TILDA cohort, significant correlations were observed between haemodynamic variables and demographic factors (Table 4). Central pulse pressure (cPP) and AP were significantly and positively associated with female sex, while AP was also significantly associated with age. The presence of Down syndrome was negatively correlated with cSBP and cDBP. Among the risk factors, hypertension exhibited significant positive correlations with cSBP, cPP, Aix@75% and significant negative correlations with AP.

Additionally, triglyceride levels were significantly correlated with Aix@75. With the exception of age and low-density lipoproteins, no significant correlations were found between the history of CVD and any other cardiovascular risk factors or haemodynamic variables.

Based on the outcomes of the bivariate correlations for HA-ID, the individual multivariate logistic regression analyses for each one of the central haemodynamic variables, with history of CVD as the dependent variable, included sex, age and level of intellectual disability, waist circumference, statins use, diabetes, anti-hypertensive medication and LDL plus one of the

TABLE 3 | Mobil-O-Graph outcomes by sex and age.

	HA-ID (*N* = 121) Mean (SD)		IDS-TILDA (*N* = 103) Mean (SD)	
	Male	**Female**	**Male**	**Female**
Central SBP (mmHg) total	*N* = 60	*N* = 61	*N* = 40	*N* = 63
40–49 years	NA	NA	111.6 (10.3) *n* = 5	118. 7 (14.9) *n* = 6
50–59 years	NA	NA	120.2 (17.0) *n* = 17	121.8 (16.044) *n* = 20
60–69 years	121.5 (17.21) *n* = 28	120.6 (20.0) *n* = 31	118.7 (18.8) *n* = 13	120.0 (19.5) *n* = 28
70–79 years	120.1 (18.4) *n* = 27	125.3 (21.0) *n* = 23	119.8 (14.6) *n* = 4	123.2 (18.4) *n* = 9
80–89 years	109.6 (11.1) *n* = 5	142.9 (31.6) *n* = 7	94.00 (NA) *n* = 1	*n* = 0
Central DBP (mmHg) total	*N* = 60	*N* = 61	*N* = 40	*N* = 63
40–49 years	NA	NA	77.0 (7.5) *n* = 5	81.0 (10.0) *n* = 6
50–59 years	NA	NA	86.4 (9. 8) *n* = 17	83.2 (15.7) *n* = 20
60–69 years	84.0 (11.2) *n* = 28	81.6 (11.9) *n* = 31	82.0 (10.3) *n* = 13	79.6 (12.7) *n* = 28
70–79 years	83.2 (12.0) *n* = 27	81.3 (13.6) *n* = 23	86.0 (3.9) *n* = 4	79.2 (8.8) *n* = 9
80–89 years	71.0 (8.2) *n* = 5	89.0 (12.2) *n* = 7	66.0 (NA) *n* = 1	*n* = 0
Augmentation pressure (AP) total	*N* = 60	*N* = 61	*N* = 27	*N* = 46[b]
40–49 years	NA	NA	7.4 (3.1) *n* = 5	11.4 (6.4) *n* = 5
50–59 years	NA	NA	8.3 (3.4) *n* = 12	12.4 (5.0) *n* = 14
60–69 years	9.8 (8.19) *n* = 28	11.5 (10.2) *n* = 31	11.0 (6.9) *n* = 6	14.9 (8.4) *n* = 20
70–79 years	9.4 (8.44) *n* = 27	14.4 (10.6) *n* = 23	13.7 (7.8) *n* = 3	14.3 (8.0) *n* = 7
80–89 years	11.8 (7.73) *n* = 5	21.6 (20.4) *n* = 7	3.0 (NA) *n* = 1	*n* = 0
Augmentation index normalised for HR (AIx@75) total[a]	*N* = 60	*N* = 61	*N* = 31	*N* = 56[a]
40–49 years	NA	NA	19.80 (7.50) *n* = 5	22.20 (14.91) *n* = 5
50–59 years	NA	NA	21.38 (10.93) *n* = 13	24.23 (9.07) *n* = 17
60–69 years	21.86 (11.32) *n* = 28	28.10 (10.90) *n* = 31	23.62 (13.23) *n* = 8	24.42 (14.49) *n* = 26
70–79 years	20.22 (13.22) *n* = 27	31.04 (11.65) *n* = 23	21.75 (15.11) *n* = 4	31.5 (10.49) *n* = 8
80–89 years	21.20 (2.95) *n* = 5	36.29 (5.79) *n* = 7	15.00 (NA) *n* = 1	*n* = 0

Abbreviations: DBP: diastolic blood pressure; HA-ID: Healthy Ageing and Intellectual Disability; HR: heart rate; IDS-TILDA: Intellectual Disability Supplement to the Irish Longitudinal Study on Ageing; MAP: mean arterial pressure; PP: pulse pressure; SBP: systolic blood pressure; SD: standard deviation.
[a]IDS-TILDA data available for *n* = 87.
[b]IDS-TILDA data available for *n* = 73.

haemodynamic variables (cSBP, cDBP, cPP, AP or Aix@75) as independent variables (Table S1). Hypertension, weight, BMI, total cholesterol and HDL were not included due to significant correlations with the already included independent variables. These analyses showed that waist circumference, statins use and LDL were most often significant predictors for history of CVD and that the haemodynamic variable did not contribute to explaining the history of cardiovascular risk for HA-ID.

Based on the outcomes of the bivariate correlations for IDS-TILDA, the individual multivariate logistic regression analyses for each one of the haemodynamic variables, with history of CVD as the dependent variable, included sex, age and level of intellectual disability, LDL and anti-hypertensive medication plus one of the five haemodynamic variables (cSBP, cDBP, cPP, AP or Aix@75) as independent variables (Table S2). However, unlike in the HA-ID analysis, none of the variables were statistically significant contributors to the regression analyses.

4 | Discussion

This is the first study to report on central blood pressure and augmentation index in older adults with ID. The analyses of

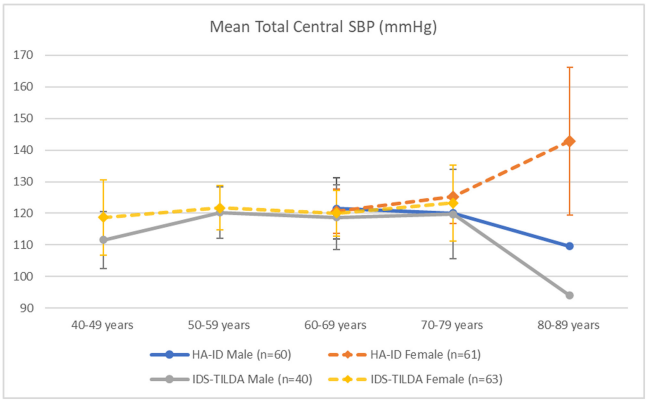

FIGURE 1 | Mean central systolic blood pressure by age and sex.

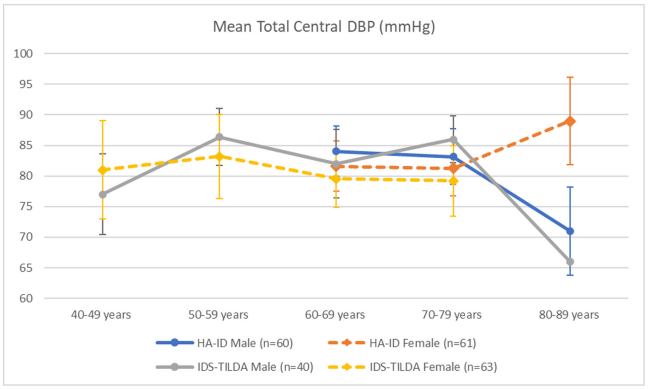

FIGURE 2 | Mean central diastolic blood pressure by age and sex.

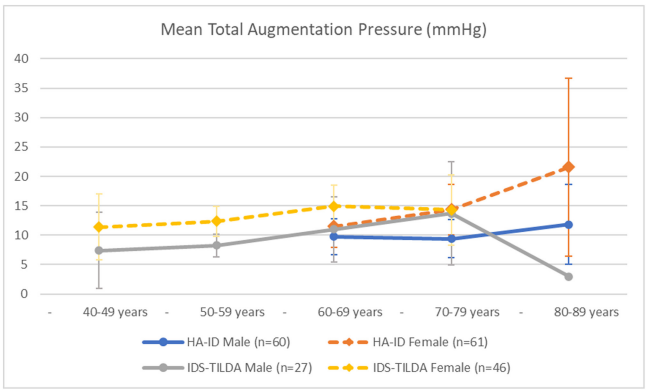

FIGURE 3 | Mean total augmentation pressure by age and sex.

these two well-characterised cohorts, the HA-ID cohort starting at 60 years and the IDS-TILDA cohort starting at 40 years, allow for a better understanding of these outcome measures across a broad age range, as well as differences between men and women. In the older HA-ID cohort, we saw differences between men and women, with women having higher blood pressures and higher augmentation index, and with only women demonstrating an age effect for cSBP and cDBP.

Compared to a worldwide 'reference cohort' ($n = 27\,253$), a population characterised by either having hypertension and/or other cardiovascular risk factors, the cSBP values by age and

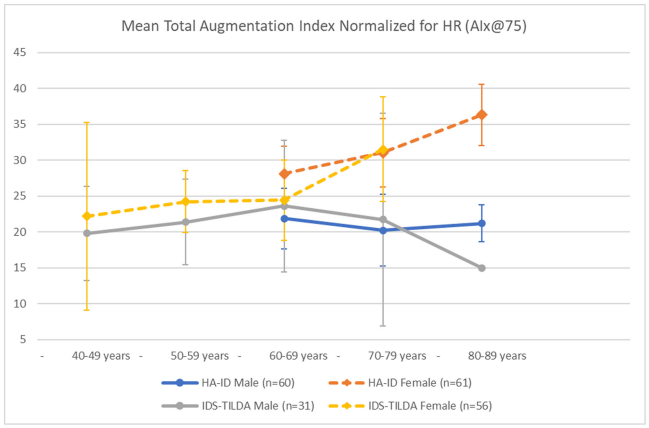

FIGURE 4 | Mean total AiX@75 by age and sex.

sex categories for adults with intellectual disabilities in our study were quite similar to peers without intellectual disabilities (Herbert et al. 2014). Large studies, such as the Framingham Heart Study, have shown that with age, blood pressure (mean arterial pressure [MAP], SBP and DBP) increases up to the age of 50 years. This increase is attributed to an increase in peripheral vascular resistance. However, after the age of 50, DBP tends to decrease, resulting in a rise in PP or arterial pulsatility, due to increased large artery stiffness (Franklin et al. 1997). With regard to sex, women have indeed been shown to have lower blood pressures (both peripheral and central) than men up to their 60 s, after which the gap narrows and then reverses, likely due to stiffening of the large arteries after menopause (Franklin et al. 1997) (McEniery et al. 2005). This finding is extremely relevant considering women have increased CVD mortality risk at lower levels of brachial SBP than men (Elfassy et al. 2023). Our results show that adults of 40 years and up (IDS-TILDA cohort) do not show a gap in blood pressure between sexes, and that women after 60 years of age (HA-ID cohort) demonstrate higher central blood pressure as well.

With regards to Aix, a large normotensive and hypertensive cohort presented with 23.8% (SD = 10.9%) and 32.7% (SD = 13.4%), whereas our cohorts presented with 25.3% (SD = 11.2%) (HA-ID) and 22.5% (IQR 13%–36%) (IDS-TILDA) (Obayashi et al. 2019; Obayashi et al. 2021). These results indicate that our cohorts have slightly higher AP and Aix than the normotensive cohort, but not as high as the hypertensive cohort, which is in line with the unselected nature of our cohorts.

Augmentation pressure and Aix@75 showed a similar impact of sex as cSBP. There were higher AP and Aix@75 for women compared to men, but no interaction effects for sex*age were observed for AP and Aix@75. This aligns with the results of the Anglo-Cardiff Collaborative Trial and other studies, in which AP and Aix were higher in women compared with men regardless of age and hypertension status (McEniery et al. 2005; Chang et al. 2023; Eikas et al. 2023; Costa-Hong et al. 2018). Additionally, the lack of an age effect in our older cohorts aligns with other studies in which changes in Aix are more prominent in younger individuals (< 50 years), whereas subjects older than 50 years typically exhibit more evident changes in aortic PWV, due to different proposed physiological changes (McEniery et al. 2005). The finding from that same

TABLE 4 | Correlation coefficients between haemodynamic outcomes, risk factors for CVD and history of CVD.

	cSBP		cDBP		cPP		AP		Aix@75		History of CVD	
	HA-ID	IDS-TILDA	HA-ID	IDS-TILDA	HA-ID	IDS-TILDA	HA-ID	IDS-TILDA	HA-ID	IDS-TILDA	HA-ID	IDS-TILDA
Sex[b]	0.125	0.084	−0.009	−0.100	**0.190***	**−0.210***	**0.191***	**0.326****	**0.375****	0.145	0.0.063[e]	0.059[d]
Age	0.152	0.033	−0.028	−0.112	**0.245****	0.154	0.170	**0.222***	0.143	0.107	**0.216***	**0.333[d]***
Level of ID	**−0.269**[a]**	−0.061	**−0.220**[a]**	0.062	**−0.236**[a]**	−0.139	**−0.186**[a]**	−0.145	−0.154[a]	−0.040	***p*=0.015**[d]**	0.112[d]
Down syndrome[b]	**−0.215***	**−0.278****	**−0.261****	**−0.316****	0.355	−0.080	−0.025	−0.132	−0.001	−0.170	−0.107[e]	−0.075[d]
Living circumstances[c]	*p*=0.219	*p*=0.785	*p*=0.068	*p*=0.525	*p*=0.736	*p*=957	*p*=0.431	*p*=397	*p*=0.791	*p*=0.952	*p*=0.242[d]	0.072[d]
Level of support	0.040	−0.171	−0.011	−0.184	0.067	−0.063	−0.020	−0.039	0.114	0.078	***p*=0.028***[d]	0.047[d]
Height	0.019	0.040	0.092	0.101	−0.054	−0.040	−0.153	−0.238	**−0.197***	−0.095	0.046	−0.095
Weight	0.107	0.171	0.078	0.129	0.089	0.118	−0.121	−0.169	−0.027	−0.072	**0.283****	−0.028
BMI	0.106	0.133	0.045	0.040	0.115	0.148	−0.038	−0.018	0.090	−0.026	**0.307****	0.015
Waist circumference	0.023	0.212	−0.029	0.086	0.060	0.200	−0.081	0.001	−0.011	−0.008	**0.292****	−0.154
Smoking[b]	−0.118	−0.030	−0.031	−0.062	−0.140	0.018	−0.103	−0.020	**−0.251***	0.027	0.150[e]	−0.054[d]
Physical activity level	−0.072	0.068	−0.088	0.054	−0.031	0.042	−0.163	0.059	**−0.251***	0.007	−0.108	0.110[d]
Physical fitness (2MST)	0.202	−0.079	**0.320****	0.163	0.784	−0.286	−0.021	−0.223	−0.049	−0.052	0.020	0.074
Statins use[b]	−0.071	0.004	−0.051	−0.040	−0.058	0.045	−0.046	**0.237***	−0.046	0.053	**0.336 **[e]**	0.162[d]
Diabetes[b]	0.011	−0.054	−0.091	0.025	0.093	−0.101	0.101	−0.068	0.158	−0.048	**0.302**[e]**	−0.097[d]
Hypercholesterolaemia[b]	0.174	−0.158	0.137	−0.078	0.135	−0.144	−0.027	**−0.233***	−0.027	−0.182	0.206[e]	−0.176[d]
Hypertension[b]	**0.204***	**0.330****	0.151	−0.170	0.164	**0.294****	0.086	**−0.317****	**0.218***	**0.230***	**0.300 **[e]**	−0.105[d]
Anti-hypertensive medication[b]	**0.192***	0.072	0.143	0.133	0.154	−0.029	0.088	−0.103	0.155	0.014	**0.272**[e]**	0.287[d]**
Total cholesterol	**0.210***	−0.097	**0.256***	0.034	0.082	−0.147	0.106	−0.128	0.113	−0.072	**−0.355****	−0.244
High-density lipoprotein	0.141[a]	−0.020	0.178[a]	−0.200	0.100[a]	0.152	**0.236***[a]	0.022	0.188[a]	−0.205	**−0.297**[a]**	0.121
Low-density lipoprotein	0.152[a]	−0.109	**0.253***[a]	0.035	−0.025[a]	−0.159	−0.020[a]	−0.177	0.094[a]	−0.134	**−0.373***[a]	**−0.309***
Triglycerides	NA	−0.047	NA	0.153	NA	−0.190	NA	−0.017	NA	0.329*	NA	−0.093
History of CVD[b]	−0.020[b]	0.117	−0.032[b]	−0.001	−0.002[b]	0.163	−0.030[b]	0.152	-0.030[b]	−0.019	NA	NA

Note: Results are presented as Pearson correlation coefficients unless otherwise indicated.
Abbreviations: 2MST: two-minute step test; Aix@75: Augmentation Index normalized for HR; AP: augmentation pressure; BMI: body mass index; cDBP: central diastolic blood pressure; cPP: central pulse pressure; cSBP: central systolic blood pressure; CVD: cardiovascular disease; HA-ID: Healthy Ageing and Intellectual Disability; ID: intellectual disability; IDS-TILDA: Intellectual Disability Supplement to the Irish Longitudinal Study on Ageing; NA: Not available for HA-ID.
[a]Spearman correlation coefficient (combination continuous and non-normally distributed variables).
[b]Point-biserial correlation coefficient (combination continuous–binary variables).
[c]ANOVA test, reporting *p*-values (combination continuous–categorical variables).
[d]Chi-square test, reporting *p*-values (combination categorical variables).
[e]Phi coefficient (combination binary-binary variables).
*Correlation significant at the 0.05 level.
**Correlation significant at the 0.01 level.

study that PWV was not different between sexes, suggests that the higher Aix in women is not caused by differences in large artery stiffness per se, but rather due to differences in wave reflection, possibly related to their shorter average height (McEniery et al. 2005). This is supported by our data, which shows a significant inverse correlation between height and Aix@75 in the HA-ID cohort.

Correlations between cardiovascular risk factors, the presence of CVD and haemodynamic outcomes were different between the cohorts, likely due to age-related differences in the prevalence of those variables. Correlation coefficients for sex and age were only significant with cPP, AP and Aix@75, which is not in line with previous research that reported significant correlations between all haemodynamic outcomes (Obayashi et al. 2021). Previous research has also shown positive correlations between measures of body composition and central BP and/or augmentation index (Bittencourt et al. 2023; Chen et al. 2023; Chang et al. 2023; Harbin et al. 2018), but these correlations were not found in the HA-ID or IDS-TILDA cohorts. With regards to lifestyle behaviours, non-smoking and higher levels of physical activity were inversely correlated with Aix@75 in the HA-ID cohort, but in contrast with other studies, no correlation was found with fitness and Aix@75 (Grosser et al. 2024; Hamasaki and Yanai 2023). With regards to Down syndrome, both cohorts showed that having Down syndrome was correlated with lower cSBP and cDBP, in line with previous research, but surprisingly, this did not result in lower cPP, lower AP, lower Aix@75 or lower history of CVD. None of the haemodynamic variables contributed significantly to explaining history of CVD in the HA-ID cohort, contrasting other large epidemiological research that found that haemodynamic variables were able to predict cardiovascular disease risk (McEniery et al. 2014; Chirinos et al. 2012; Vlachopoulos et al. 2010) and all-cause 15-year mortality (Wang et al. 2010). However, the sample size for the exploratory regression analyses was small, and a larger sample size would have potentially had more power to support other contributing predictors of CVD.

This study expands the current literature by, for the first time, describing a fairly new population of ageing adults with intellectual disabilities. Additionally, individuals with intellectual disabilities have been mostly excluded from large-scale studies in the general population, highlighting the need for comprehensive characterisation of health concerns in this population, beyond electronic health records studies that suffer from underdiagnosis of health issues (de Leeuw et al. 2024; de Winter et al. 2012a). Objective measurements are therefore critical when screening for cardiovascular health in this population. Observing similar sex and age effects in ageing adults with intellectual disabilities as in the general population underscores the need for health promotion and cardiovascular health screening, especially for women after 60 years of age. Haemodynamic variables (mostly Aix@75) seem promising in supporting the detection of individuals at risk for CVD, although further research in larger samples starting at an even younger age is necessary to identify trends from middle-aged and up.

Strengths of this study include the large sample of older adults with ID from two cohorts differing in age, which allows for an in-depth understanding of the progression of these outcomes during the aging process in this population. Other strengths are the standardisation of measurements through using the same device, trained testers, and a controlled data collection environment. Another strength is the use of the Mobil-O-Graph, which employs a validated generalised transfer function to calculate aortic pulse waves.

The determination of central blood pressures, augmentation pressure and augmentation index has been thoroughly validated with invasive measurements and can therefore be validly used for comparison between individuals and groups. This study has some weaknesses as well. Although the parallel analysis of the datasets provides novel insights, combining the datasets would have likely resulted in additional power for the multivariate analyses; however, this was not possible due to the restrictions imposed by different institutional and national data sharing policies. Given the heterogeneity of the study population and the complex and dynamic environment of individuals with intellectual disabilities, data collection of large-scale studies in this population is particularly prone to missing data, especially for the more involved measurements, and this could potentially limit the generalisability of our findings. Although missing data limited some statistical options, these two study cohorts remain the largest and most comprehensively characterised study samples to date in this population, enabling unique analyses. Additionally, even with these two large cohorts, the breakdown by age and sex still resulted in some very small sample sizes for the older age groups, possibly skewing those outcomes. Finally, the cross-sectional nature of this data is not a suitable design for the predictive ability of the haemodynamic variables for the presence of CVD, but we are looking forward to performing this with longitudinal data in the future as these measurements will be included in next waves of data collection.

Different from the general population, we observed age-related increases in Aix@75 throughout all the age categories up to 80–89 years. In the general population, the increase in Aix@75 until around 60 years has been attributed to an increase in the magnitude of the wave reflection, whereas the stiffening of the large arteries after 60 years is more visible in increases in aortic pulse wave velocity (McEniery et al. 2005). It is unclear how these physiological processes of wave reflection and arterial stiffening influence vascular aging in adults with ID and their risk of CVD, as some previous research does seem to support this relationship (O'Brien et al. 2023). It is recommended to include validated measurements of carotid-femoral pulse wave velocity and arterial stiffness in future studies to elucidate this.

5 | Conclusion

Based on two large cohorts of older adults with ID, central blood pressures, augmentation pressure and augmentation index confirm a similar cardiovascular risk in this population compared to cohorts in the general population with known cardiovascular risk factors and cardiovascular disease. Females had higher central blood pressures, augmentation pressure and augmentation index than males, and females showed an additional age-related increase in central blood pressure. Longitudinal research is

needed to further investigate the predictive ability of central blood pressure and augmentation index to determine the contribution of large artery stiffening to cardiovascular risk in older adults with ID.

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Ethics Statement

Ethical approval was granted by the ethics committees of both universities and from each of the participating service providers (HA-ID: MEC-2019-0562, IDS-TILDA: 181210).

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

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

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.