Research Paper

Early-life determinants of cardiometabolic outcomes and accelerated biological ageing in Colombia

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ABSTRACT

Background: Adverse childhood experiences (ACEs) are critical early-life determinants of long-term health, yet their association with biological ageing and cardiometabolic risk remains poorly understood. We examined the association between ACEs, cardiometabolic outcomes and age acceleration among older adults in Colombia.
Methods: Data were drawn from 3,385 adults aged ≥60 years (1,726 women, 1,659 men) from the nationally representative Health, Well-Being, and Ageing Study (SABE-Colombia). Five ACEs before 15 years old were assessed: emotional abuse, domestic violence, poor self-reported health, scarcity of food, and forced childhood migration due to armed conflict. Biological ageing was estimated using Klemera-Doubal Method for Biological Age (ΔKDMAge). Associations between ACEs and cardiometabolic outcomes (cardiovascular disease [CVD], diabetes, hypertension, and obesity) were evaluated using logistic and Poisson regression models; and associations with biological ageing using linear regression models, adjusting for sociodemographic factors.
Results: Among women, emotional abuse (OR=1.68), domestic violence (OR=1.55), scarcity of food (OR=1.44), and poor health status (OR=1.66) were associated with increased odds of CVD (OR=1.68). Among men, forced childhood migration was associated with higher risks of diabetes (OR=1.60), CVD (OR=1.55), and hypertension (OR=1.43). Forced childhood migration was also associated with age acceleration (ΔKDMAge β =1.52), with a stronger association in women (ΔKDMAge β =2.67). Dose-response associations were observed between cumulative ACEs and CVD in women and hypertension in men.
Conclusions: Early-life adversity, particularly forced childhood migration, is associated with higher cardiometabolic risk and accelerated biological ageing in later life, emphasizing the long-term biological costs of social and political instability.

INTRODUCTION

Adverse Childhood Experiences (ACEs) have been linked to cardiometabolic outcomes and other health outcomes across the life course [1, 2]. Research has suggested an increased risk of cardiovascular disease [1, 3, 4], diabetes [5, 6], and obesity [5, 7, 8] among individuals exposed to one or more ACEs compared to non-exposed individuals. In previous studies exposure to emotional abuse or maltreatment during childhood doubled the likelihood of developing cardiometabolic diseases in adulthood [9–13]. Several

meta-analyses have supported an association between single ACEs and health outcomes [7, 14, 15], and a dose-dependent relationship between cumulative ACEs and multimorbidity [16], violence, mental illness, and substance use [17]. In addition, exposures to ACEs are not equally distributed across genders. Prior research suggests that women are more likely to experience childhood adversity and have poorer health outcomes compared to men if they are exposed [1, 4]. However, one study in the UK found no gender differences [18].

The relationship between ACEs and later health outcomes can be understood through intermediate biological and psychosocial mechanisms. Chronic early-life stress may induce physiological dysregulation, including inflammation, hormonal imbalance, and accelerated biological aging [1, 19, 20]. These health mechanisms can be captured through markers of biological aging, which reflect the speed at which an individual's biological age progresses relative to their chronological age [21, 22].

Intermediate biological mechanisms include stress-related dysregulation of neuroendocrine and inflammatory systems such as the hypothalamic–pituitary–adrenal (HPA) axis activation and chronic low-grade inflammation [23, 24]. People age at different rates due to a combination of genetic, environmental, and psychosocial factors, and understanding these differences remains a central challenge in longevity science. Integrating these mechanistic insights offers a theoretical framework to explain how early adversity "gets under the skin," clarifying causal pathways and informing interventions aimed at mitigating long-term health risks [25]. A key objective in geroscience is to understand why and how we age, as biological age may better reflect an individual's overall physiological state than chrono-logical age [26, 27].

Life-course epidemiology demonstrates that early-life adversity is associated with long-term dysregulation of stress-response systems, including persistent activation of the HPA axis and elevated inflammatory burden [1, 19, 28]. HPA dysregulation links ACEs to accelerated aging through chronic cortisol elevation, increased allostatic load, and downstream metabolic disturbances [24]. ACE exposure is also associated with biomarkers of accelerated aging, including epigenetic age acceleration [14], shortened telomere length [29], and increased allostatic load [30, 31, 32]. Accelerated biological aging, in turn, is associated with higher risks of cardiometabolic disease, multimorbidity, functional decline, and mortality [21, 27, 32]. Although our cross-sectional study cannot formally test mediation pathways, these mechanisms provide conceptual framework for interpreting the observed associations.

Colombia is a particularly relevant country for the study of long-term consequence of ACEs, since a violent civil war occurred between 1964 and 2015 (Conflicto armado interno de Colombia), with more than 200,000 deaths [33]. We investigated the association between ACEs, including early forced migration related to the conflict, and cardiometabolic outcomes and biological age acceleration in a national sample of the Colombian population. The sample was recruited in 2015 and the average age was 69 years; therefore, a substantial proportion of participants had been exposed to the civil war during childhood and adolescence and a fraction of them migrated for this reason.

RESULTS

Study population and characteristics

Participants' characteristics stratified by gender are presented in Table 1. In both samples (overall and biomarkers subsample), study participants were on average 69.1 (S.D. 7.05) years old and 56% were women. Most of the participants were Mestizo (42-44%), followed by White participants (27-29%). The study population had a high prevalence of low childhood socioeconomic position (73-79%), indicating an extensive experience of early life disadvantage within this generation.

ACEs were also prevalent. Only 28.6% of the entire sample reported experiencing no ACEs, meaning over 70% had faced at least one significant adversity during childhood. The most commonly reported ACEs included scarcity of food (25-28%), poor childhood health (11-12%), and domestic violence (16-19%). A gender difference emerged in the experience of domestic violence, with women consistently reporting higher prevalence (19.2% overall sample; 18.4% in the biomarker subsample) compared to men (15% overall sample; 15.9% biomarker subsample). While other ACEs showed minor fluctuations between sexes, this particular difference was pronounced. The cumulative burden of ACEs also highlighted the widespread nature of these experiences, with a considerable proportion reporting multiple ACEs: two ACEs (16-20%), three ACEs (8-11%) and four or more ACEs (2-4%). Participants reporting more than one ACE were more likely to be women and non-white ($p<0.001$). Women more frequently reported emotional abuse and domestic violence compared to males. Men were more likely to report poor health status and scarcity of food. By ethnic groups, Indigenous and Afro-Colombian women had higher ACEs scores than White women and men.

Table 1. Baseline characteristics, ACEs, and cardiometabolic outcomes of the study participants by gender (SABE Colombia-2015 overall sample n= 18,044 and biomarker subsample n=3,385).

Characteristics/ Categories	Overall sample			Subsample biomarkers		
	Total	Women	Men	Total	Women	Men
N	18,044	10,101	7,943	3,385	2,026	1,359
Sociodemographic characteristics						
Mean chronological age, years[a] (SD)	69.1 (7.05)	68.8 (6.98)	69.4 (7.13)	69.1 (7.07)	68.8 (6.94)	69.7 (7.24)
Mean biological age, years (SD)	NA	NA	NA	69.1 (9.77)	68.8 (9.34)	69.7 (10.4)
Ethnicity, n (%)						
Afro Colombian	2,125 (11.8%)	1,150 (11.4%)	975 (12.3%)	343 (10.13%)	195 (9.6%)	148 (10.8%)
Indigenous	1,438 (8.0%)	687 (6.8%)	751 (9.5%)	213 (6.2%)	89 (4.3%)	124 (9.1%)
Mestizo	7,757 (43.0%)	4,262 (42.2%)	3,495 (44.0%)	1432 (42.3%)	870 (42.9%)	562 (41.3%)
Other	1,772 (9.8%)	1,105 (9.9%)	667 (8.4%)	434 (12.8%)	282 (13.9%)	152 (11.1%)
White	4,952 (27.4%)	2,897 (28.7%)	2,055 (25.9%)	963 (28.4%)	590 (29.12%)	373 (27.4%)
Low childhood SEP (Yes)	10,728 (59.5%)	7,924 (78.4%)	6587 (82.9%)	2,032 (60%)	1,201 59.3%	831 (61.1%)
Single adversity (age <15 years, Yes)						
Emotional abuse, n (%)	792 (4.4%)	467 (4.6%)	325 (4.1%)	160 (4.7%)	104 (5.1%)	56 (4.1%)
Scarcity of food, n (%)	4,805 (26.6%)	2,537 (25.1%)	2,268 (28.6%)	898 (26.5%)	506 (25.0%)	392 (28.8%)
Poor childhood health, n (%)	2,168 (12.0%)	1,131 (11.2%)	1,037 (13.1%)	425 (12.6%)	253 (12.5%)	172 (12.7%)
Domestic violence, n (%)	2,984 (16.5%)	1,792 (17.7%)	1,192 (15.0%)	588 (17.4%)	372 (18.4%)	216 (15.9%)
Forced childhood migration[c], n (%)	525 (2.9%)	291 (2.9%)	234 (2.9%)	99 (2.9%)	57 (2.8%)	43 (3.2%)
Cumulative childhood adversity (age <15 years) ACEs score [b], n (%)						
ACEs 0	5,156 (28.6%)	2,797 (27.7%)	2,359 (29.7%)	913 (27.0%)	532 (26.3%)	381 (28.0%)
ACEs 1	7,112 (39.4%)	4,051 (40.1%)	3,061 (38.5%)	1397 (41.3%)	847 (41.8%)	550 (40.5%)
ACEs 2	3,758 (20.8%)	2,141 (21.2%)	1,617 (20.4%)	554 (16.4%)	329 (16.2%)	225 (16.6%)
ACEs 3	1,551 (8.6%)	843 (8.3%)	708 (8.9%)	379 (11.2%)	232 (11.5%)	147 (10.8%)
ACEs 4+	467 (2.6%)	269 (2.7%)	198 (2.5%)	142 (4.2%)	86 (4.2%)	56 (4.3%)
Adult cardiometabolic outcomes (age >60 years old), n (%)						
Cardiovascular disease, n (%)	2,331 (12.9%)	1,355 (13.4%)	976 (12.3%)	458 (13.5%)	291 (14.4%)	167 (12.3%)
Diabetes, n (%)	2,925 (16.2%)	1,846 (18.3%)	1,079 (13.6%)	566 (16.7%)	369 (18.2%)	197 (14.5%)
Hypertension, n (%)	9,374 (52.0%)	5,818 (57.6%)	3,556 (44.8%)	1,847 (54.6%)	1,206 (59.5%)	641 (47.2%)
Obesity, n (%)	6,021 (33.4%)	3,877 (38.4%)	2,144 (27.0%)	862 (25.5%)	615 (30.4%)	247 (18.2%)

[a]Data is displayed as n (%) for categorical variables, mean (SD), and median for age. [b]Cumulative ACEs score range 0-4 and more. [c]Between 0-15 years old.

Cardiometabolic outcomes

Analysis of adult cardiometabolic outcomes showed significant gender differences. Hypertension was highly prevalent, affecting over half of the population (52%). Women showed a higher prevalence of hypertension (57.6% overall; 59.5% biomarker subsample) compared to men (52.6% overall; 59.6% biomarker subsample). Diabetes was also more prevalent among women than men (18% vs 13.6% overall sample). The most significant gender difference was observed in obesity, where women consistently showed a substantial higher prevalence (38.4% overall; 30.4% biomarker subsample) compared to men (27% overall; 18.2% biomarker subsample).

Supplementary Table 4 reports odds ratios and prevalence ratios for the association between ACEs and cardiometabolic outcomes in the overall sample. In adjusted models for demographic factors and low childhood SEP, women exposed to emotional abuse showed a strong and significant association with increased odds/prevalence of CVD (OR=1.68, PR=1.51, both $p<0.001$). This was not observed in men or for other cardiometabolic outcomes in either gender. Similar to emotional abuse, childhood maltreatment showed a strong and significant association with increased risk of CVD in women (OR=1.55, PR=1.44, both $p<0.001$), but this was not observed for other outcomes. Associations with CVD in men were less strong (OR=1.24, PR=1.20, p=0.020 and p=0.018 respectively).

Forced childhood migration was associated with increased risk of diabetes (OR=1.60, PR=1.47, p=0.005 and p=0.003 respectively), CVD (OR=1.55, PR=1.46, p=0.012 and p=0.006 respectively), and hypertension (OR=1.43, PR=1.20, p=0.008 and p=0.002 respectively) only among men. Scarcity of food was associated with increased risk of CVD in women (OR=1.44, PR=1.34, both $p<0.001$), but not with other outcomes. Poor childhood health was associated with increased risk of

CVD (OR=1.66, PR=1.50, both $p<0.001$) in women and hypertension (OR=1.16, PR=1.09, p=0.027 and p=0.013) in men.

Supplementary Table 4 also illustrates the dose-response relationship between the cumulative adverse childhood experiences and the risk of cardiometabolic diseases, particularly strong among women. Tests for trend show that as the number of ACEs increases, the risks of cardiovascular diseases (p=<0.001), diabetes (women p=0.002 and men p=0.006) and hypertension (women p=0.005 and men p=0.0002) also increase. No significant dose-response association was observed between ACEs and obesity in either gender. In general, the confidence intervals for women were narrower than those for men.

Biological age acceleration

We observed strong correlations between biological age and chronological age (KDMAge r = 0.83), which was expected since chronological age is a component of the KDM clock (Supplementary Table 3). Supplementary Table 5 reports the associations between adverse childhood experiences and biological ageing. Among all exposures, only forced childhood migration was associated with accelerated aging, with a stronger association in women. In the overall population, forced childhood migration was associated with 1.52-year higher biological age (ΔKDMAge β =1.52, 95% CI = 0.57, 2.47, p=0.002). The association was substantially stronger in women (β =2.67, 95% CI = 1.24 – 4.10), indicating that women exposed to forced migration were on average, 2.67 years biologically older than their chronological age. Forced childhood migration was also associated with higher odds of accelerated ageing (OR=1.69, 95% CI = 1.09 – 2.48, p=0.019). These findings, suggest that exposures to early-life forced migration in conflict settings has a significant long-term impact on biological ageing, with greater vulnerability observed among women.

Poor childhood health was associated with higher biological age acceleration in later lifer (ΔKDMAge β=0.53, 95% CI =0.04 – 1.01). In contrast, food scarcity during childhood showed a protective effect among men (β=-0.35, 95% CI= 0.73 – 0.03), suggesting lower biological age acceleration, although the association was not statistically significant. Similarly, cumulative childhood adversity in men was associated with lower biological age acceleration (β=-0.44; 95% CI -0.86 – 0.02). This unexpected finding may reflect unmeasured resilience pathways or suggesting a potential adaptative or selective-survivorship pattern or some type of bias; therefore, but this pattern requires cautious inter-pretation. No associations were observed between emotional abuse and accelerated biological aging. Adjusting for age, gender, ethnicity and low childhood SEP produced minimal changes in effects sizes or significant values, indicating that these factors were not major confounders.

DISCUSSION

We found an association between adverse childhood experiences and the risk of CVD, diabetes, and hypertension in adulthood, with significant gender differences. Our findings are consistent with previous literature, in particular the studies by Zou et al. [10], Souama et al. [13] and Danese et al. [6] linking childhood adversity to elevated cardiometabolic risk. Specifically, we found that women exposed to emotional abuse, scarcity of food, and domestic violence during childhood had a substantially increased risk of CVD, ranging from 27%-71%.

We observed that men who migrated before age 15 had 55% higher odds of heart diseases and stroke, 60% higher odds of diabetes, and 43% higher odds of elevated blood pressure, while such increases were not found in women. These findings are consistent with previous research by Schooling et al. [34], who identified a sex-specific critical period in early childhood migration for the development of hypertension and heart disease in men (migration at ages 0-7 OR = 3.17, 95% CI: 1.70, 5.91).

We found a clear dose-response association between the number of ACEs and cardiovascular disease and diabetes risk, with women being more vulnerable (Supplementary Table 5). Women with three ACEs had a 148% higher risk of heart disease than those with minimal or no ACEs and were twice as likely to have higher odds of heart disease compared to males with similar ACEs exposure. These gender differences in dose-response are supported by existing literature [3, 35–37].

Overall, the higher odds of cardiovascular diseases among women with multiple ACEs may be explained by biological, socioeconomic and behavioural factors, and their cumulative consequences. Our findings suggest that specific ACEs in this population could potentiate each other when in combination and increase cardiovascular disease risk. Women are likely to experience heart disease in ways that are different from men [38, 39].

A key mechanistic insight that warrants emphasis is the role of neuroendocrine pathways [23, 24]. Chronic hypercortisolism and dysregulation of the hypothalamic–pituitary–adrenal axis represent critical

mechanisms linking ACEs to accelerated biological aging. Persistent activation of the HPA axis can lead to increased allostatic load, chronic inflammation, and metabolic dysregulation, which collectively contribute to biological wear-and-tear and cardiometabolic risk [40, 41]. These pathways provide a plausible explanation for the observed associations between early-life adversity, sex-specific vulnerability, and age-related health outcomes.

The most important finding from our analyses is the novel connection between early forced childhood migration and accelerated biological ageing, but not with childhood emotional abuse, maltreatment, and scarcity of food; the latter observations contradict previous studies [42–46]. This is a relevant finding as previous studies linked childhood maltreatment to accelerated ageing [44–46] but not accelerated aging with forced childhood migration. Additionally, prior studies used prospective cohorts, and exposure to maltreatment was drawn from juvenile and adult court records rather than retrospective self-reported maltreatment like here, which is potentially subject to recall bias. Overall, across most ACEs types and biological ageing, we found a lack of statistically significant associations.

This study suggests that girls exposed to forced childhood migration during childhood tend to be biologically older than girls not exposed in later life. Previous research on the health impacts of forced childhood migration has primarily addressed women's sexual, reproductive, and mental health [47, 48]. One study in the literature has reported that migration leads to the age acceleration of tissues within migrant groups compared to non-migrants [49]. The novel connection between this early life stressor and age acceleration in women needs to be studied using longitudinal data to elucidate underlying mechanisms linking these factors to accelerated ageing, particularly in the Colombian-specific context. Our findings are specific to a Colombian subsample of older individuals. The findings of our study (including internal migration and other ACEs) should be interpreted within the context of Colombia's civil war and terrorism that occurred when a large proportion of the SABE participants were children (1955 to 1965) and led to the death of 200,000 citizens. The pathways between ACEs and biological ageing may differ across populations, age groups, or types of ACEs.

Lastly, although dose-response relationships of cumulative ACEs with the acceleration of age in later life have been reported in previous studies [50–53] we did not observe such association in the SABE's overall population. This finding is confirmed by a previous study [43] that used similar childhood adversity scores ranging from 0 to 3. The mixed evidence may be explained by the fact that studies used different operationalisations, types, and combinations of ACEs, and the use of broader ACE scores.

Limitations of the study

There are several limitations that need to be considered. First, an important limitation is the use of a deterministic approach to model biological aging. Contemporary geroscience views aging as a non-linear, stochastic process. While the KDM provides mean estimates suitable for population-level analyses, it may not capture stochastics bursts of accelerated aging at the individual level. Future longitudinal studies may better capture these dynamics. Second, the study was based on a retrospective investigation of ACEs and other risk factors, i.e., the design is limited in its ability to establish causal associations and we may not have been able to account for all confounders. Third there was limited ACEs assessment in SABE. For example, there was no information on other vital ACEs such as physical and sexual child abuse, parental mental illness, unemployment, and racism. Fourth, SABE did not collect data on the context of the exposure, duration, and severity. Fifth, self-reporting by older adults may introduce bias in ACEs recall or measurement. This could be particularly true for adversities like emotional abuse and domestic violence, which were still broadly socially accepted in the study context when adversities occurred in our population from 1955 to 1965. While people with self-reported diseases like cardiometabolic diseases can be biased in their recall of past ACEs, this is unlikely for people showing accelerated ageing based on biomarker measurements. A sixth concern is that the study relies on self-reported diagnoses for conditions such as hypertension and diabetes. While self-reports are commonly used in large epidemiological studies when clinical measurements are unavailable, they may result in underreporting of these conditions and could introduce bias.

A final concern is whether a customised version of the BA predictors can accurately compute the Klemera and Doubal index. Other studies have encountered this limitation due to data availability or the absence of biomarkers included in the original list. A number of studies using simplified biological ageing estimates, or different combinations of biomarkers to estimate BA suggests that these can be as reliable as traditional methods [46, 54–59]. Despite the extensive developments in quantifying BA rates, a universally accepted method for estimating BA is not currently available. In addition, studies in biological ageing show different methods of data collection, data availability, and association assumptions in different settings.

CONCLUSIONS

Our study reinforced existing evidence from a Colombian population that adverse childhood experiences, particularly emotional abuse, domestic violence, food insecurity and poor early health elevate cardiovascular risk in older women, while forced childhood migration emerged as a driver of diabetes, hypertension in older men, and accelerated biological ageing in older women. Cumulative ACEs exposure was associated with increased CVD, hypertension and diabetes risk but not with markers of biological ageing, except in those who experienced forced childhood migration. These individuals exhibited signs of accelerated biological aging, which means they tend to be biologically older than adults not exposed to forced childhood migration, suggesting the enduring physiological imprint of early-life trauma linked to armed conflict. As one of the first studies to capture this association between forced childhood migration and age acceleration in a Latin American context, our findings suggest long-term biological costs of conflict related childhood adversity. Future research should build on this foundation with longitudinal data, broader population, and more diverse ACEs, particularly in LMICs.

MATERIALS AND METHODS

Study design and participants

We used data from the Health, Well-Being, and Ageing Study (SABE-Colombia, first wave), which employed multistage random cluster sampling across 240 municipalities. Of 36,153 eligible older adults, 23,694 were surveyed (66% response rate). The present study included 18,044 participants with complete ACEs and self-reported health data during household interviews (Supplementary Figure 1). A subsample of 4,092 individuals from the full cohort, randomly sampled from five major cities (Barranquilla, Bogotá, Cali, Medellín, and Bucaramanga) was selected for biomarker analysis (Supplementary Figure 2).

Each participant provided written informed consent, and a local ethics committee approved the study. Blood samples were collected using a vacutainer tube and stored at–80° C following standardized protocols. Further information on the representativeness of the sample, survey implementation, data collection, quality control, and ethical approval is provided elsewhere [60, 61]. The study excluded institutionalized older people or those with significant cognitive impairment. Data on social determinants, physical function, behaviors, self-reported health status, and blood samples were collected in 2015. Questionnaires were filled by interviewers, and blood pressure, anthropometric measurements and blood samples were taken at each respondent's residence.

Adverse childhood experiences

We adapted a list of ACEs from World Health Organization (WHO) ACE-IQ questionnaire [62]. As the original SABE study did not capture all ACEs, we supplemented it with emotional abuse and forced migration available in other SABE's questionnaires. Five ACEs were included: emotional abuse, scarcity of food, poor childhood health, domestic violence, and childhood migration. All responses were dichotomized ('yes/one or more times' vs. 'no/never') to indicate exposure. Forced childhood migration was defined as a positive response to having being 'displaced due to armed conflict' and based on reported date of forced migration prior to age 15.

Biomarkers

We included 11 out of 17 markers representing cardiometabolic function for the quantification of individual biological ages, including triglycerides (mg/dL), glycated haemoglobin (HbA1C) (%), low-density lipoprotein (LDL) cholesterol (mg/dL), high-density lipoprotein (HDL) cholesterol (mg/dL), non-HDL cholesterol (mg/dL), total cholesterol (mg/dL), systolic blood pressure (mmHg), arm circumference (cm), calf circumference (cm), body mass index (BMI) and A Body Shape Index (ABSI) [63]. ABSI is a measure of body shape that estimates risk of related abdominal obesity; while BMI only considers heigh and weight, ABSI also incorporates waist circumference to better account for the distribution of body fat, particularly visceral around the abdomen, which is a known risk factor for mortality [63]. The measurement protocols for these markers have been previously described [64, 65]. Diastolic blood pressure, pulse pressure, waist circumference, knee height, 30-Second Sit-to-Stand Test, and handgrip strength were excluded because they had missing values between 30%-49%. Height and weight were excluded to avoid multi-collinearity with body mass index BMI and ABSI. Markers with skewed distributions were log-transformed (HbA1C and triglycerides).

Biological age

The Klemera-Doubal Method for Biological Age (KDMAge) is reported in Supplementary Table 1 and was calculated using the *BioAge* R package [66, 67]. KDMAge predicts individuals' biological age and provides insights into accelerated aging. KDMAge employs a relatively simple statistical model to predict

BA based on a limited set of organ functions and inflammatory markers. KDMAge has been demonstrated to be a reliable predictor of mortality [68] and provides stable results for practical assessment of the BA [55]. In addition, as it provides a more accessible method for quantifying BA, it is one of the most cross-validated biological age indicators [52, 55, 56, 67, 69, 70].

For our linear regression models, we utilized ΔKDMAge residuals, where negative values indicate slower aging and positive values indicate faster aging. For our logistic regression, the continuous KDMAge variable was dichotomized to classify individuals into "KDMAge acceleration" or "KDMAge non-acceleration". Participants with positive KDM Age values > chronological age were classified as "KDMAge acceleration"; while participants with negative or zero values < chronological age were classified as "KDMAge non-acceleration". Supplementary Table 2 describes the models used for the KDMAge.

We validated KDM implementation by: i) replicating NHANES-derived parameters from Levine [71], This means that our implementation of KDMA biological age algorithm was correct by reproducing the same parameters previously derived from NHANES data; ii) confirming age-biomarker correlations in our data were within ranges consistent with prior KDM application (r=0.73-0.89), iii) verifying that the distribution of biological age and its correlations with chronological age were comparable to those reported in studies relative to prior studies using older populations [22, 53, 56, 72]; and iv) conducting a sensitivity analyses using alternative biomarkers combination separately for women, men, and the overall population. For each specification, KDM parameters were recalibrated and biological age acceleration recomputed; across models, the magnitude and direction of associations remained unchanged.

Diseases in adulthood

SABE provides data on outcomes from self-reported diagnoses of cardiovascular diseases (CVD), hypertension (HTA), and diabetes. CVD includes self-reported episodes of heart attack, angina, and thrombosis. BMI was calculated based on physical measurements of height and weight (kg / [height (m)]^2) and used a cut-off to indicate obesity (BMI >=30).

Covariates

The covariates included in the analyses were demographic factors (age, sex, and ethnicity) and low childhood socioeconomic position. These variables were self-reported at wave 1, at the same time as the biological samples and measures were taken. The ethnic groups of the participants were Mestizo (mixed European and Amerindian heritage), white, Afro-Colombian (including raizales, palenqueros, and Afro-descended populations), indigenous, and others. Low childhood socioeconomic position was assessed using a question about socioeconomic position before 15 years old. Responses were categorized as high, intermediate, or low, and then dichotomised into "Yes" (low) and "No" (high or intermediate). Supplementary Figure 3 presents the Direct Acyclic Graph (DAG) illustrating the role of confounders and competing exposures in the relationship between ACEs, biological ageing, and health outcomes.

Statistical analysis

To study ACE exposures, we first examined each ACE individually and then calculated an overall ACEs score [18] by summing the number of single adversities.

To examine the associations between single and cumulative ACEs with four diseases, we conducted logistic regression and Poisson regression analyses stratified by gender. We fitted two sets of models for each ACE operationalisation: a crude model and a model adjusted for age, ethnicity and low childhood SEP. To test the trend with the number of ACEs, we performed a Cochran Armitage test. Using both methods, odds ratios and prevalence ratios, provided a more complete understanding of the associations and ensured that results were not biased by method-specific limitations.

Associations between ACEs and biological age measures were estimated using (1) linear regression (continuous ΔKDMAge residual) and (2) logistic regression (dichotomous KDMAge acceleration as dependent variable). Results are interpretable as effect sizes on Beta coefficients and odds ratios, accompanied by 95% confidence intervals and p-values. A two-sided p-value of < 0.05 was considered statistically significant. Several sensitivity analyses were conducted by comparing results with published biological age estimates and by replicating the same models separately in the training and testing datasets.

All analyses were performed in R Studio version 4.1.2 (The R Foundation for Statistical Computing, Vienna, Austria). R packages used in the analysis included *compareGroups* to validate correlations, and the *ggbetweenstats* package [73] and the *Sjplot* package for regression modelling [74].

AUTHORS CONTRIBUTIONS

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Juan Carlos Rivillas. The first draft of the manuscript was written by Juan Carlos Rivillas and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

ETHICAL STATEMENT AND CONSENT

Ethical approval for the SABE-Colombia study was obtained from the Ethics committees of the University of Caldas (CBCS-021-14) and University of Valle (No. 09-014 y 011-015). All participants provided written informed consent prior to participation. The present study is a secondary analysis of the de-identified publicly available data, and no additional ethical approval was required.

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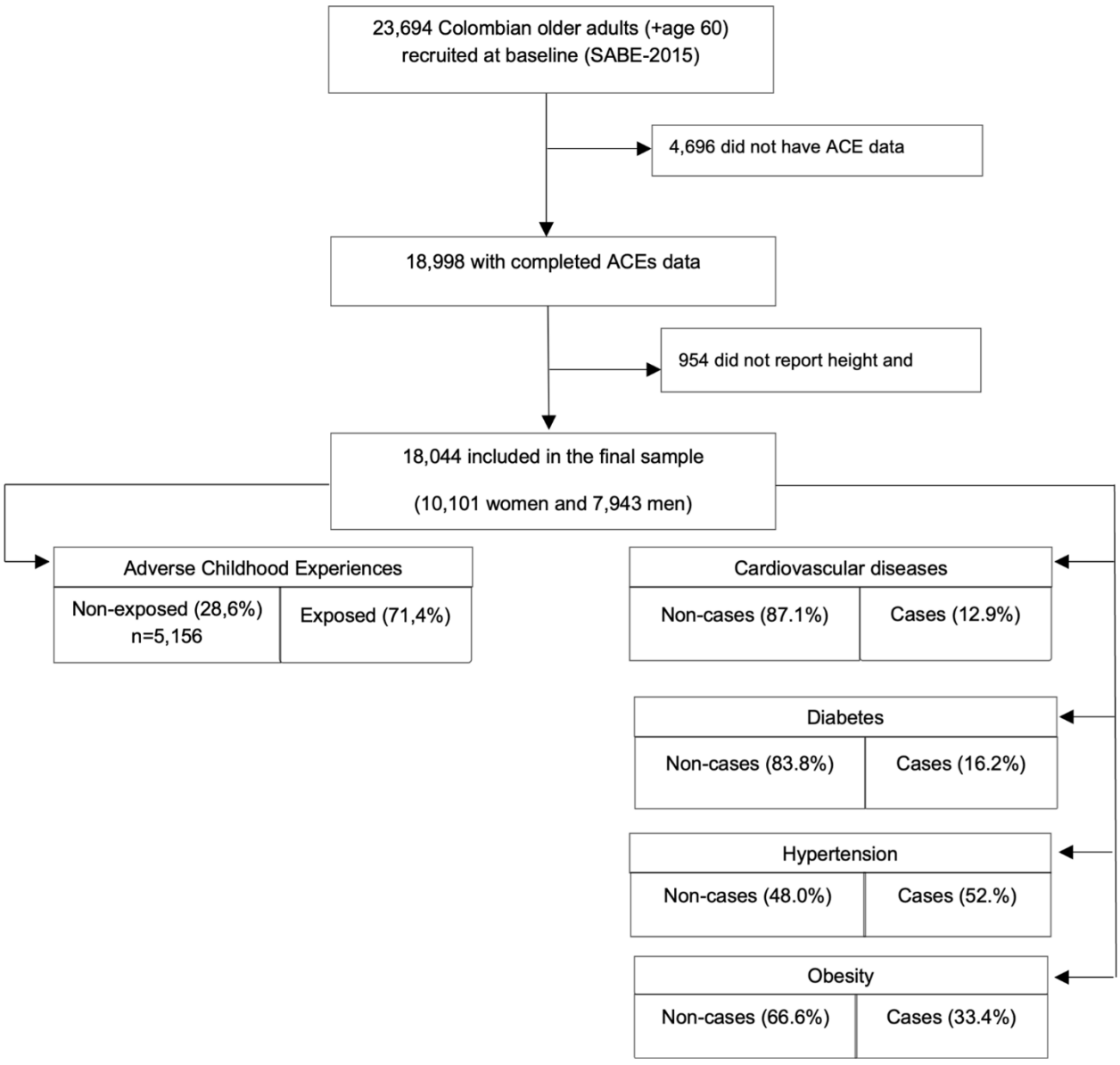
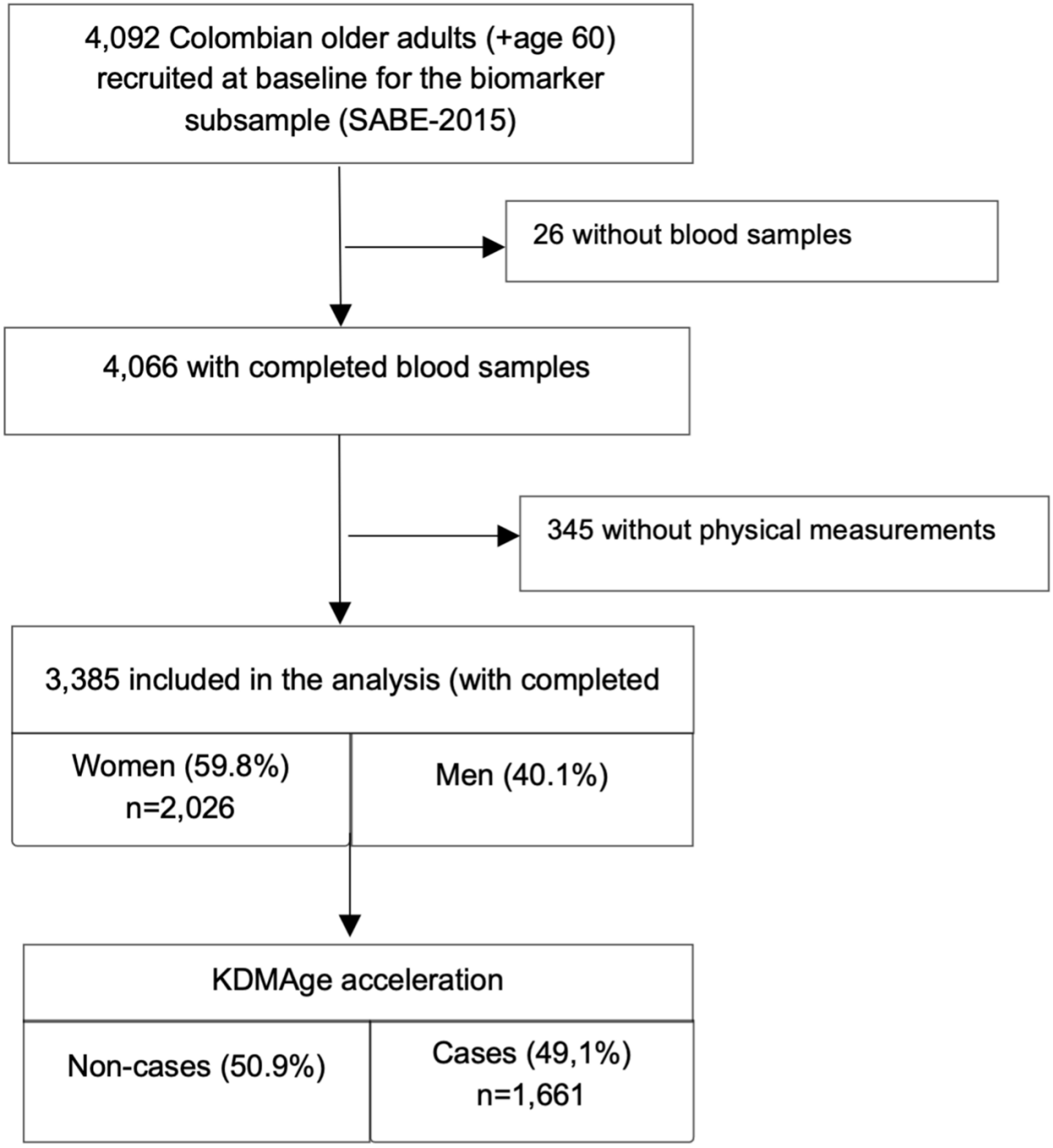
SUPPLEMENTARY MATERIALS

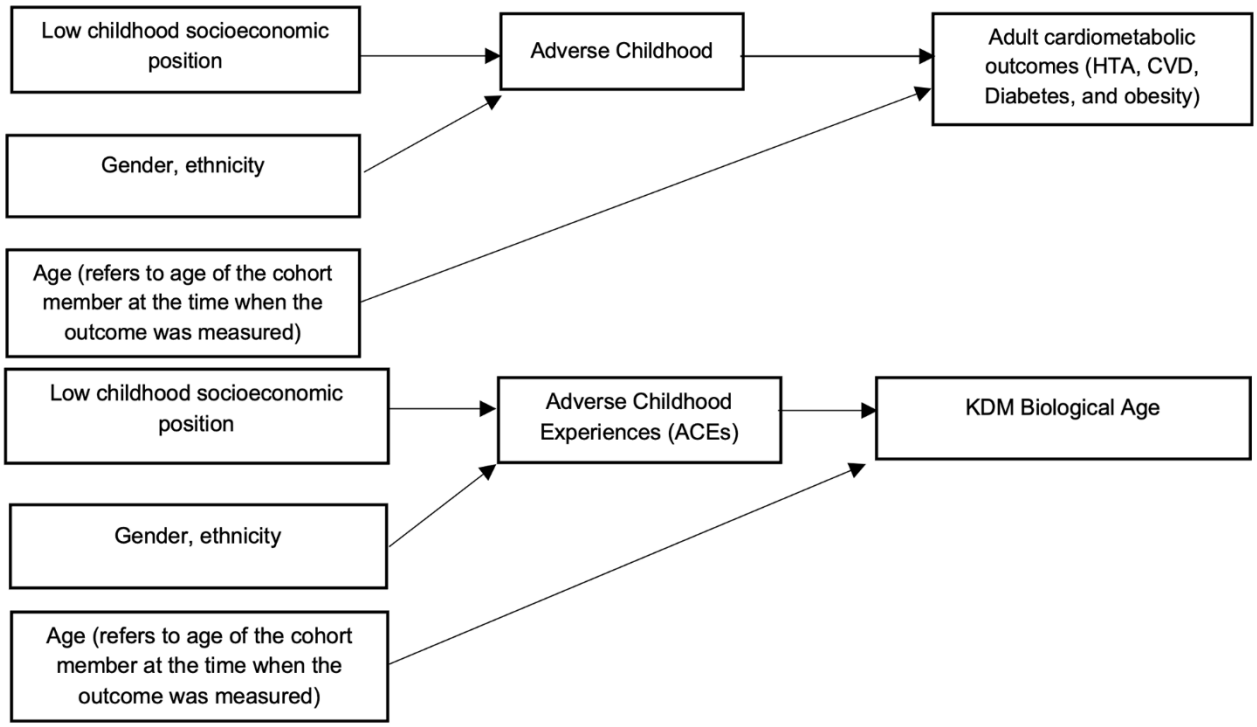
Supplementary Figures



Supplementary Figure 1. Study population selection process and profile.



Supplementary Figure 2. Study population selection process and profile (Subsample of biomarkers).



Supplementary Figure 3. This study is guided by the direct causal path and the indirect causal path of childhood adversity as exposures on adult health outcomes and biological ageing.

Supplementary Tables

Please browse Full Text version to see the data of Supplementary Tables 4, 5.

Supplementary Table 1. Biomarkers Included in the SABE-COLOMBIA Biological Age Versions.

Biomarkers	KDMAge men	KDMAge women
Triglycerides	Yes	-
HbA1c	Yes	Yes
Low-Density lipoprotein	Yes	Yes
High-Density Lipoprotein	Yes	-
Non-High-density Lipoprotein	Yes	Yes
Total cholesterol	Yes	Yes
Systolic blood pressure	Yes	Yes
A Body Shape Index	Yes	Yes
Body mass index	Yes	Yes
Calf circumference	Yes	Yes
Arm circumference	Yes	Yes

Supplementary Table 2. Definition and estimation of the Klemera-Doubal Method Biological Age (KDMAge).

Ítems	Klemera-Doubal Method Biological Age (KDMAge)
Description	KDMAge is computed based on regression models of biomarkers on age and represents the predicted physiological age of an individual (72).
Methods	The equation takes information from m number of regression lines of chronological age regressed on m biomarkers: $$KDM\ Biological\ Age = \frac{\sum_{j=1}^{m}\left(x_j - q_j\right)\frac{k_j}{S_j^2} + \frac{a}{S_{BA}^2}}{\sum_{j=1}^{m}\left(\frac{k_j}{S_j}\right)^2 + \frac{1}{S_{BA}^2}}$$
Interpretation	Positive KDMAge residual suggests that the individual is aging faster than their chronological age, meaning their biological age is higher than their actual age. ΔKDMAge residual values greater than 0, suggest faster biological ageing, whereas values less than or equal to 0, indicates slower biological ageing.
Advantages	Provides reliable and stable results and has been validated in different population samples.

Supplementary Table 3. Summary statistics for the study population with biomarkers available.

Characteristics	Women (N=2,026)	Men (N=1,359)	Overall (N=3,385)
Chronological age (year), mean ± SD[a]	68.8 (6.94)	69.7 (7.24)	69.1 (7.07)
Biological age KDMAge (years), mean ± SD[a]	68.8 (9.34)	69.7 (10.4)	69.1 (9.77)
ΔKDMAge residual	0.00 (6.25)	0.00 (7.42)	0.00 (6.74)
KDMAge acceleration [b]	1,006 (49.7%)	655 (48.2%)	1,661 (49.1%)
KDMAge non-acceleration	1,020 (50.3%)	704 (51.8%)	1,724 (50.9%)
Biomarkers included in KDMAge algorithms, mean ± SD			
HbA1c (%)	13.4 (1.28)	14.1 (1.23)	13.6 (1.31)
Low-density lipoprotein LDL (mg/dL)	127 (31.8)	120 (31.5)	124 (31.9)
High-density lipoprotein HDL (mg/dL)	46.5 (11.1)	41.2 (10.6)	44.4 (11.2)
Total Cholesterol (mg/dL)	200 (38.0)	185 (37.4)	194 (38.4)
Non-HDL (mg/dL)	154 (38.2)	144 (37.3)	150 (38.1)
Triglycerides (mg/dL)	151 (53.9)	144 (54.0)	148 (54.0)
Glucose (mg/dL)	93.9 (9.30)	93.4 (9.76)	93.7 (9.49)
SBP (mm Hg)	138 (23.9)	140 (25.0)	139 (24.3)
Calf circumference (cm)	34.8 (3.85	34.5 (3.42)	34.7 (3.68)
Arm circumference (cm)	29.2 (4.14)	28.0 (3.25)	28.7 (3.85)
A body shape index (ABSI)	0.082 (0.006)	0.083 (0.005)	0.083 (0.006)
Body-mass index BMI	27.8 (4.99)	26.3 (4.18)	27.2 (4.74)

(a) Included 3,385 participants for analysis. Mean values (standard deviation) for continuous variables and n (%) for categorical variables. (b) Employed to construct KDMAge. (c) Cardiovascular diseases, diabetes and hypertension self-reported. (e) BMI ≥ 30 kg/m² or higher defines obesity.

Supplementary Table 4. Analysis of Associations Between Adverse Childhood Experiences (ACEs) and Cardiometabolic Outcomes in Adulthood, Stratified by Gender in Older Adults in Colombia (using overall sample n=18,044).

Notes for interpretation: (a) CI confidence interval, Model adjusted for demographic factors (Age, gender, and ethnicity); and childhood socioeconomic position. Odds Ratios (ORs), Prevalence Ratios (PRs).

Supplementary Table 5. Associations of adverse childhood experiences with biological age acceleration (using the biomarker subsample n=3,385).

Notes for interpretation: CI confidence interval, Models: (a) unadjusted models; and (b) Multivariate model (Age, gender, ethnicity, and childhood socioeconomic position-adjusted model). Beta coefficient should be interpreted in years in KDMAge.