Early life adversity is associated with phenotypic age but not delay discounting in chronologically young people

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Abstract

Early life adversity results in accelerated ageing, which has been detected using molecular markers of ageing such as telomere length and DNA methylation, even in chronologically young people. Further, animal models have shown that accelerated ageing leads to a decreased willingness to wait for delayed rewards. Our study aimed to develop a simpler and cheaper marker of phenotypic age, to investigate links between early-life adversity, ageing and delay discounting. In 250 UK younger adults (ages 17-38, 121 females, 129 males), we measured early life adversity, assessed phenotypic age using 9 markers of ageing and physical function, and took two measures of delay discounting (hypothetical and experiential). As expected, lower childhood socioeconomic status was associated with greater phenotypic age for chronological age. Participants of lower childhood socioeconomic status, and those with higher childhood trauma scores had poorer self-rated health and believed that they were more likely to experience premature death. Greater phenotypic age predicted worse self-rated health and lower perceived survival odds and mediated the association between childhood socioeconomic status and self-rated health. Contrary to our predictions, there were no associations between either early life adversity or phenotypic age and delay discounting. Neither was self-rated health associated with delay discounting. Better perceived survival odds, however, predicted greater hypothetical delay discounting. These findings demonstrate that accelerated ageing can be detected in chronologically young people, using non-invasive low-cost phenotypic age markers. The young adults in our sample who reported greater childhood adversity were not only aged according to our compound phenotypic marker, but also rated their own health as being poorer and believed that they were more likely to experience premature death. This suggests that people may have an awareness of their physical state, which they may use to inform their estimates of their own survival odds. However, our results did not support our predictions regarding the antecedents of delay discounting.

Introduction

A growing number of studies have demonstrated that early life adversity influences adult health and wellbeing (e.g., Belsky et al., 2017; Kuhlman et al., 2020; Ridout et al., 2018; Suzuki, 2018). People who grow up in impoverished environments, or under stressful circumstances, age more rapidly, with effects being visible even in middle aged adults (Belsky et al., 2015). Studies using molecular markers of ageing, such as telomere length and DNA methylation, have even shown detectable differences in children and young adults based on exposure to adversity (Brody et al., 2021; Esposito et al., 2016; Jovanovic et al., 2017; Shalev et al., 2013). There are also associations between childhood trauma, socioeconomic hardship (including neighbourhood disadvantage), and molecular markers of ageing (Alexeeff et al., 2019; Dhingra et al., 2018; Lang et al., 2020; Marini et al., 2020; Wolf et al., 2018). Relatedly, neighbourhood quality has been found to be associated with allostatic load – the cumulative damage accrued in the body as a result of chronic activation of stress hormones, which is known to affect health (Buschmann et al., 2018; Carbone, 2020; Guidi et al., 2021). Yet, there are numerous ways to operationalise and measure early life adversity, and a meta-analysis comparing effects by adversity type showed that early life adversity characterised by threat, but not that characterised by socioeconomic adversity, was associated with cellular ageing (Colich et al., 2020). Given the variety of ways in which early life adversity can be conceptualised and measured, further investigation is needed to compare the effects of these different types.

Whilst molecular markers of ageing have become increasingly popular, their associations with stress and adversity can be small (Colich et al., 2020; Pepper et al., 2018). Blood and saliva sampling can be perceived as invasive and is less acceptable to some research participants (Mayeux, 2004). Furthermore, it can be practically challenging to collect and store the necessary blood or saliva samples, and expensive to analyse them. By comparison, phenotypic age markers, such as balance, grip strength, anthropometry and facial age are relatively cheap and non-invasive, making them more practical and cost-effective for large studies and field work (Xia et al., 2017). However, with such markers, it is desirable to collect measures reflective of multiple organ systems, since these may age at different rates (Belsky et al., 2015). In this study, we created a composite phenotypic age marker, which we used to investigate whether differences in phenotypic age can be seen in young adults who have experienced greater childhood adversity. We constructed a panel of non-invasive phenotypic markers of ageing and physical function, inspired by those from Belsky et al.'s (2015) study of the pace of ageing in middle-aged adults. Our panel included measures such as body mass index, waist-to-hip-ratio, lung function, blood pressure, resting heart rate, motor coordination, balance, and grip strength. We also collected perceived facial age ratings from independent observers. Such ratings have been found to be clinically useful markers of ageing in older adults (≥70 years old; Christensen et al., 2009).

In addition to being associated with adult health and ageing, prior studies have shown that early life adversity can be associated with differences in adult behaviour. For example, early experiences of adversity and lower childhood socioeconomic status have been associated with impulsivity and with measures of time preference, such as delay discounting (a measure of willingness to wait for a later-larger reward, Griskevicius et al., 2011; Lovallo, 2013; Sweitzer et al., 2013). It is possible that time preferences are accelerated by experiences of early life adversity because they cue an unpredictable environment, in which future rewards may not materialise (Griskevicius et al., 2011; Pepper & Nettle, 2013, 2017). However, it may also be that early life adversity leaves people in a poorer physical state, reducing subjective life expectancy, and thereby shortening time horizons (Nettle et al., 2013). Indeed, studies on starlings suggest that telomere attrition experienced during development (an indicator of

accelerated ageing) is associated with greater adult impulsivity (Dunn et al., 2019; Nettle et al., 2015). If an awareness of physical state affects delay discounting, we might therefore expect phenotypic age to mediate the association between early life adversity and delay discounting. We therefore investigate whether greater phenotypic age is associated with lower perceived survival odds, and steeper delay discounting.

Based on the rationale reviewed above, we hypothesise that greater exposure to adversity should lead to greater relative phenotypic age. Greater phenotypic age should, in turn, predict poorer self-perceived health and survival odds (indicators of self-perceived state). Finally, poorer self-rated health and survival odds may be associated with greater delay discounting (Figure 1). The objectives of this study were therefore as follows:

O1. To develop a novel compound measure of phenotypic age using easy-to-measure non-invasive markers.

O2. To establish whether early life adversity and cSES predict phenotypic age in young adults.

O3. To explore whether phenotypically older people are aware of their relatively poor state, using measures of self-rated health and perceived survival odds and, if so, to investigate whether phenotypic age mediates the association between adversity and perceived state.

O4. To establish whether phenotypic age, self-rated health, and perceived survival odds are subsequent predictors of delay discounting.

Methods

Participants.

Two hundred and fifty participants (121 female, 129 male), aged 17-38 (mean = 21.6 years, SD = 3.92), were recruited to the study via a range of sources in Newcastle upon Tyne, UK, with the aim of recruiting younger people from varied socioeconomic backgrounds. To ensure that the sample were chronologically younger than those participants used by Belsky et al. (2015), we used 38 years as the upper age boundary in recruitment. Recruitment groups included the Newcastle University student body, the Newcastle Institute of Neuroscience Research Participation Pool, and local community organisations in a socioeconomically deprived area of the city. Of the 250, a sub-sample of 140 completed the self-rated health, delay discounting, and subjective awareness measures.

The study had Newcastle University Research Ethics Committee approval (approval number 01208_1). Participants gave informed consent as part of the electronic questionnaire completed on their arrival at the laboratory. Participants gave separate consent for facial photographs to be taken and rated by observers (see below).

Measures of early-life exposures.

Childhood trauma questionnaire. Participants completed the Childhood Trauma Questionnaire (Pennebaker & Susman, 1988). This records the presence of up to six traumatic experiences (death, divorce, violence, sexual abuse, illness, or other) experienced prior to the age of 17, and retrospectively assesses the level of psychological distress associated with each experience (using a 7-point scale, from 1, *not at all traumatic* to 7, *extremely traumatic*). This information was used to derive two variables: a count of the childhood traumas experienced, and a combined score for the level of subjective stress associated with those traumas experienced. Since childhood trauma count and childhood trauma score were highly correlated ($r = 0.91$, $p < 0.001$), only the latter was included in our analyses. We chose to use the trauma score because it contains more information: it will naturally be higher if the count of traumas is greater, but it is also sensitive to the extent to which the events were reported as subjectively stressful.

Childhood socioeconomic adversity. We used three measures of childhood socioeconomic adversity: subjective childhood socioeconomic status (cSES); index of multiple deprivation of residential neighbourhood (IMD); and a neighbourhood quality rating obtained by having independent coders rate Google Streetview images of the neighbourhood in which the respondent grew up (see below).

Subjective childhood socioeconomic status (cSES). Participants rated their agreement with three statements, developed by Griskevicius et al. (2011), designed to measure cSES: (a*) "My family usually had enough money for things when I was growing up"*; (b) "*I grew up in a relatively wealthy neighbourhood"*; (c) "*I felt relatively wealthy compared to the other kids in my school"*. Responses, on a scale from 1, *strongly disagree*, to 7, *strongly agree*, were summed.

Index of Multiple Deprivation (IMD). Participants were asked for their postcode or, if they could not remember their postcode, their street address at age 5. If they could remember neither their postcode nor their street address at age 5, they were asked for the earliest postcode they could remember, with the corresponding age at residence. Childhood addresses and postcodes were used to obtain an area score from the Office for National Statistics' 2015 English Indices of Multiple Deprivation (IMD; Office of National Statistics, 2015). The IMD combines seven area-level economic and social indicators into a single score: income deprivation, employment deprivation, education, skills and

training deprivation, heath deprivation and disability, crime, barriers to housing and services, and living environment deprivation. The scores are combined using a weighting established based on the academic literature and the robustness of the indicators. These IMD scores are considered a useful objective measure of an individual resident's SES (Danesh et al., 1999). We used the lower layer super output area (LSOA) scores, which are the smallest areas for which scores are available. A higher score indicates greater deprivation, with the possible range being from 0.48 to 92.60. IMD scores were only available for participants who grew up in England. For those participants who grew up abroad, or in other parts of the UK, IMD score is missing from our data (n = 58).

Neighbourhood quality rating. Using the childhood postcodes and addresses, we obtained Google Streetview Inline Frames (IFrames). These IFrames enabled us to embed Google Streetview for participants' childhood neighbourhoods into our survey to allow independent observers to virtually explore the neighbourhoods and rate them for perceived safety and quality following the methods of Odgers et al. (2012). Each neighbourhood was virtually explored by between 25 and 27 independent observers, who were recruited via Prolific [www.prolific.co], an online participant recruitment platform that offers a high-quality participant pool of research-participant volunteers. To avoid order and fatigue effects, random subsets of the neighbourhoods were presented to each observer, resulting in a slight variation in the number of ratings per neighbourhood. Observers then answered the following questions, again following Odgers et al. (2012): 1) *"Does this seem like a safe place to live?"* (Answer on a scale from 0, *not at all safe* to 5, *perfectly safe*), 2) *"How safe would you feel walking at night in this neighbourhood?"* (Answer on a scale from 0, *not at all safe* to 5, *perfectly safe*), 3) "*Do you think this is a good neighbourhood?"* (Answer on a scale from 0, *a very bad neighbourhood* to 5, *a very good neighbourhood*). Following the method of Odgers et al. (2012), we summed responses to give a single score, henceforth referred to as neighbourhood quality score.

Phenotypic age markers.

We developed a composite phenotypic age measure, comprising several markers that have previously been found to be useful indicators of ageing and physical functioning, even in those who haven't reached old age (Belsky et al., 2015): body mass index (BMI), waist-to-hip-ratio (WHR), lung function (FEV_1/FVC ratio), mean arterial pressure (MAP), resting heart rate (BPM), motor coordination (seconds taken to complete test), balance (unipedal stance test time), grip strength taken from the dominant hand (lbs of pressure), and perceived face age as rated by independent observers. The supplement contains details of calculation methods and further information on how each marker was measured.

Not all our markers correlated with chronological age in this sample (supplement table S2), which is to be expected given the restricted range of participant ages. Moreover, the overall Measure of Sampling Adequacy according to the Kaiser-Meyer-Olkin (KMO) test was low (0.5). Thus, a principal components analysis (PCA) approach to calculating a single phenotypic age score was not deemed suitable (Jia et al., 2017; Zhang et al., 2014). We therefore took the approach of using a sex-specific sum of z-scores: that is, we divided the data based on sex (due to the sexual dimorphism expected for most of our measures) and calculated a z-score for each participant, for each marker. The sum of those z-scores gave us our age score, henceforth called *phenotypic age*. Since z-scores were calculated separately for males and females, sex is not included as a covariate in models predicting phenotypic age. To ensure that a higher z-score corresponded with greater phenotypic age, scores for the unipedal stance test, lung function, and grip strength were reversed, since a higher score on these measures corresponds to a better physical condition. For a small number of cases, individual markers were

missing, and a sex-specific mean was imputed before the z-score was calculated. Further details are given in the supplement.

Subjective measures of state.

Self-rated health. In a sub-sample of 140 participants, self-rated health was recorded using a measure from the UK Census (UK Census, 2011) The question took the form of "*How would you describe your health in general?*" Options were on a Likert scale from '*very good*' (1) to '*very bad*' (5) (see supplement for more details). A higher score on this measure therefore represents poorer self-rated health.

Perceived survival odds. In the sub-sample of 140 participants, we also measured perceived odds of premature death and self-reported health behaviour. The former was intended to explore whether more phenotypically aged people subjectively felt their lives would be shorter, and the latter because it has been argued that those with poorer somatic prospects should be expected to put less effort into protective health behaviour (refs). Perceived odds of premature death were measured by asking a question previously used in the Health and Retirement Study: "*What do you think the chances are that you will live to be 75 or more? 0 is 'no chance' and 100 is 'definitely'*." Seventy-five years is taken as the cut-off for premature death (Remington et al., 2013). Responses to this question have previously been found to behave like probabilities, and to covary with relevant variables such as smoking behaviour (Hurd & McGarry, 1995).

Delay discounting.

In the subsample of 140 participants, we also took two measures of delay discounting, a hypothetical discounting task (HDDT), and an experiential discounting task (EDT).

Hypothetical discounting task (HDDT). Participants made 20 hypothetical choices between a larger monetary reward "in a year's time" (the delayed reward) and a smaller monetary reward "today" (the immediate reward). The delayed reward option was always £100, while the immediate reward options ranged from £1 to $99, presented in ascending order (to encourage consistent answers), with both the delayed reward choices and the delay period held constant. The range of k parameters (k expresses the point of indifference between immediate and delayed rewards) represented by these choices were between 0.27123 and 0.00003 (where k = (A-V)/(VD), with A being the amount of the delayed reward, V being the present subjective value of the delayed reward and D being the delay). This range of values is slightly larger than the range that can normally be expected in similar populations (Kirby et al., 1999; Kirby & Maraković, 1995). Our outcome variable from this measure is simply the number of times the immediate reward is chosen, as in previous studies (Pepper & Nettle, 2013). As such a higher delay discounting score represents greater impatience, or lower willingness to wait.

Experiential discounting task (EDT). In the EDT, participants are presented with repeated choices between smaller-sooner monetary rewards and later-larger ones with the delays (which are a matter of seconds) being experienced rather than explicitly stated and hypothetical. The task was presented on a computer using the Inquisit software (Millisecond, Seattle, WA; www.millisecond.com). The EDT script, taken from the Inquisit test library (http://www.millisecond.com/download/library/), is based on the EDT task described by Reynolds and Schiffbauer (2004). We modified the Inquisit script to present rewards in Great British Pounds, rather than US dollars, which is the default. We also modified task instructions to make them more suitable for our participants. After seeing task instructions, all participants experienced a practice round. In each EDT trial, participants see two light bulbs labelled with different amounts of money and must choose one. Our instructions emphasised that there was no

correct answer, and that participants should simply choose as they prefer. Of the two options presented, the first is always 30p. The second varies but is always less than 30p. If participants choose the larger reward, they experience a delay of either 0, 7, 14 or 28s, increasing with each round of trials. The smaller immediate reward is received without delay, but there is some probability that it will not be received at all (a 0.3 probability in all trials). Upon receiving their chosen amount of money, participants must click on an illuminated bank symbol to store the money and move to the next trial. The participants' "bank balance" is always visible at the bottom of the screen. To ensure that participants experience both delayed and immediate outcomes, they are presented with a forced choice (only one option can be selected) if they selected the same option in the 4 previous trials. The EDT uses an adjusting-amount procedure to determine an indifference point for each participant (the point of indifference between immediate and delayed rewards). When the larger delayed reward is chosen, the value of the small immediate ward is increased, and whenever the participant chooses the smaller immediate reward, its value decreases in the subsequent trial. Once a participant has chosen the same number of larger delayed and smaller immediate rewards across 6 trials, the indifference point is established and the round ends. Each round consists of at least 16 trials. If the indifference point is established after 16 trials, the round ends. If the indifference point cannot be established, the trials continue until an indifference point can be determined. To prevent the task from continuing indefinitely for participants with an unclear indifference point, rounds have a set duration of 20 times the length of the delay used for delivering the larger reward in that round. If a round is completed before this maximum duration, the participant experiences an inter-round interval. This ensures that participants do not complete the EDT more quickly by continuously selecting the immediate reward. After the practice round, all participants were presented with a shortened version of the initial instructions, as a reminder before beginning the 4 real rounds (one round for each delay length: 0, 7, 14 or 28s). Indifference points from each of these rounds were used to calculate the area under the curve, which was our main outcome variable from the EDT. As such, higher indifference points, which represent greater patience, give a larger value for the area under the curve.

Analysis.

All statistical analyses were performed using R (R Core Team, 2021). The R script used for analysis is available alongside our data on the Open Science Framework: https://osf.io/pkqt5/. The following packages were used for data processing, description, analysis, and visualisation: tidyverse (Wickham et al., 2019), corrplot (Wei & Simko, 2017), pysch (Revelle, 2021), ggplot2 (Wickham, 2016), purrr (Henry & Wickham, 2020), and apaTables (Stanley, 2021).

We first examined distributions of, and Pearson's correlations between, our key measures of adversity and of phenotypic age. Since our childhood socioeconomic adversity variables (cSES, IMD score and neighbourhood quality score) were only moderately correlated (Table 2), we ran a multiple linear regression model predicting phenotypic age using all the childhood socioeconomic variables as separate predictors of phenotypic age. Since it is qualitatively different from socioeconomic adversity, we ran a separate model with childhood trauma score as the predictor. Both models controlled for chronological age as there was some variation in this (ages ranged from 17-38). As phenotypic age was the outcome variable, and phenotypic age was calculated with respect to sex, we did not control for sex in these models.

To assess how early life adversity related to self-rated health, and perceived odds of survival beyond 75 (the threshold before which death is considered premature), we ran multiple linear regression models predicting self-rated health and perceived survival odds from our childhood socioeconomic

adversity variables, and from childhood trauma score. We then did the same to assess the relationships between phenotypic age and self-rated health, and phenotypic age and perceived survival odds. We ran mediation analyses using the R PROCESS macro (Hayes, 2022) to assess whether phenotypic age was a mediator of the associations between cSES and subjective health/perceived survival odds.

We then ran separate multiple linear regression models predicting our delay discounting measures (hypothetical delay discounting and EDT area under the curve) from our childhood socioeconomic variables, our childhood trauma score, and phenotypic age. Both models controlled for chronological age and sex. We did not look for mediation effects, as originally planned, as there were no associations to be mediated.

Results

Descriptive statistics for the continuous predictor variables are shown in table 1.

Table 1.

Descriptive statistics for our continuous predictor variables: age and the early life adversity measures

	n	Mean	SD	Median	Min	Max	Possible range
Chronological age	250	21.61	3.92	20.00	17	38	17 - 38
cSES	250	14.28	4.14	15.00	3	21	3 - 21
Postcode IMD score [*]	192	15.33	14.12	10.50	1	69	0.53 – 87.80
Childhood trauma count [#]	139	1.57	1.25	2.00	0	5	0 - 6
Childhood trauma score [#]	139	7.01	6.26	6.00	0	28	0 - 42
Neighbourhood quality score	237	10.47	1.50	10.67	5	14	0 - 15

Note. *n* = sample size. *SD* = standard deviation. [*] For postcode IMD score, some data are missing where participants could not recall or did not want to give their childhood postcode, or where they were not resident in England as a child, meaning that an IMD score was not available. A higher IMD score indicates grater deprivation. [#] Childhood trauma questions were only asked of a subset of participants (n = 139). Since childhood trauma count and childhood trauma score were highly correlated, we used only childhood trauma score in our main analyses. Childhood trauma count is included here solely to give descriptive information.

Objective 1: Phenotypic age markers and the associations between them

Descriptive information for our phenotypic age markers is given in the supplement (table S1). There were significant positive correlations between some, but not all, individual phenotypic age markers (table S2). These correlations were small to medium in size ($r = 0.13 – 0.41$). Phenotypic age was associated with chronological age ($r = 0.21$, $p < 0.001$).

Objective 2: Early life adversity and phenotypic age

Neighbourhood quality scores were significantly correlated with postcode IMD scores ($r = -0.47$, $p < 0.001$, table 2), such that higher deprivation according to the IMD was associated with a lower perceived neighbourhood quality, in line with the findings of Odgers et al (2012). There was also a significant association between cSES and neighbourhood quality scores: the neighbourhoods of those participants who reported higher cSES were given higher neighbourhood quality ratings by our independent observers ($r = 0.29$, $p < 0.001$, table 2). In line with this, there was also a significant negative correlation between cSES and IMD scores ($r = -0.57$, $p < 0.001$, table 2), indicating decreasing area deprivation with increasing cSES.

There were no significant associations between neighbourhood quality scores and reported childhood trauma scores ($r = -0.14$, $p = 0.09$), or between IMD scores and childhood trauma scores ($r = -0.07$, $p = 0.47$). However, higher cSES scores were associated with lower childhood trauma scores ($r = -0.24$, $p < 0.01$).

Zero-order correlations revealed associations between both subjective cSES and postcode IMD score and phenotypic age (table 2). Perceived neighbourhood quality was not correlated with phenotypic age, despite its association with both cSES and IMD score. In a multiple linear regression model including all our cSES variables, and controlling for chronological age, only cSES predicted

phenotypic age (table 3). Our childhood trauma score did not significantly predict phenotypic age in a model controlling for chronological age ($\beta = 0.41$, $p = 0.16$, $n = 139$, $F_{(2,136)} = 1.37$, $R^2 = 0.01$).

Table 2.

Means, standard deviations, and correlations with confidence intervals for adversity variables, chronological and phenotypic age.

Variable	*M*	*SD*	1	2	3	4	5
1. cSES	14.28	4.14					
2. Postcode IMD score	15.33	14.12	-.57** [-.66, -.47]				
3. Neighbourhood quality score	10.47	1.50	.29** [.17, .40]	-.47** [-.57, -.35]			
4. Childhood trauma score	7.01	6.26	-.24** [-.39, -.07]	-.07 [-.25, .12]	-.14 [-.30, .03]		
5. Chronological age	21.61	3.92	-.11 [-.23, .01]	.27** [.13, .39]	-.03 [-.16, .09]	-.05 [-.21, .12]	
6. Phenotypic age	0.00	3.34	-.27** [-.38, -.15]	.30** [.16, .42]	-.12 [-.25, .01]	.12 [-.04, .28]	.28** [.16, .39]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. A higher cSES score indicates higher SES, whilst a higher postcode IMD score indicates a more deprived childhood postcode and a higher neighbourhood quality score indicates a better perceived quality of neighbourhood.

Table 3.

Regression results using phenotypic age as the outcome variable and socioeconomic adversity variables as the predictors.

Predictor	*Beta*	*Beta* 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-0.24	[-0.75, 0.27]			
cSES	-0.71*	[-1.30, -0.12]	.03	[-.02, .07]	
Postcode IMD score	0.51	[-0.16, 1.18]	.01	[-.02, .04]	
Neighbourhood quality score	-0.10	[-0.70, 0.50]	.00	[-.01, .01]	
Chronological age	0.10	[-0.53, 0.73]	.00	[-.01, .01]	
					R^2 = .106** 95% CI [.02,.18]

Note. Beta indicates the standardized regression weights. sr^2 represents the semi-partial correlation squared. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively. * indicates $p < .05$. ** indicates $p < .01$.

Objective 3: Early life adversity, phenotypic age and their associations with perceived health and survival odds

Zero-order correlations revealed associations between cSES and subjective health, with participants of higher cSES reporting better health. A higher childhood trauma score was also associated with poorer subjective health. People with a higher cSES score also perceived their odds of survival beyond 75, the age before which death is considered premature, to be better. Meanwhile, people with higher childhood trauma scores perceived their odds of survival beyond 75 to be poorer (table 4).

Table 4.

Means, standard deviations, and correlations with confidence intervals for early life adversity measures, self-rated health, and perceived survival odds

Variable	*M*	*SD*	1	2	3	4	5
1. cSES	14.28	4.14					
2. Postcode IMD score	15.33	14.12	-.57** [-.66, -.47]				
3. Neighbourhood quality score	10.47	1.50	.29** [.17, .40]	-.47** [-.57, -.35]			
4. Childhood trauma score	7.01	6.26	-.24** [-.39, -.07]	-.07 [-.25, .12]	-.14 [-.30, .03]		
5. Subjective health	2.00	0.74	-.32** [-.46, -.16]	.13 [-.05, .31]	-.08 [-.24, .09]	.28** [.12, .43]	
6. Perceived survival odds	77.69	16.47	.33** [.18, .47]	-.09 [-.27, .09]	.13 [-.03, .29]	-.23** [-.38, -.06]	-.32** [-.46, -.16]

Note. M and *SD* are used to represent mean and standard deviation, respectively.
Values in square brackets indicate the 95% confidence interval for each correlation.
The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014).
* Indicates $p < .05$. ** indicates $p < .01$.

In multiple linear regression models controlling for chronological age and sex, cSES and childhood trauma scores remained significant predictors of self-rated health (table 5). The models also revealed sex differences in self-rated health, with females reporting better subjective health (table 5; $\text{Mean}_{\text{male}} = 2.16$, $\text{SD}_{\text{Male}} = 0.76$, $\text{Mean}_{\text{Female}} = 1.91$, $\text{SD}_{\text{Female}} = 0.72$). Full models also confirmed that the relationship between cSES and perceived survival odds held when controlling for chronological age and sex (table 6). The models revealed a sex difference in perceived survival odds, such that males

perceived themselves as less likely to survive beyond the age of 75 – the threshold for premature death (table 6; Mean $_{male}$ = 72.69, SD $_{Male}$ = 20.24, Mean $_{Female}$ = 80.59, SD $_{Female}$ = 13.11).

Models controlling for chronological age revealed that greater phenotypic age corresponded with poorer self-rated health ($\beta = 0.13$, $p < 0.001$, $n = 139$) and lower perceived odds of survival beyond the age of 75 ($\beta = -0.07$, $p < 0.05$, $n = 139$).

Mediation analysis using the R PROCESS macro revealed that around 33% of the association between cSES and self-rated health was mediated by phenotypic age ($\beta_{indirect} = -0.08$, 95% CIs = -0.15, -0.02). However, phenotypic age was not a significant mediator of the association between cSES and perceived survival odds ($\beta_{indirect} = 0.03$, 95% CIs = -0.0003, 0.08).

Table 5.

Regression results from models including socioeconomic adversity variables and childhood trauma score, and controlling for age and sex, both with self-rated health as the outcome.

Predictor	*Beta*	*Beta* 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	*r*	Fit
(Intercept)	0.41	[-0.28, 1.10]				
cSES	-0.26*	[-0.48, -0.05]	.05	[-.03, .12]	-.27**	
Postcode IMD score	0.03	[-0.24, 0.30]	.00	[-.01, .01]	.13	
Neighbourhood quality score	-0.03	[-0.24, 0.18]	.00	[-.01, .01]	-.07	
Chronological age	-0.36	[-0.84, 0.12]	.02	[-.03, .06]	-.12	
Sex	-0.34	[-0.73, 0.04]	.03	[-.03, .08]	-.22*	
						R^2 = .121* 95% CI [.00, .21]
(Intercept)	0.51	[-0.08, 1.09]				
Childhood trauma score	0.28**	[0.12, 0.44]	.08	[-.00, .17]	.28**	
Chronological age	-0.18	[-0.55, 0.19]	.01	[-.02, .03]	-.09	
Sex	-0.36*	[-0.69, -0.03]	.03	[-.02, .08]	-.16	
						R^2 = .115** 95% CI [.02, .21]

Note. beta indicates the standardized regression weights. sr^2 represents the semi-partial correlation squared. *r* represents the zero-order correlation. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.

Table 6.

Regression results from models including socioeconomic adversity variables and childhood trauma score, and controlling for age and sex, both with perceived survival beyond age 75 as the outcome.

Predictor	*Beta*	*Beta* 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	*r*	Fit
(Intercept)	-0.70*	[-1.37, -0.03]				
cSES	0.31**	[0.10, 0.52]	.07	[-.02, .16]	.31**	
Postcode IMD score	0.08	[-0.18, 0.35]	.00	[-.02, .02]	-.09	
Neighbourhood quality score	0.07	[-0.14, 0.27]	.00	[-.02, .02]	.09	
Chronological age	-0.10	[-0.57, 0.37]	.00	[-.01, .01]	-.05	
Sex	0.41*	[0.03, 0.78]	.04	[-.03, .10]	.25**	
						R^2 = .140** 95% CI [.01,.23]
(Intercept)	-0.93**	[-1.52, -0.35]				
Childhood trauma score	-0.24**	[-0.40, -0.08]	.06	[-.02, .13]	-.23**	
Chronological age	-0.28	[-0.65, 0.09]	.01	[-.02, .05]	-.12	
Sex	0.49**	[0.16, 0.82]	.06	[-.02, .13]	.23**	
						R^2 = .124** 95% CI [.03, .22]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. sr^2 represents the semi-partial correlation squared. *r* represents the zero-order correlation. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.

* indicates $p < .05$. ** indicates $p < .01$.

Objective 4: Phenotypic age and delay discounting

There was a significant negative association between the HDDT and EDT scores (r = -0.25, p < 0.01), indicating that greater patience during the EDT was associated with fewer immediate choices on the HDDT. None of the socioeconomic adversity measures significantly predicted HDDT scores, and neither did childhood trauma score, nor phenotypic age (table S3). Measures of early socioeconomic adversity and trauma score did not predict impatience in the EDT, and neither did phenotypic age (table S4). Since neither early life adversity nor phenotypic age predicted delay discounting, we did not test for mediation effects. There was, also no association between self-rated health and either HDDT or EDT scores (tables S3 & S4). Neither did perceived survival odds predict EDT scores (table S4). There was, however a small, significant association between perceived survival odds and HDDT score such that greater perceived survival odds were associated with steeper delay discounting.

Figure 3 shows a summary of which of our predictions were supported by our data. Some measures of early life adversity (cSES and childhood trauma score) were associated with greater phenotypic age which, as predicted, was subsequently associated with poorer self-rated health and lower perceived survival odds. However, self-rated health and perceived survival odds did not predict experiential delay discounting and only perceived survival odds were significantly associated with HDDT scores. The association, however, was small, and not in the expected direction, and thus did not support of our predictions.

Discussion

In this study, we developed a compound phenotypic age marker to establish whether phenotypic age differences could be detected in younger people who had experienced greater early life adversity, and whether phenotypic age would be associated with poorer self-rated health, poorer perceived survival odds, and greater delay discounting. We predicted that greater early adversity would be associated with greater phenotypic age which, in turn, would predict poorer health and perceived survival odds, and greater delay discounting.

In line with our expectations, cSES and childhood neighbourhood IMD scores were correlated with phenotypic age scores. However, despite its relationship with the other measures of childhood socioeconomic adversity, childhood neighbourhood quality (as rated by independent observers via Google Streetview) was not associated with phenotypic age. Further, in a model including all childhood socioeconomic adversity measures as predictors, only cSES was a significant predictor of phenotypic age. This may be a result of our sample being chronologically young, or of the cross-sectional design of our study, since longitudinal changes in neighbourhood socioeconomic status have been found to be associated with changes in telomere attrition in older adults (Brown et al., 2021). Contrary to our predictions, there was no association between childhood trauma score and phenotypic age, despite childhood trauma score being associated with both self-rated health and perceived survival odds. This is surprising, given that a recent meta-analysis revealed that early life adversity characterised by threat, but not by deprivation or SES, was associated with ageing (Colich et al., 2020).

Participants of lower cSES had higher perceived odds of premature death, as did those with higher childhood trauma scores. A greater phenotypic age was associated with poorer self-rated health and lower perceived survival odds, suggesting that participants of lower cSES are not only in a poorer state, but also know that they are, and believe that this will increase their odds of premature death. Formal mediation analysis supported a mediating effect of phenotypic age in the association between cSES and self-rated health, but not in the relationship between cSES and perceived survival odds. An open question is whether people, even when they are chronologically young and generally healthy, can proprioceptively sense their physiological state, or whether this is something that they infer based on their knowledge of their environments or the likely consequences of their health behaviour. Whilst some have attempted investigations into the possibility that people may sense their own impending deaths (Miglietta et al., 2009; Ngeh, 2003), it remains difficult to discern whether people can really do this, or how. It may be because they sense their own internal state but recall biases and alternative mechanisms are not easily ruled out.

We did not detect any of the predicted associations between early life adversities and our hypothetical and experiential measures of delay discounting. Neither did we see the predicted association between phenotypic age and delay discounting. This was counter to our expectations, given that prior studies have indicated associations between early life socioeconomic adversity and adult delay discounting (Acheson et al., 2019; Fields et al., 2014; Griskevicius et al., 2011; Lovallo, 2013; Sweitzer et al., 2013). However, reviews have demonstrated that findings are mixed overall (Fields et al., 2014), and some studies have found associations to be moderated by other factors, such as genotype (Sweitzer et al., 2013) or immediate environment (Griskevicius et al., 2011). Our study may also be limited in terms of power, as we only had delay discounting data for a subsample of our participants. Given that meta-analysis has revealed both cumulative and concurrent stress to be associated with measures of delay discounting and impulsivity (Fields et al., 2014), an interesting question remains: Do people who have experienced more early adversity also experience more stress as adults, and could adult stress levels account for some of the association previously ascribed to early life adversity?

Self-rated health and perceived survival odds were not associated with area under the curve from the experiential discounting task. We did find that our measure of perceived survival odds was associated with hypothetical delay discounting, but the direction of the association was contrary to our prediction. On the basis that poorer survival odds introduce collection risk (the risk that circumstances will intervene to prevent the collection of future rewards), we predicted that they would be associated with greater delay discounting (Bulley & Pepper, 2017; Mell et al., 2019; Pepper & Nettle, 2013, 2017). However, we found a small but significant association in the opposite direction. Whilst this association may be spurious, it may also indicate that more-complex relationships are at play. For example, there may be interactions between early life experiences, current perceptions of environment, and physiological state. Interactions between factors such as cSES and genotype (Sweitzer et al., 2013) and cSES and risk primes (Griskevicius et al., 2011), and cSES and current scarcity (Griskevicius et al., 2013) have been found to predict delay discounting in prior studies. However, such studies require replication (Pepper et al., 2017) and attempts at research synthesis are needed.

A key strength of this study is that it provides a novel measure of phenotypic age, which is cheap, non-invasive, and uses portable equipment, making it suitable for field studies. Our measure showed associations with measures of early adversity which are similar to, or stronger than, associations seen using biomarkers of ageing such as telomeres and DNA methylation (for effect sizes from meta-analyses, see Colich et al., 2020; Pepper et al., 2018), even in an age-restricted set of chronologically young people. A potential limitation of the measure is that it doesn't cover all organ systems because, without taking blood and urine samples, measures such as blood glucose, cholesterol and urea nitrogen cannot be used. However, our measure of phenotypic age shows a moderate association with self-rated health ($r = 0.41$) – a stronger association, indeed, than those seen with popular biomarkers such as telomere length, epigenetic clocks, biological age measures calculated using the Klemera-Doubal method, and innovative composite measures such as pace of ageing (correlations for which range between $r = -0.02$ and $r = -0.28$; Belsky, Moffitt, et al., 2017). This is not to say that the association with self-rated health provides an index of the performance of our measure relative to others. Merely, that the fact that we see associations of similar magnitudes and directions to those using other biological age markers in other studies indicates a degree of predictive validity for our measure of phenotypic age. We note that another measure has recently been developed, which has also been referred to as Phenotypic Age (Liu et al., 2018). Though our approaches are similar in that they use a linear combination of markers, Liu et al. (2018) combine clinical chemistry biomarkers, while we have used low-cost non-invasive markers of physical functioning, which may come into their own for use in field studies, rather with than clinical samples.

Using a novel composite measure of phenotypic age, this study adds to a body of literature showing effects of early adversity on ageing. Our findings show that the effects of early adversity can be detected using non-invasive low-cost ageing markers, even in chronologically young adults. The study also adds to a body of mixed findings around the predictors of delay discounting, suggesting that more investigation is needed to fully understand the interplay between early environment, physical state, and perceptions of current environment in influencing preferences regarding delayed rewards.

Ethical approval

The study had Newcastle University Research Ethics Committee approval (approval number 01208_1). Participants gave informed consent as part of the electronic questionnaire completed on their arrival at the laboratory. Participants gave separate consent for facial photographs to be taken and rated by observers.

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Conflicts of interest

The authors have no conflicts of interest to declare.

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Figure captions

Figure 1. *The predicted relationships between adversity, phenotypic age, self-perceived health & survival odds, and delay discounting.*

Figure 2. *Plots representing the zero-order correlations between A) cSES and phenotypic age ($r = -0.27$, $p < .0001$), B) Phenotypic age and self-rated health ($r = 0.41$, $p < .0001$), C) cSES and self-rated health ($r = -0.31$, $p < .001$), and D) Phenotypic age and delay discounting score ($r = -0.06$, $p = .51$).*

Figure 3. *A summary showing which of the predicted relationships between adversity, phenotypic age, self-perceived health & survival odds, and delay discounting are supported by our data.*