

Credit scores, cardiovascular disease risk, and human capital

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Credit scores are the most widely used instruments to assess whether or not a person is a financial risk. Credit scoring has been so successful that it has expanded beyond lending and into our everyday lives, even to inform how insurers evaluate our health. The pervasive application of credit scoring has outpaced knowledge about why credit scores are such useful indicators of individual behavior. Here we test if the same factors that lead to poor credit scores also lead to poor health. Following the Dunedin (New Zealand) Longitudinal Study cohort of 1,037 study members, we examined the association between credit scores and cardiovascular disease risk and the underlying factors that account for this association. We find that credit scores are negatively correlated with cardiovascular disease risk. Variation in household income was not sufficient to account for this association. Rather, individual differences in human capital factors—educational attainment, cognitive ability, and self-control—predicted both credit scores and cardiovascular disease risk and accounted for ~45% of the correlation between credit scores and cardiovascular disease risk. Tracing human capital factors back to their childhood antecedents revealed that the characteristic attitudes, behaviors, and competencies children develop in their first decade of life account for a significant portion (~22%) of the link between credit scores and cardiovascular disease risk at midlife. We discuss the implications of these findings for policy debates about data privacy, financial literacy, and early childhood interventions.

credit score | cardiovascular disease risk | human capital | consumer finance

What can a person's credit history tell you about their future health? The credit score—a computationally derived metric of a person's financial history—was initially devised to stratify consumers based on the likelihood that they will pay back their debts. The use of credit scores as a screening tool has proven so successful that it has expanded well beyond consumer credit and into our everyday lives. Companies use credit scores to set interest rates for home mortgages and college loans, to tier premiums for car insurance, to price cell phone contracts and cable TV bills, and even to confer eligibility for employment (1). The expansion of credit scores into domains not directly related to creditworthiness raises questions about what personal characteristics underpin credit scores and whether these characteristics constitute legitimate grounds for screening individuals (2, 3). Nowhere is this concern more pronounced than in the application of credit scores to grade health risks. Life insurance companies have begun incorporating credit scores into predictive models of health and longevity (4, 5).

Despite the proven use of credit scoring techniques for facilitating risk assessment across industry, very little is known about the underlying factors that determine why credit scores are so broadly predictive. Some proponents of credit-scoring practices beyond lending argue that the same attributes that determine creditworthiness also guide behavior in other domains of life (6).

Individuals who avoid risky and impulsive decisions, who plan and manage their finances, and who generally organize their lives are more likely to achieve better job performance, safer driving, and better health (7). The credit score, in this light, is a distillation of difficult-to-observe psychological traits and personal background into a prognostic index with high penetrance and wide application (7–9). In the current study we test this hypothesis by examining the association between credit scores and a seemingly distally related indicator: risk for cardiovascular disease. Established evidence that behavioral factors are central determinants of cardiovascular disease (10, 11) leads us to ask whether credit scores and cardiovascular disease risk are related because financial management and health management share a common constellation of contributory personal characteristics (12).

Individuals vary in their education, cognitive abilities, and noncognitive skills such as self-control, planfulness, and perseverance (13). Extensive social science research has documented that these three factors—collectively titled “human capital”—have stable and enduring effects on social and occupational attainment, earnings, and health (14–18). Here we test if the personal characteristics that make up human capital are the same characteristics that underpin credit scores and that account for covariation between credit scores and cardiovascular disease risk. To clarify whether the link between credit scores and cardiovascular disease risk can be attributed specifically to human

Significance

Credit scoring is expanding into domains beyond lending. Today, credit scores are used by employers, utility companies, and automobile insurers to index high-risk behavior. Life insurance companies even incorporate credit scores into actuarial models. This expansion is controversial, as it is unclear what personal attributes credit scores capture. Following 1,000 individuals from birth to midlife, we show that low credit scores predict cardiovascular disease risk. We also show that the reason credit scores are broadly applicable as a risk stratification tool is because they capture enduring histories of human capital, beginning in childhood. These findings call attention to privacy concerns in a surveillance society, the value of financial literacy, and the importance of early childhood intervention to promote healthy aging.

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capital factors, we also examine two alternative hypotheses: (i) credit scores may be linked to cardiovascular disease risk due to recent adverse shocks (such as job loss or illness) that undermine both credit scores and physical health and (ii) credit scores may be linked to cardiovascular disease risk due to a common association with income. We test these hypotheses in the context of the Dunedin (New Zealand) Study, a 1972–1973 birth cohort of 1,037 individuals followed from birth and repeatedly assessed, most recently in 2012, when study members were 38 y old. The cohort has suffered less than 5% attrition and spans the entire range of socioeconomic status so individuals with poor credit scores and/or high cardiovascular disease risk have not selectively dropped out of the study. As the cohort study members approach midlife, many are primed for major property purchases, starting a business, and taking out a life insurance policy. Amid these contingencies, their credit scores weigh heavily on their future life chances.

Results

Are Low Credit Scores Linked to Increased Risk for Cardiovascular Disease? We assessed our study members' credit scores by linking to administrative records acquired from the Veda Company, the largest credit reference agency in New Zealand and Australia (see *Methods* for more details). The credit scores of our study members ranged from 12 to 961 (mean = 675.2; SD = 168.6). We assessed our study members' cardiovascular disease risk using the Framingham cardiovascular risk score, an omnibus measure of cardiovascular disease risk widely used by health care practitioners and insurance companies to predict future morbidity and mortality (19). Each study member's cardiovascular disease risk was computed using risk factors collected during clinical assessments, including total cholesterol, HDL cholesterol, systolic blood pressure, diabetes status, and smoking status (see *Methods* for more details). Cardiovascular risk scores were then translated to a Framingham heart age for ease of communication and interpretation. Because cohort members were all born within a 12-mo period, their chronological age was fixed at 38 y; however, their heart age varied considerably, ranging from 22 to 85 y (mean = 38.5 SD = 8.0). Fig. 1 shows that study members

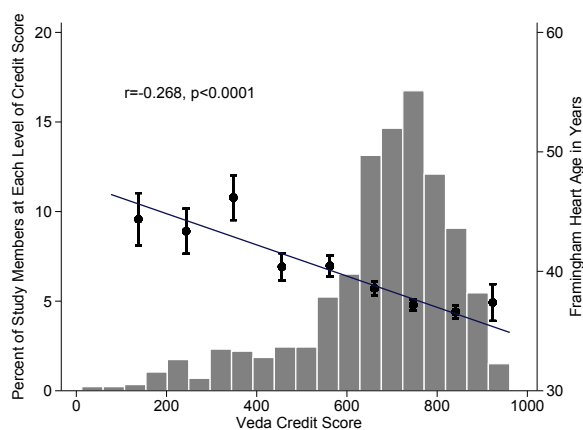


Fig. 1. Association between credit scores and Framingham heart age. The histogram shows the distribution of study members' Veda credit scores (mean = 675.2). The dots and SE bars show average heart age for study members with credit scores <200, 201–300, 301–400, 401–500, 501–600, 601–700, 701–800, 801–900, and >900. All cohort members were aged 38 y at the time of credit reporting and heart age assessment. Heart age ranged from 22 to 85 y (mean = 38.5 y). The regression line shows the association between Veda credit score and Framingham heart age ($r = -0.268$). This association was similar for men ($r = -0.236$) and women ($r = -0.259$). The sex-adjusted association ($r = -0.247$) was used for all subsequent analyses examining the association between credit scores and heart age.

with lower age 38 credit scores were at greater risk for age 38 cardiovascular disease; each 100-point difference in credit score was associated with a 13-mo difference in heart age.

We next turned to test possible explanations for the observed association between study members' credit scores and their heart age.

Do Recent Adverse Shocks Account for the Link Between Credit Scores and Cardiovascular Disease Risk? The association we observed between credit scores and heart age could arise from recent adverse shocks that negatively affect financial and physical well-being. Three such examples are as follows: (i) physical illness impairing job productivity leading to financial distress; (ii) unemployment causing financial distress leading to impaired health; and (iii) extraordinary shocks leading to both financial distress and to impaired health. We tested the degree to which the association between credit scores and heart age was attributable to such adverse shocks. We acquired records from the New Zealand Ministry of Social Development relating to sickness and unemployment claims in the last 6 y and identified study members who were recently seriously ill ($n = 81$) or unemployed ($n = 88$). We also used self-reports to identify study members exposed to a set of exogenous shocks causing massive financial and physical distress (20, 21): the 2010–2011 Christchurch earthquakes ($n = 93$; see *Methods* for details).

On average, study members exposed to any of these recent events ($n = 222$) had credit scores that were 97 points lower ($\beta = -0.25$, $P < 0.001$) and heart ages that were 19 mo older ($\beta = 0.08$, $P = 0.017$), confirming the expected association of recent adverse shocks with impaired financial and physical well-being. A structural equation model (SEM) examining the residual correlation between credit scores and heart age revealed that adjustment for these adverse shocks reduced the correlation between credit scores and heart age by 6% (from $r = -0.247$ to $r = -0.232$; see Figs. S1 and S2 for sensitivity analyses). To test the significance of this reduction we replicated the SEM model just described, but constrained credit-score-to-heart-age covariance to its initial level absent adjustment for recent adverse shocks (covariance = -300.11 ; $r = -0.247$). This constrained model did not result in significantly poorer fit compared with the unconstrained model [$\Delta\chi^2(1)$, $n = 817$] = 0.42, $P = 0.516$ }. This model comparison indicates that, although adverse shocks may have lowered credit scores or exacerbated cardiovascular disease risk, adverse shocks did not account for a significant portion of the cohort's covariance between credit scores and cardiovascular disease risk.

Are Credit Scores and Cardiovascular Disease Risk Linked Because of Income? The association we observe between credit scores and heart age could arise from a common association with income. Health inequalities are not equally distributed across income levels. Also, although income is not a direct input into credit-scoring models, income clearly affects the ability to pay back loans and bills. Under this hypothesis, credit scores may simply represent income differences grading individuals' opportunity to invest in their health and avoid certain health risks. To address this possibility, we collected information about each study member's age 38 household income and examined how adjustment for income affected the correlation between credit scores and heart age. As expected, study members with higher household incomes were more likely to have higher credit scores ($\beta = 0.31$, $P < 0.001$) and younger heart age ($\beta = -0.14$, $P < 0.001$); an NZ \$10,000 rise in household income predicted a 12-point higher credit score and a 3-mo younger heart age. An SEM model examining the residual correlation between credit scores and heart age revealed that the credit-score-to-heart-age correlation was reduced by 15% (from $r = -0.247$ to $r = -0.209$) after adjusting for income (Fig. S2). We tested the significance of this reduction by replicating the aforementioned SEM model, but constraining

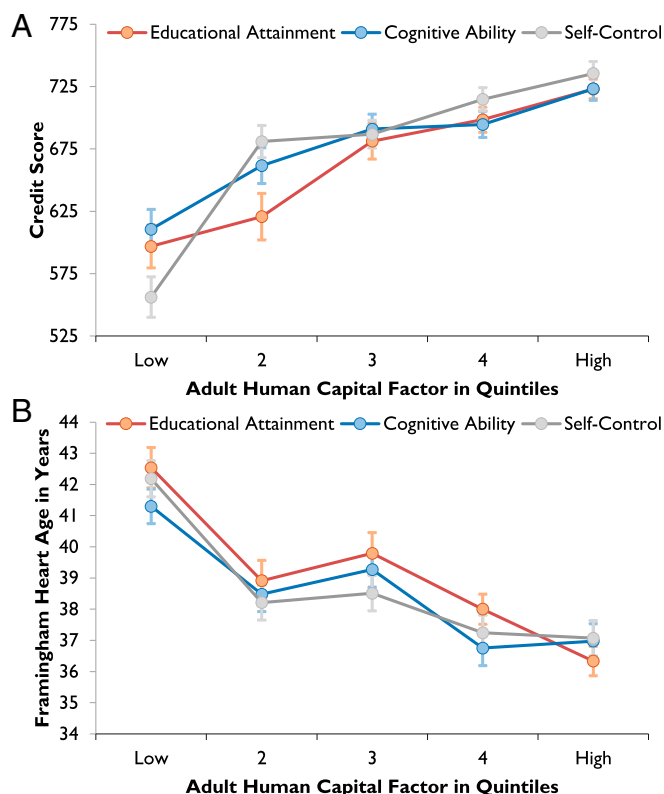


Fig. 2. Associations between adult human capital factors, credit scores, and heart age. Individual differences in study members' educational attainment, adult cognitive ability, and self-control were associated with their (A) credit score (educational attainment: $\beta = 0.28$, $P < 0.001$; cognitive ability: $\beta = 0.24$, $P < 0.001$; self-control: $\beta = 0.37$, $P < 0.001$) and (B) heart age (educational attainment: $\beta = -0.23$, $P < 0.001$; cognitive ability: $\beta = -0.20$, $P < 0.001$; self-control: $\beta = -0.23$, $P < 0.001$). The dots and SE bars show the association between each quintile of a given adulthood human capital factor with credit score and heart age. Quintiles are shown for illustrative purposes; all statistical analyses were performed using continuous measures.

credit-score-to-heart-age covariance to its initial level absent adjustment for household income. This constrained model did not result in significantly poorer fit compared with the unconstrained model [$\Delta\chi^2(1)$, $n = 817$] = 2.23, $P = 0.135$ }, indicating that income did not account for a significant portion of the covariation between credit scores and cardiovascular disease risk. This model was also not significant after simultaneously accounting for both household income and adverse shocks (Fig. S3).

Previous studies have suggested that year-to-year fluctuations in earnings make past-year income a less reliable indicator than permanent income, which is based on a multiyear assessment (22). To account for this possibility, we conducted a sensitivity analysis replacing past-year income with 7-y income, assessed by averaging household income across age 32 and age 38 assessments. This analysis confirmed that income did not account for a significant portion of the covariation between credit scores and cardiovascular disease risk (Fig. S4).

Are Credit Scores and Cardiovascular Disease Risk Linked Due to Contemporaneous Human Capital Factors? Alternatively, the association we observe between credit scores and heart age could arise from a common set of behaviors and competencies that either promote or mitigate prudent financial decision making and health management. Social science research has long recognized the contribution of human capital factors such as

educational attainment, cognitive ability, and self-control as early predictors of health and successful aging. Although no similar program of research identifying the psychological underpinnings of credit scores has yet to develop, the industry-wide use of credit scoring models for stratifying population segments into risk tiers suggests that, in practice, credit scores may already be serving a similar function as a proxy for personal characteristics of prudent decision-making. To test the contribution of human capital factors in explaining the link between credit scores and cardiovascular disease risk, we assessed study members' educational attainment, cognitive ability, and self-control, and then examined the extent to which these human capital factors accounted for covariation between credit scores and cardiovascular disease risk. Educational attainment was assessed based on study members' highest level of education by age 38. Cognitive ability was assessed using IQ tests at the age 38 study assessment. Self-control at age 38 was assessed using personality ratings of conscientiousness, a trait indexing a propensity to be planful, organized, and perseverant (see *Methods* for details).

All three adult human capital factors—educational attainment, cognitive ability, and self-control—predicted both higher credit scores (Fig. 2A) and younger heart age (Fig. 2B). All three human capital factors were also positively associated with income (educational attainment: $\beta = 0.39$, $P < 0.001$; cognitive ability: $\beta = 0.35$, $P < 0.001$; self-control: $\beta = 0.38$, $P < 0.001$); however, multivariate regression models revealed that all three human capital factors were associated with creditworthiness and heart age independent of income (Tables S1–S4).

To examine the role of human capital factors in explaining covariation between credit scores and heart age, we used the same SEM framework we used for adverse shocks and for income, only this time substituting human capital factors (Fig. 3). In contrast to the modest changes in correlation between credit scores and heart age evidenced when adjusting for adverse events and income, adjusting for human capital factors reduced the correlation between credit scores and heart age by 45% (from $r = -0.247$ to $r = -0.136$). As before, we tested the significance of this reduction by constraining credit-score-to-heart-age covariance to initial levels absent adjustment for adult human capital factors.

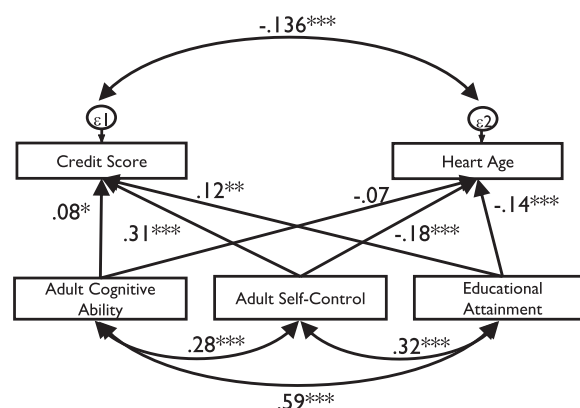


Fig. 3. Effect of adult human capital factors on the covariation between credit scores and heart age. This structural equation model illustrates the role of human capital factors in the correlation between credit scores and heart age. Values associated with the paths represent standardized parameter estimates. Single headed arrows represent causal paths and double-headed arrows represent covariances. Sex (not presented) is included as a covariate with paths to all variables. Comparison with a model where the covariance between credit scores and heart age is constrained to initial levels showed that human capital factors accounted for a significant source of the link between credit scores and heart age. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

extending the use of credit scores to domains beyond lending (2, 3), we find that credit scores modestly but significantly predict risk for cardiovascular disease in a cohort of adults entering midlife. Interestingly, credit scores did not simply act as a proxy for income; instead, the link between credit scores and cardiovascular disease risk was largely explained by study members' level of human capital: their educational attainment, cognitive ability, and self-control. The temporal precedence of human capital in this relationship was supported by our observation that childhood human capital factors assessed in the first decade of life partially explained the covariation between credit scores and heart age. Although childhood human capital did not explain the credit score–heart age connection completely, its contribution was notable, given that 30 y elapsed between predictor and outcome measurements. These findings raise several considerations for policy and future research.

The first consideration relates to the social welfare climate of our New Zealand-based study. Our finding that adverse life events did not significantly attenuate the association between credit scores and heart age should be interpreted within the context of New Zealand's social service programs and universal health care policy. A recent analysis of New Zealand census data indicated that 5% of bankruptcies could be attributed to ill health or the absence of health insurance (26). This figure stands in stark contrast to the United States, where it has been reported that >60% of bankruptcies are due to medical-related expenses (27). The issue of uncollected medical debt damaging credit scores, a growing concern in the United States (28, 29), should be less influential in our New Zealand-based study. Our empirical findings are largely consistent with this assumption. Linking to administrative records of study members' social welfare benefits, we find that the general association between credit scores and heart age could not be explained by study members' recent illness lowering their credit score or recent unemployment raising their cardiovascular disease risk. Furthermore, exposure to the Christchurch earthquakes, examples of extraordinary exogenous shocks, did not materially affect the link between credit scores and cardiovascular disease risk. Nevertheless, our finding that ~45% of the correlation between credit scores and heart age could be accounted for by human capital factors raises the question of what unobserved sources may be contributing to the remaining heterogeneity. Previous research has noted the practical challenges inherent in identifying the myriad situational circumstances (e.g., local market conditions, inaccuracies in credit history reporting, and adverse personal events) that may affect the predictive validity of credit scores (30). International replications of our study are needed.

A second consideration relates to the extension of credit scoring practices to domains beyond lending. Our finding that credit scores predict risk for cardiovascular disease provides empirical support for credit-based scoring techniques in the life insurance industry. Our integration of psychometric assessment to explain the nature of this relationship is a first step in understanding why credit scores and cardiovascular disease risk are linked. Our findings suggest that life insurance companies that acquire an applicant's credit score are also indirectly acquiring information about that applicant's educational attainment, intelligence, and personality, right back to childhood. We suspect that citizens are unaware of the degree to which credit scores provide insight into such private and sensitive domains. Individuals who may never consent to a personality test or an IQ test before applying for insurance are unwittingly consenting to reveal this information when granting access to their credit scores. Credit scoring is an unobtrusive measure that reveals quite a bit about a person. Future debate on the extended use of credit scoring techniques should consider threats to privacy and confidentiality. Interestingly, in developing countries where credit scores are either unobtainable or unreliable, measurement of an

individual's conscientious personality is successfully being used as a substitute for credit scores (31).

A third consideration relates to the timing of interventions to promote financial and physical well-being. We found that a portion of the link between credit scores and cardiovascular disease risk was explained by personal characteristics already present in the first decade of life. Compared with children in the top quintile of self-control, children in the bottom quintile went on to develop heart ages that were on average 4 y older and credit scores that were on average 103 points lower. Other childhood human capital factors had similar effects. Childhood self-control is of particular interest because of evidence that self-control is amenable to intervention early in life, and because economic models suggest that early investment may be more cost-effective than later remediation (32, 33). Our findings suggest that interventions successful at improving children's self-control may benefit their financial and physical well-being decades later in life.

Our life chances often depend on how we are evaluated by others. Today more than ever, many of these evaluations—obtaining a college loan, getting a job interview, and securing a mortgage—are conducted by people we will never meet, using proprietary algorithms to which we are not privy. In these impersonal contexts, credit scores have replaced personal reputational relationships as rationalized gauges of character and competence. Although policy debate about the consequences of expanded credit scoring jumps ahead, our understanding of the psychological factors that lead credit scores to be useful lags behind. In this study, we present evidence that the reason credit scores predict distally related domains such as cardiovascular disease risk is because credit scores capture enduring histories of human capital.

Methods

A more detailed report of the study design and analyses is available in the [SI Text](#).

Dunedin Study Sample. Participants are members of the Dunedin Multidisciplinary Health and Development Study, which tracks the development of 1,037 individuals born in 1972–1973 in Dunedin, New Zealand.

Age 38 Outcomes.

Credit scores. Credit scores were acquired at the age 38 assessment phase from the Veda Company. The Veda credit score algorithm is proprietary. Scores are based on 5-y histories of consumer credit activity and include the following factors: the number and types of credit applications and inquiries, age of credit file, residential stability, adverse information such as payment defaults and judgments, and the existence of any current or prior insolvency information. Factors such as race, national origin, marital status, occupation, salary, employment history, medical or academic records are not included in Veda scoring. Of the 961 study members who participated in the age 38 assessment, 11 did not consent to a credit rating search and 82 were not credit active in the last 5 y (the majority of whom resided overseas) and consequently had reports with no credit history, resulting in 868 valid observations. Eight study members were flagged by Veda as insolvent and were not given a credit score by Veda. We repeated all analyses with these individuals coded to the lowest credit score and with these individuals excluded from analyses. The results were unchanged. Here we report analyses based on the 860 study members with a credit score.

Heart age. Heart age is an estimate of vascular age based on the Framingham cardiovascular disease (CVD) risk score, a single multivariable function that predicts risk of developing all-CVD and its constituents (19). The 10-y CVD risk for each study member was computed using sex-specific factors collected at the age 38 assessment phase including: total cholesterol, HDL cholesterol, systolic blood pressure, diabetes status, and smoking status. Framingham CVD risk was then translated to heart age using the heart age calculators made available by the Framingham group (19). We excluded pregnant study members and study members who did not consent to phlebotomy, resulting in 900 valid observations.

Age 38 Covariates.

Social welfare benefits. We conducted record linkage with the New Zealand Ministry of Social Development to identify study members who drew on

government benefits. Because by New Zealand law, credit scores may only refer back a maximum of 5 y for consumer credit activity, study members who received either unemployment ($n = 88$) or sickness ($n = 81$) related benefits in the prior 6 y were counted as having received a benefit. Only one benefit can be received at any given time.

Exposure to the Christchurch earthquakes. Christchurch, New Zealand, suffered two major earthquakes during the age 38 assessment, which took place in 2010–2011. We used self-reports of study members' residential histories to identify study members living in the Christchurch area at the time of the earthquakes ($n = 93$). Credit scores were retrieved in 2012; thus, the impact of these natural disasters on the financial status of our Christchurch study members would have been captured.

Household income. Following the census (34), study members were asked to list their sources of income and given the choice of 13 different income categories to report their total pretax annual income from all sources. Study members also reported pretax annual income for cohabitating partners/spouses (if relevant). Age 38 household income was the total annual income for the study member and their partner/spouse. The median household income for our New Zealand-based study members was NZ \$82,500 (SD = 41,274). Seven-year household income was assessed by averaging annual household incomes at ages 32 and 38 (median, NZ \$72,500; SD, 31,720).

Age 38 Human Capital Factors.

Educational attainment. Education level was measured on a five-point scale relevant to the New Zealand educational system: 1 = no secondary school qualifications (14.7%), 2 = school certificate (14.9%), 3 = high school graduate or equivalent (14.2%), 4 = postsecondary diploma or certificate (27.3%), 5 = bachelor's degree or higher (28.8%).

Adult cognitive ability. IQ at age 38 was assessed using the Wechsler Adult Intelligence Scale—Fourth Edition (35) (mean = 100, SD = 15).

Adult self-control. Adult self-control was assessed via multiple informant reports of the personality trait of conscientiousness, as previously described (36).

Childhood Human Capital Factors.

Childhood socioeconomic status. Children's socioeconomic status (SES) was measured with a six-point scale assessing the occupational status of study members' parents when study members were children. The scale places each occupation into one of six categories (from 1, unskilled laborer; to 6, professional) on the basis of educational levels and income associated with that occupation in data from the New Zealand census. Childhood SES was the average of the highest SES level of either parent from birth through age 15. This variable reflects the socioeconomic conditions experienced by the participants while they were growing up.

Childhood cognitive ability. IQ was assessed in childhood at ages 7, 9, 11, and 13 using the Wechsler Intelligence Scale for Children—Revised (37) and was averaged across the four childhood assessment phases (mean = 100, SD = 15).

Childhood self-control. Children's self-control during their first decade of life was measured using a multioccasion/multi-informant strategy, as previously described (18). Briefly, the composite score includes nine measures: observational ratings of children's lack of control (3 and 5 y of age) and parent, teacher, and self-reports of impulsive aggression, hyperactivity, lack of persistence, inattention, and impulsivity (5, 7, 9, and 11 y of age).

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Supporting Information

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SI Text

Dunedin Study Sample. Participants are members of the Dunedin Multidisciplinary Health and Development Study, a longitudinal investigation of health and behavior in a complete birth cohort. Study members ($n = 1,037$; 91% of eligible births; 52% male) were all individuals born between April 1972 and March 1973 in Dunedin, New Zealand, who were eligible for the longitudinal study based on residence in the province at age 3 and who participated in the first follow-up assessment at age 3 (1). The cohort represents the full range of socioeconomic status in the general population of New Zealand's South Island and is primarily white. Assessments were carried out at birth and at ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, and, most recently, 38 y, when 95% of the 1,007 Study Members still alive took part. At each assessment wave, each study member is brought to the Dunedin research unit for a full day of interviews and examinations. The Otago Ethics Committee approved each phase of the study, and informed consent was obtained from all study members. It was important to the study that our actions not negatively impact the study members, and as such we negotiated "research only" access to the data, which would not affect individual credit scores.

Statistical Analyses. We used two methods to test the relative contribution of our predictor variables to our age 38 outcome measures (credit scores and heart age): (i) linear regression and (ii) structural equation modeling (SEM).

Linear regression models were used to test the association between predictor variables and each one of our age 38 outcome measures. Standardized β coefficients (denoted as β) are presented to facilitate comparisons across predictor variables. All statistical tests included controls for sex.

SEM models were used to test whether predictor variables accounted for a significant portion of the covariation between our two age 38 outcome measures. SEM models were estimated using STATA 13 (Stata Corp) with direct maximum likelihood. These models are presented in Fig. 3, Figs. S1–S4 (adult models), and Fig. 5 (childhood model). The adult models include paths between age 38 predictor variables to age 38 credit scores and to age 38 Framingham heart age. Predictor variables were allowed to be correlated. Importantly, disturbance terms between credit scores and heart age were also correlated to account for unexplained shared variance. The childhood model (Fig. 5) has the same form as the adult model (Fig. 3) with the exception that adult human capital factors were replaced with their childhood antecedents. For all models, sex was included as an exogenous variable with paths to all observed measures.

SEM significance tests involved comparisons of nested models. Nested model forms were identical to their comparators except that the covariance between credit scores and heart age was constrained to initial levels before the inclusion of predictor variables. Poorer fit of the constrained models would indicate that predictor variables accounted for a significant portion of the covariation between credit scores and heart age. Comparisons of nested models were assessed using χ^2 difference tests, as well as multiple indices of model fit including the comparative fit index (CFI), the Tucker-Lewis index (TLI), and the root mean square error of approximation (RMSEA). The CFI and TLI range from 0 to 1, with 0 indicating the absence of model fit and 1 indicating perfect model fit (2). RMSEA values of less than 0.05 are generally accepted as indicators of good model fit in the social sciences; those between 0.05 and 0.08 are indicative of an adequate model fit (3).

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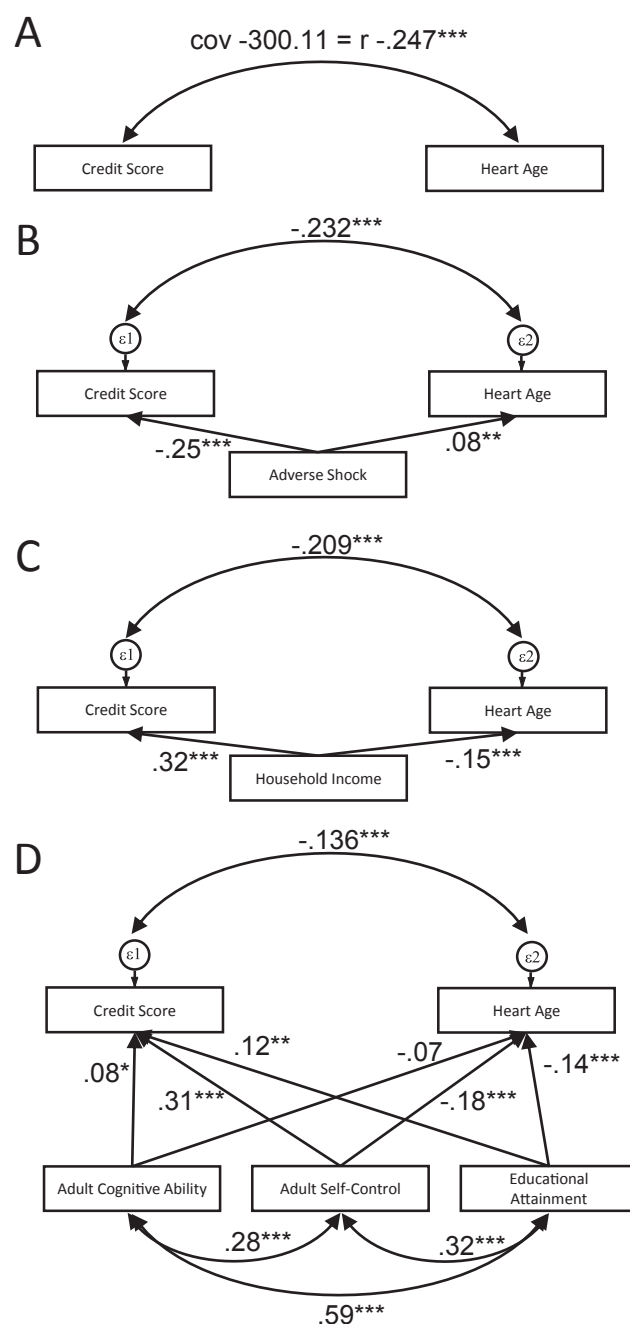


Fig. S1. Effect of income, extraordinary shocks, and adult human capital factors on the covariation between credit scores and heart age. Credit score and heart age are correlated (A). Structural equation models illustrate the roles of adverse shocks (B), income (C), and human capital factors (D) on the correlation between credit scores and heart age. Values associated with the paths represent standardized parameter estimates. Single headed arrows represent causal paths and double-headed arrows represent covariances. Sex (not presented) is included as a covariate with paths to all variables. Comparisons of models B–D to a model where the covariance between credit scores and heart age is constrained to initial levels (covariance of model A) showed that only human capital factors accounted for a significant source of the link between credit scores and heart age. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

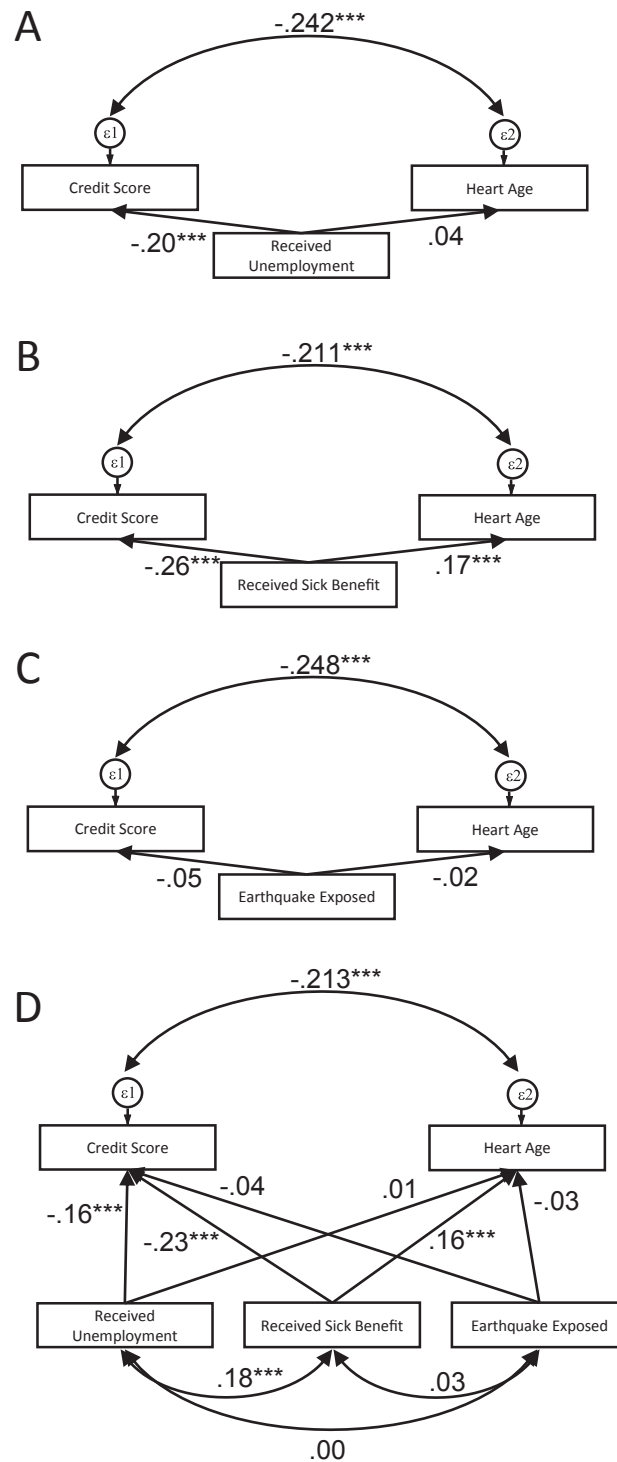


Fig. S2. Effect of extraordinary shocks on credit scores, heart age, and their covariation. The structural equation models presented here illustrate the separate effects of each adverse shock on the covariation between credit scores and heart age: (A) received unemployment benefits, (B) received sickness benefits, and (C) exposed to Christchurch earthquake. D shows that controlling for all three shocks simultaneously lowered the correlation between credit scores and heart age from -0.247 (Fig. S1) to -0.213 , a reduction of 14%. A comparison with a model where the covariation between credit scores and heart age was constrained to initial levels did not result in significantly poorer fit compared with the unconstrained model $\{[\Delta\chi^2(1), n = 817] = 1.93, P = 0.165\}$, indicating that adverse shocks did not account for a significant portion of the covariance between credit scores and heart age. The values associated with the paths represent standardized parameter estimates. Sex (not presented) is included as a covariate with paths to all variables. $*P < 0.05$, $**P < 0.01$, $***P < 0.001$.

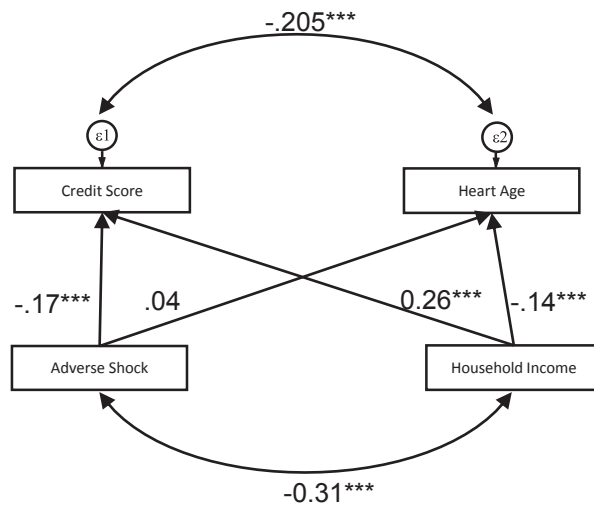


Fig. S3. Combined effect of extraordinary shocks and household income on credit scores, heart age, and their covariation. The structural equation model presented here illustrates the combined effects of adverse shocks and household income on the covariation between credit scores and heart age. Simultaneously controlling for adverse shocks and household income lowered the correlation between credit scores and heart age from -0.247 (Fig. S1) to -0.205 , a reduction of 17%. A comparison with a model where the covariation between credit scores and heart age was constrained to initial levels did not result in significantly poorer fit compared with the unconstrained model $\{[\Delta\chi^2(1), n = 817] = 2.95, P = 0.09\}$, indicating that the combined effects of adverse shocks and household income did not account for a significant portion of the covariance between credit scores and heart age. The values associated with the paths represent standardized parameter estimates. Sex (not presented) is included as a covariate with paths to all variables. $***P < 0.001$.

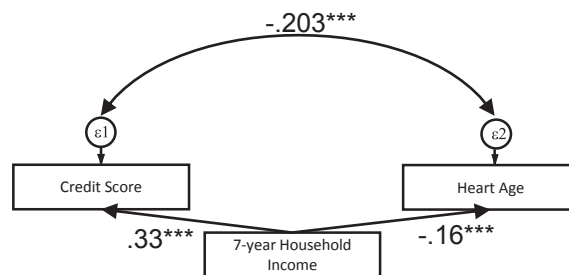


Fig. S4. Effect of 7-y household income on credit scores, heart age, and their covariation. This structural equation model is the same as Fig. S1C, except household income at age 38 has been replaced with 7-y household income across ages 32 and 38. The values associated with the paths represent standardized parameter estimates. Sex (not presented) is included as a covariate with paths to all variables. $***P < 0.001$.

Table S5. Mediation of childhood human capital factors by adult human capital factors for credit scores and heart age

Childhood factor	Total effect				Direct effect				Indirect effect				Proportion of effect mediated
	Coefficient	SE	β	P value	Coefficient	SE	β	P value	Coefficient	SE	β	P value	
Credit score													
Socioeconomic advantage	23.60	5.72	0.16	<0.001	3.76	5.39	0.03	0.49	19.84	1.93	0.13	<0.001	0.84
Cognitive ability	2.50	0.67	0.21	<0.001	0.33	0.66	0.03	0.61	2.17	0.14	0.19	<0.001	0.87
Self-control	39.08	6.82	0.23	<0.001	9.70	6.50	0.06	0.14	29.38	2.05	0.17	<0.001	0.75
Heart age													
Socioeconomic advantage	-1.23	0.26	-0.17	<0.001	-0.44	0.25	-0.06	0.08	-0.79	0.06	-0.11	<0.001	0.64
Cognitive ability	-0.11	0.03	-0.19	<0.001	-0.02	0.03	-0.04	0.51	-0.09	0.00	-0.16	<0.001	0.82
Self-control	-1.48	0.32	-0.18	<0.001	-0.30	0.31	-0.04	0.33	-1.18	0.07	-0.14	<0.001	0.80

We used SEM to test the degree to which the effects of childhood human capital factors on heart age and credit scores were mediated by adult human capital. To account for multiple correlated mediators, each SEM model included a childhood human capital factor, all three adult human capital factors (educational attainment, adult cognitive ability, and adult self-control), and our two age 38 outcome measures (credit scores and heart age). SEM allows for the decomposition of childhood human capital effects into direct effects - paths that lead directly from childhood human capital to heart age and credit scores, and indirect effects, all remaining paths whereby the association between childhood human capital factors and credit scores or heart age are mediated by adult human capital factors. For each childhood human capital factor, the proportion of effect mediated represents the proportion of the total childhood human capital effect that is attributable to adult human capital factors.