The Great Recession worsened blood pressure and blood glucose levels in American adults

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Longitudinal, individual-specific data from the Multi-Ethnic Study of Atherosclerosis (MESA) provide support for the hypothesis that the 2008 to 2010 Great Recession (GR) negatively impacted the health of US adults. Results further advance understanding of the relationship by (*i*) illuminating hypothesized greater negative impacts in population subgroups exposed to more severe impacts of the GR and (*ii*) explicitly controlling for confounding by individual differences in age-related changes in health over time. Analyses overcome limitations of prior work by (*i*) employing individual-level data that avoid concerns about ecological fallacy associated with prior reliance on group-level data, (*ii*) using four waves of data before the GR to estimate and control for underlying individual-level age-related trends, (*iii*) focusing on objective, temporally appropriate health outcomes rather than mortality, and (*iv*) leveraging a diverse cohort to investigate subgroup differences in the GR's impact. Innovative individual fixed-effects modeling controlling for individual-level age-related trajectories yielded substantively important insights: (*i*) significant elevations post-GR for blood pressure and fasting glucose, especially among those on medication pre-GR, and (*ii*) reductions in prevalence and intensity of medication use post-GR. Important differences in the effects of the GR are seen across subgroups, with larger effects among younger adults (who are likely still in the labor force) and older homeowners (whose declining home wealth likely reduced financial security, with less scope for recouping losses during their lifetime); least affected were older adults without a college degree (whose greater reliance on Medicare and Social Security likely provided more protection from the recession).

recession | blood pressure | glucose | economic stress

Few Americans were untouched by the Great Recession (GR), the most significant economic upheaval since the Great Depression of the 1930s (1). Though one might expect major economic downturns to negatively affect physical health, the evidence is, in fact, mixed. Whereas some influential studies that exploit natural experiments conclude that economic downturns improve health (2–6), others draw the reverse conclusion (7, 8).

Uniquely rich data from the Multi-Ethnic Study of Atherosclerosis (MESA) are used to test the hypothesis that the GR negatively affected health by exploiting four waves of pre-GR longitudinal data, collected between 2000 and 2007, and a fifth wave, collected after the onset of the GR in 2010 to 2011. The 2008 GR was a major shock: Its timing and depth were largely unanticipated (9), and resulted in levels of aggregate economic stress not experienced since the Great Depression. Few Americans were left untouched by the GR: Over 70% of Americans aged 40+ reported they were affected by the shock (10). Its impact varied substantially across the US population. After many years of rapid increases in house prices, those who purchased homes a few years before the onset of the recession lost much if not all of their savings; many lost their homes. Unemployment rocketed, with increases being especially large among minorities and those with less education.

This research addresses several key limitations in the literature on health impacts of economic downturns. First, attempts to reconcile existing evidence (11) have concluded that macro- and individual-level analyses have failed to capture heterogeneous impacts across demographic and socioeconomic subgroups (2–6, 12). Using individual-level health data, we investigate impacts on subgroups differentially exposed to the economic downturn.

Second, studies linking health with individual experiences of a recession, such as job loss, may not uncover causal relationships, since individuals who lose a job, even from plant closings, are unlikely to be statistically exchangeable with those who do not (13, 14). Similar concerns arise for those who lost a home in the GR, since they are more likely to have borrowed beyond their means. To address this concern, we take an intent-to-treat approach and link post-GR changes in health trajectories to membership in population subgroups with more or less exposure to economic stresses from the GR. For example, contrasts are drawn between people who did and did not own a home pre-GR and differences are interpreted as the average effect on the more exposed group, namely homeowners, because homes are the most valuable asset owned by many Americans and the collapse of the housing market is a defining feature of the GR. Moreover, while homeowners are treated as a group, our models take into account fixed, individual-specific traits that affect health, and we compare offsets in health markers post-GR relative to pre-GR trends that are individual-specific.

Third, many prior studies have relied on mortality, an incomplete indicator of the full range of potential health effects that, apart from harvesting of the frailest, is unlikely to reflect

Significance

Longitudinal data from the Multi-Ethnic Study of Atherosclerosis (MESA) document deleterious health impacts of the economic and social stresses associated with the Great Recession, with significant increases in blood pressure and glucose. Effects are independent of underlying age-related trends in these biomarkers. Larger effects are seen in population subgroups more severely hit by the recession: younger adults (i.e., those more likely still in the labor force) and older homeowners (whose declining home wealth likely reduced a key element of their financial security). Results also reveal greater impacts on blood pressure and glucose among those on medication, and a concomitant reduction in medication use and intensity of treatment—a potential pathway by which the Great Recession affected people on medications more.

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Data deposition: The data and analysis code used for these analyses are available via the MESA website (https://www.mesa-nhlbi.org).

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impacts of recent economic changes (12, 15, 16). We focus instead on objectively measured health markers, namely blood pressure and fasting glucose, whose physiological regulatory processes are known to function as important contributors to major health conditions such as heart disease and are responsive to stressful conditions such as those experience by many during the GR (17–19).

Fourth, few prior studies have disentangled age-related health changes from effects of economic downturns. Using four measures covering 8 y pre-GR, we estimate individual-specific trends in each health marker and identify the offset in the trend attributable to GR-related stresses. In combination, these innovations provide estimates of the GR's impact on important health parameters that can credibly be interpreted as causal. We also provide some evidence suggesting mechanisms at play.

We build on recent research that shows individuals with diabetes who presented at a clinic after the onset of the GR have worse glycemic control than those who presented pre-GR (20). Using five waves of longitudinal data on both glucose and blood pressure in US adults collected since 2000 in sites across the United States we test the hypothesis that the GR affected health status and, by exploiting the cohort diversity, investigate the heterogeneity of those impacts. We contrast older adults (≥65 y) who have health insurance through Medicare and income from Social Security and pensions with younger adults who are potentially more exposed to job loss, loss of health insurance, and possibly loss of wealth, since most rely on defined contribution pension savings invested in the stock market. These contrasts are enriched by also separating those with and without a college degree, since less educated older adults likely rely largely on Social Security and Medicare and would be least exposed to the negative impacts of the GR whereas unanticipated reductions in wealth are likely to be more salient sources of stress from the GR for those who have a college degree. Since a defining feature of the GR was the collapse of housing values and the foreclosure crisis, we complement these indirect comparisons of exposure with a more direct indicator of exposure to stress by contrasting individuals who did and did not own a home before the GR. The last time MESA measured homeownership pre-GR was in 2004 to 2005, well before the onset of the GR, and thus unlikely to be related to endogenous asset reallocations in anticipation of the recession. In the absence of detailed information about pre-GR home values, wealth, or occupation, homeownership is our best direct marker of exposure to the deleterious impact of the recession.

Results

For the MESA cohort overall, age at the time of the GR (i.e., in 2008) ranged from 50 to 91 with an average age of 66.7 y. Educational attainment was relatively high, with 39.4% reporting having completed a college education and 71.4% reporting being a homeowner 2004 to 2005. Average levels of BP and glucose parameters were higher among those aged 65 and older. With the exception of mean arterial pressure (MAP), patterns of change from 2000 to 2010–2012 generally reflected greater increases in blood pressure and glucose levels for those aged less than 65. Notably, in all cases, patterns of change from 2000 to 2010–2012 also show relatively small average changes but, as indicated by the SDs, wide individual variability within both age groups, though the variability is greater in the older age group. Medication use rises with age. About one in five younger respondents is on hypertension medication at baseline, with twice that fraction among older respondents. During the 10 y of the study, another one in five goes on medication and so, by 2010 to 2012, almost two-thirds of older adults are on hypertension medication. Fewer respondents are on medication for glucose control, although those rates also rise substantially during the study period so that about one in six respondents reports being on medication. See Table S1 for additional descriptive statistics.

Measured Blood Pressure and Glucose Levels. Estimates of the effect of the GR are reported in Table 1. Each cell reports the difference, or offset, between the biomarker post-GR and its predicted level based on a fixed-effects linear regression of the trajectory of changes pre-GR that allows each trajectory to be individual-specific. The offsets are multiplied by the median time since the baseline measure (3,429 d). Results are reported separately by age at the onset of the GR in 2008 (<65 vs. >=65) and by medication status [on vs. not on medication at examination 5 (2010 to 2012)], as analyses indicated differences in the patterns of change from pre- to postrecession by age and by medication status.

For all blood pressure outcomes, those on medication, regardless of age, show significant increases in systolic blood pressure (SBP), pulse pressure (PP), and MAP. Tests for age differences also indicate that the increases are significantly larger for those aged less than 65 (e.g., increases of 12.7 vs. 7.9 mmHg in SBP for those <65 vs. >65+, with parallel increases of 8.1 vs. 5.8 mmHg in PP and 7.2 vs. 4.1 mmHg in MAP). By contrast, for those not on medication, there are similar increases from pre- to post-GR for both age groups for SPB and PP. MAP, however, shows a significant increase for the younger age group, and is the

Table 1. Estimates of the impact of the Great Recession on blood pressure and glucose: Offsets relative to the postrecession level predicted by individual-specific prerecession aging trends, stratified by age and medication status

Outcomes	On medication postrecession			Not on medication postrecession		
Age in October 2008	<65 y	≥65 y	Effect difference (<65 − ≥65)	<65 y	≥65 y	Effect difference (<65 − ≥65)
Offset [SE] in blood pressure, postrecession, mmHg						
Systolic blood pressure	12.65	7.93	4.72	4.45	2.85	1.60
	[1.27]*	[0.97]*	[1.59]*	[0.60]*	[0.90]*	[1.08]
Pulse pressure	8.12	5.76	2.35	3.84	3.55	0.29
	[0.85]*	[0.70]*	[1.10]*	[0.43]*	[0.68]*	[0.80]
Mean arterial pressure	7.20	4.12	3.07	1.92	0.47	1.45
	[0.80]*	[0.56]*	[0.98]*	[0.39]*	[0.52]	[0.65]*
Offset [SE] in log (blood glucose), postrecession (log mg/dL), scaled by 100						
Log blood glucose	10.19	5.76	4.43	1.49	0.59	0.91
	[4.30]†	[3.39]	[5.48]	[0.43]*	[0.47]	[0.64]

All models include controls for time-varying covariates and individual-specific fixed effects (for the rate of change in the biomarker since baseline). SEs reported in brackets are robust to heteroscedasticity. Changes in log glucose, scaled by 100, can approximately be interpreted as percentage change. The approximation is better the smaller the change; approximation errors are typically in the third significant digit. Thus, a change in log glucose of +10.19 translates to a 10.73% increase.

*$P < 0.05$ (before and after Hommel adjustment for multiple testing across the tests in Tables 1 and 2).

†$P < 0.02$ before Hommel adjustment; $P < 0.09$ after Hommel adjustment.

only blood pressure index to exhibit a significant age difference for those not on medication.

For fasting glucose, significant increases from pre- to post-GR are seen among the younger age group, although the effect for those on medications is not statistically significant after adjusting for multiple testing. Though smaller, the increases seen in the older age group are not significantly different from those in the younger group.

Medication Use. We next examined how medication use and intensity of treatment were affected by the GR. Incidence rate ratios (IRRs) from fixed-effects Poisson regression models for medication use (any use and intensity) are presented in Table 2. For medication use, the IRR is interpreted as the ratio of medication use prevalence post-GR to pre-GR and, for medication intensity, the IRR is the ratio of medication intensity on an ordinal intensity scale post-GR to pre-GR. Results for BP medications reveal a significant, positive annual trend for use of medication and for increases in the intensity of treatment (number of classes of BP medications used) over time in both age groups (Table 2). For glucose, there are also positive trends in use and intensity of medications, with larger increases among the younger age group (Table 2).

Notably, the patterns of change from post-GR reveal a decline in medication use relative to pre-GR trends. After the onset of the GR, there is a significant decline of 17% (IRR 0.83) in use of BP medications among older adults and significant declines in the number of classes of medications used, 24% among older adults and 14% among younger adults.

Medication use for diabetes displays a similar pattern of decline. In both age groups, there are significant declines in use of any medication and in the intensity of medications being used. The relative declines are approximately three times larger among younger relative to older adults. However, the differences between age groups were statistically significant only for medication intensity.

Differences by Education and Homeownership. The economic impacts of the GR varied dramatically across the US population. To more fully investigate its impact on health, additional analyses explored differences by education and homeownership.

Results for medication use are shown in Table 3. There is no evidence for differences by education but there are differences by homeownership, with older homeowners reporting a larger reduction in intensity of treatment for hypertension than older individuals who did not own homes.

In contrast, as shown in Table 4, analyses of actual levels of BP and glucose reveal several differences by education and homeownership in the patterns of change from pre- to post-recession. For BP, educational differences are only evident among older adults on medication, with significantly larger increases among those who completed college relative to those who did not. Results for mean arterial blood pressure illustrate this pattern (details for the other BP outcomes can be found in Table S2). Among younger adults on medication and those not on medication, there are no differences by education. For glucose, the only significant educational difference is among the younger adults on medication, where the less educated show a significant and very large percentage increase in glucose—a pattern not evident for the more educated.

Analyses by homeownership reveal the largest effects among older adults on medication, where homeowners had significantly greater increases in MAP and glucose (Table 4). There are no significant differences between homeowners and nonowners in the younger group on medication and in both age groups not on medication.

Discussion

Findings from analyses of longitudinal data from before and after the GR are consistent with the hypothesis that stresses surrounding the onset of the GR in 2008 had a deleterious effect on the health of US adults as indicated by two major, objective health indicators, BP and glucose. Importantly, as discussed further below, results indicate greater impacts among population subgroups that were more likely to be deleteriously impacted by the GR (e.g., younger adults who were more likely to be in the labor force and older, more highly educated adults who were more likely to own homes and to have investment income). The latter finding is further substantiated by contrasts between homeowners and nonowners, wherein older homeowners had significantly larger post-GR increases in BP and glucose. Confidence in these findings is strengthened by the fact that five waves of longitudinal measures of BP and glucose spanning the period 2000 through 2012 from the MESA study are analyzed using empirical methods designed to take into account individual-specific prerecession trends.

BP and fasting glucose show patterns of substantial and significant increases post-GR. The increases are greater for those on medication at the time of assessment, indicating greater vulnerability to economic stress in individuals on medications, a vulnerability indexed by the fact that these people had shown evidence of BP and glucose dysregulation to an extent that required medication. In addition, the onset of the recession was accompanied by a substantial decline in medication use and treatment intensity for control of both blood pressure and glucose—a pattern that runs counter to the expected increased use of medication with age/time. These shifts in medication use and treatment intensity, alone, cannot explain the worsening of the health indicators, and the evidence suggests that the stresses of the GR took their greatest toll on those who are on medication.

Table 2. Estimates of the impact of the Great Recession on medication use for blood pressure and glucose: Ratio, post- to prerecession, adjusted for time trend

Medication use	Any medication			Medication intensity		
Age in October 2008, y	<65	≥65	Effect ratio (≥65/<65)	<65	≥65	Effect ratio (≥65/<65)
Blood pressure medications						
Postrecession	0.94 [0.04]	0.83 [0.02]*	0.88 [0.04]*	0.86 [0.04]*	0.76 [0.02]*	0.88 [0.05]*
Time trend (current use relative to last year)	1.08 [0.01]*	1.06 [0.00]*	0.99 [0.01]	1.10 [0.01]*	1.09 [0.00]*	0.99 [0.01]
Glucose medications						
Postrecession	0.71 [0.07]*	0.87 [0.05]*	1.22 [0.13]	0.70 [0.06]*	0.89 [0.05]*	1.26 [0.13]*
Time trend (current use relative to last year)	1.16 [0.02]*	1.07 [0.01]*	0.93 [0.02]*	1.16 [0.02]*	1.07 [0.01]*	0.92 [0.02]*

Poisson fixed-effects incidence rate ratios and robust SEs are reported in square brackets.
*$P < 0.05$ for IRRs different from 1.0 (after Hommel adjustment for multiple testing across the tests in Tables 1 and 2).

Table 3. Estimates of the impact of the Great Recession on medication use by education and homeownership: Ratio, post- to prerecession, adjusted for time trend

Outcomes by education	On any medication		Medication intensity		Outcomes by homeownership	On any medication		Medication intensity	
Age in October 2008, y	<65	≥65	<65	≥65	Age in October 2008, y	<65	≥65	<65	≥65
Blood pressure									
Not completed college	0.96	0.81	0.85	0.75	Not own home	0.96	0.87	0.88	0.85
	[0.05]	[0.02]*	[0.05]*	[0.03]*		[0.08]	[0.04]*	[0.08]	[0.04]*
Completed college	0.91	0.85	0.88	0.77	Own home	0.94	0.81	0.86	0.72
	[0.07]	[0.04]*	[0.07]	[0.04]*		[0.05]	[0.02]*	[0.05]*	[0.02]*
Effect ratio†	0.95	1.04	1.03	1.03	Effect ratio‡	0.98	0.92	0.97	0.85
	[0.09]	[0.06]	[0.10]	[0.06]		[0.09]	[0.05]	[0.10]	[0.05]*
Glucose									
Not completed college	0.77	0.84	0.74	0.86	Not own home	0.69	0.78	0.63	0.80
	[0.09]*	[0.05]*	[0.08]*	[0.05]*		[0.10]*	[0.07]	[0.09]*	[0.07]*
Completed college	0.58	0.95	0.61	0.98	Own home	0.72	0.92	0.73	0.93
	[0.11]*	[0.13]	[0.10]*	[0.13]		[0.09]	[0.06]	[0.09]*	[0.07]
Effect ratio†	0.76	1.13	0.82	1.14	Effect ratio‡	1.04	1.18	1.16	1.17
	[0.16]	[0.17]	[0.16]	[0.17]		[0.20]	[0.13]	[0.21]	[0.13]

Poisson fixed-effects incidence rate ratios and robust SEs are reported in square brackets.
*$P < 0.05$ for IRRs different from 1.0.
†Not completed college vs. completed college.
‡Not own home vs. own home.

Differences by age, educational attainment, and homeownership provide additional insights by exploiting two defining features of the GR. First, the collapse of the housing and stock markets resulted in dramatic declines in the wealth of those who owned housing and those who were invested in the stock market, including those invested through a retirement account. With homeownership at historically high levels and the shift out of company-provided retirement plans, many older adults were exposed to increases in economic insecurity that were both unanticipated and unprecedented in almost a century. Second, earnings opportunities of those in the labor market were negatively affected as the labor market froze, job insecurity spiraled, and real wages declined for many workers.

For those on medication, BP rose most for the younger cohort, irrespective of level of education, all of whom were more likely in the labor market and concerned about retirement in the coming years. BP also rose significantly for those in the older cohort who were better educated and so more likely invested in the stock market and owned their home (among the better educated, 78% owned their home but only 59% among the less educated owned their home). The effects were substantially and significantly smaller for one group on medication: those age at least 65 at the start of the GR who had not completed college. They are the least likely to be working and most likely to rely on Social Security for income rather than a retirement plan invested in the stock market; these people were, therefore, likely to be the least affected by the recession among the four demographic groups.

Table 4. Estimates of the impact of the Great Recession on mean arterial pressure and glucose by age (in October 2008), education and homeownership (prerecession): Offsets relative to the postrecession level predicted by individual-specific prerecession aging trends

	On medication postrecession			Not on medication postrecession		
Outcomes	**<65 y**	**≥65 y**	**Effect difference (<65 − ≥65)**	**<65 y**	**≥65 y**	**Effect difference (<65 − ≥65)**
Education						
Mean arterial pressure postrecession offset, mmHg						
Completed college	7.04*	6.78*	0.27	2.30*	1.62*	0.68
Not completed college	7.33*	2.79*	4.54*	1.63*	−0.35	1.98*
Difference: completed vs. not	−0.29	3.99*		0.67	1.97	
log (blood glucose) postrecession offset (log mg/dL), scaled by 100						
Completed college	−3.07	3.91	−6.98	1.20	1.01	0.19
Not completed college	15.75	5.99	9.76	1.67	0.24	1.43
Difference: completed vs. not	−18.81*	−2.07		−0.48	0.77	
Homeownership						
Mean arterial pressure postrecession offset, mmHg						
Own home	7.59*	4.93*	2.66*	2.14*	0.10	2.04*
Did not own home	6.33*	2.33*	4.00*	1.56*	1.81	-0.25
Difference: own vs. not own	1.26	2.60*		0.58	−1.71	
log (blood glucose) postrecession offset (log mg/dL), scaled by 100						
Own home	8.76	11.11*	−2.35	1.28*	0.40	0.88
Did not own home	11.32*	−2.84	14.17*	1.74*	1.01	0.73
Difference: Own vs. not own	−2.56	−13.95*		−0.46	−0.61	

*$P \leq 0.05$. All models include controls for time-varying covariates and individual-specific fixed effects. See Table S2 for all models.

Direct examination of prerecession homeownership confirms that it is the older nonhomeowners who show the smaller pre- to postrecession changes. The largest (and only statistically significant) differences in the post-GR offsets by homeownership are seen (in MAP) for older adults on medication—possibly due to older adults' greater potential need for the value of this asset in retirement as well as their likely shorter time window to recoup housing value losses because of their older age.

Similar patterns are apparent for BP for those not on medication, but the effect sizes are smaller and the differences among population subgroups are largely insignificant. Given that the MESA cohort was 55 or older by the time of the recession (i.e., old enough that high BP is common), those not on medication to control such dysregulation may represent a group generally more resistant to the "wear and tear" on such regulatory systems occasioned by life stressors and thus the smaller changes pre- to postrecession.

Patterns for glucose by education and homeownership for those not on medications are similar to the patterns for BP for those not on medication, with significant increases seen among the younger cohort, again irrespective of education or homeownership. The pattern for glucose among those on medication differs from that for BP, with significant increases seen only among younger adults who do not have a college education and among older adults who own a home. These increases are very large: 11 to 15% relative to each individual's prerecession trend, possibly reflecting the extent to which these groups were (*i*) more vulnerable with respect to glucose regulation, being on medication even before the recession, and (*ii*) among the most heavily impacted by (*i*) unemployment among the younger cohort [as unemployment rates climbed 16% for those who did not complete high school while never exceeding 5% for those who completed college (21)] and (*ii*) housing market upheavals among the older cohort.

Importantly, the evidence suggests that changes in medication do not fully explain the results for BP and glucose regulation, as there is no evidence that changes in the use of medication or treatment intensity after the onset of the GR differ between those with and without a college education, though there is greater reduction in treatment intensity among older homeowners.

There are several key strengths of the present analyses relative to the existing literature. First is the longitudinal design of MESA, which has collected data from the same respondents before and after the onset of the Great Recession over a span of 10 y. Second, the research uses validated and consistently measured objective health indicators, blood pressure and fasting glucose, in combination with carefully collected information about medication use at each survey wave. Third, the empirical strategy is designed to fully exploit the survey data by using four waves of data collected between 2000 and 2008, before the recession, to estimate prerecession trends in the biological parameters for each respondent and compare projected levels in those parameters with the observed values measured in the postrecession wave of MESA. In so doing, the models include individual fixed effects which take into account all unobserved heterogeneity that is fixed over time for each respondent and thus focuses the spotlight on the impact of the GR. Fourth, the diversity of the MESA cohort with age and socioeconomic status (education, homeownership) has been leveraged to examine population variability in health outcomes and link those to variation in the rise in economic insecurity from the GR (e.g., comparing changes for younger vs. older adults, who likely vary in their involvement in the labor force, and for homeowners vs. nonowners, who were differentially invested in the housing market).

Limitations to the current analyses include the absence of more than one wave of data post-GR and the relatively short period of follow-up after the onset of the recession, and the limited number of health parameters for which longitudinal data are currently available. Future research will address both of these limitations. First, efforts are under way to secure funding to allow assays of stored longitudinal samples to augment the range of biological parameters to include other known health risk factors such as markers of inflammation. Second, the longer-term impacts of the recession will be examined when additional MESA data become available, including a wave collected in 2016 and 2017. With longer-term post-GR follow-ups there will also be opportunities to examine pre- vs. post-GR trends for more downstream health events, including major cardiovascular events and mortality.

Our findings provide support for the hypothesis that the economic and social stresses associated with the GR had a deleterious impact on adult health as seen in increases in BP and glucose. Importantly, the patterning of the health changes shows that the worst of the health impacts is among subgroups likely more negatively impacted by the Great Recession: younger adults (who are most likely still in the labor force) and better educated older homeowners (who are most likely invested in the stock market as well as their home). Direct examination of homeownership confirms that older homeowners indeed had the largest increases in BP and glucose from pre- to post-GR. The smallest effects were seen for less educated and nonhomeowning older adults (i.e., those no longer in the labor force and without "investment" in the housing market). The findings also point to significantly greater impacts among those on medication—possibly because prior medication use serves as an indication of a population subgroup with preexisting physiological vulnerability to stress. The unique strengths of the available longitudinal MESA data, allowing for controls for individual-level pre-GR age-related trajectories, serve to strengthen confidence in these findings. The findings themselves underscore the fact that economic upheavals such as the GR not only result in deleterious economic consequences that impact some population subgroups more than others but that those same population subgroups shoulder more deleterious health impacts as well.

Methods

Participants. MESA is a prospective cohort study of the determinants of subclinical cardiovascular disease in a multiethnic, population-based sample of men and women. Participants (n = 6,814) were recruited in 2000 at ages 45 to 84 y from six large geographical areas in the United States, centered around Baltimore; Chicago; Forsyth County, NC; Los Angeles; New York; and St. Paul. The baseline examination took place between July 2000 and August 2002; examination 2 from September 2002 to February 2004; examination 3 from March 2004 to September 2005; examination 4 from September 2005 to May 2007; and examination 5 from June 2010 to April 2012. Details of the study design and recruitment for MESA have been published (22).

Our analytical sample consisted of all MESA participants who completed the baseline MESA examination and examination 5 (n = 4,599). Those excluded from the study sample tended, at baseline, to have lower income and education. Conditional on age and sociodemographic characteristics, those who were not measured in examination 5 tended to be in worse health at baseline, as indicated by higher blood pressure and glucose levels. The analytical sample was, therefore, in slightly better health, at baseline, relative to the entire MESA sample, and so if the GR exacerbated health inequalities, we will tend to understate its impact.

Outcomes. Main outcomes for this study are measured blood pressure and blood glucose levels as well as changes in the use of antihypertensive and antiglycemic medications. To capture intensity of treatment, we examined a count of the number of classes of antihypertensive medications used (angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta blockers, calcium channel blockers, diuretics, vasodilators). For glucose control, we examined, first, whether or not any antiglycemic medication (oral or insulin) is used and, second, an ordinal variable to capture intensity of treatment: no medication, oral antiglycemic medications only, and insulin use (with or without oral medications). (For additional details, see *Supporting Information*.)

Covariates. All models include a fixed effect for each individual to take into account all time-invariant factors that might affect the trajectory of health outcomes during the study period. Potential time-varying confounders included in the models are employment status, income, and medication use. (For additional details, see *Supporting Information*.)

Analyses.

Analysis of measured blood pressure and glucose levels. In an effort to isolate the causal impact of the GR for each biomarker outcome, the analytical design contrasts an individual's status at the time of the postrecession MESA assessment with their prerecession assessments for that outcome, allowing for individual-specific trajectories (growth rates) of the outcome. Specifically, each biomarker, θ [i.e., SBP, pulse pressure, MAP, log(glucose)], is transformed to create the dependent variable, y, in each model that reflects the effective rate of change in the biomarker from baseline to examination w. That is, $y = (\theta_w - \theta_1)/(t_w - t_1)$, where t_w is the date of the examination w and $(t_w - t_1)$ is time elapsed since baseline. Every regression model includes an individual-specific fixed effect which allows each individual to have his/her own linear aging trend while also taking into account all individual-level unobserved heterogeneity (i.e., confounding) in the aging trend (growth rate) that is fixed over the 10-y time span of the MESA assessments and affects the rate of growth of the biomarker. Such characteristics include, for example, the respondent's genetic background, childhood environment, medical history, prestudy lifestyle, intrinsic health, and his/her propensity to invest in health and seek health care, as well as socioeconomic status at study baseline.

An indicator identifying postrecession assessments in the models spotlights the change in trajectory for an individual relative to his/her prerecession trajectory. This is our best estimate of what the individual's trend would have been in the absence of the GR.

Models also included interactions between the indicator for any medication use (yes/no) at examination 5 and the effect of the recession to estimate effects of the recession separately for those on and not on medications.

Analysis of changes in medication use. To examine the effect of the GR on medication use (yes/no) and intensity of therapy (ordinal), the medication variables were themselves examined as dependent variables in Poisson regression models that also included individual fixed effects, a linear aging effect (captured by the number of years elapsed since the baseline examination), and the time-varying covariates listed above.

All analyses (for both biomarkers and medication use) are stratified by age (<65, 65+), as preliminary analyses indicated that age was associated with differential impacts of the GR. Supplementary analyses were further stratified by homeownership in light of the dramatic impacts of the GR on the housing market; those reporting owning a home free and clear or paying a mortgage were counted as "homeowner"; nonowners were those reporting renting or other as of examination 3 (the latest wave with information on homeownership before the GR; $n = 115$ missing data at examination 3 set to nonmissing values for closest earlier examination). Possible gender differences were also considered but no significant differences were found in effects on our health outcomes so results are presented for men and women combined.

All SEs and test statistics are calculated using the robust, Huber–White sandwich estimator. To avoid false discovery from multiple testing, we used the Hommel adjustment to keep the familywise type I error under 5% for the family of 24 tests (for four biomarker outcomes in four age-by-medication status strata, plus four medication outcomes in two age strata), and report the results before and after the multiple testing adjustment (23).

Ethical approval. Data collection for all waves of the MESA study was covered by formal human subject approvals from all institutions involved in the data collection. All participants provided written consent before participation in each wave of data collection.

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