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Economic distress, obesity, and the rise in pain

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ABSTRACT

Growing obesity may have contributed to widening socioeconomic disparities in pain in the US, but some researchers have suggested that deteriorating social and economic conditions among less advantaged Americans could be the root cause. We evaluated whether widening socioeconomic disparities in pain are associated with growing economic distress, particularly among those with low socioeconomic status (SES). We also assessed whether the link between economic distress and pain is mediated by obesity. Using data from nationally-representative samples targeting Americans aged 25–74 in 1995-96 ($N = 3034$) and 2011–14 ($N = 2598$), we fit a structural equation model to estimate the contributions of economic distress and obesity to period changes in the SES disparity in different types of pain. Socioeconomic disparities in backaches and joint pain widened substantially over recent decades, although there was no significant widening for headaches. Economic distress accounted for 34% of SES widening for backaches and 41% for joint pain, but the effect was largely independent of obesity. There was little evidence that economic distress led to obesity, which in turn fueled a rise in pain. Obesity alone explained another 8% of the widening SES disparity in backaches and 17% for joint pain. Economic distress played a larger role than obesity because economic distress increased over time for those with low SES whereas it decreased slightly for those with high SES. In contrast, obesity grew at all levels of SES, albeit more for those with low SES. Unfortunately, we cannot establish the direction of causation. Our model assumes that economic distress and obesity affect pain, but it is also possible that pain exacerbates obesity and/or economic distress. If SES disparities in pain continue to widen, it bodes poorly for the overall well-being of the US population, labor productivity, and the prospects for these cohorts as they reach older ages.

1. Introduction

There has been a notable rise in pain in the US over recent decades (Zajacova et al., 2021a; Zimmer and Zajacova, 2020). Yet, rising pain has been disproportionately concentrated among disadvantaged Americans, thus widening the socioeconomic disparity in pain (Case et al., 2020; Cutler et al., 2020; Glei and Weinstein, 2021; Zajacova et al., 2021a). Some evidence suggests that increasing levels of obesity account for some of the growing disparity in pain (Glei et al., 2021). Others have argued that widening disparities in pain are a consequence of deteriorating social and economic conditions faced by less-educated Americans (Case et al., 2020). Those two explanations are not necessarily mutually exclusive. We use structural equation modeling to test these two explanations using large, nationally-representative samples of Americans in the mid-1990s and early 2010s.

According to the biopsychosocial model of pain, a person's perception and response to pain results from complex interactions among physiological, psychological, and social factors (Gatchel et al., 2007). As Zajacova et al. (2021b, p. 3) note, "social conditions shape the causes of chronic pain, its consequences, and even the very experience of pain." Socioeconomic status (SES) is likely to shape exposure to various stressors, including financial strain and work uncertainty. As proposed by McEwen (2004), prolonged exposure to stressors can suppress the immune system and contribute to atherosclerosis, obesity, bone mineral loss, and changes in brain neurons.

As shown in Fig. 1, we expect SES to have a direct relationship with perceived economic distress. In turn, economic stressors may contribute to obesity, morbidity, and the development or heightened perception of pain via their effects on the nervous, metabolic, and immune systems. The indirect effect of SES via economic distress to obesity to pain is depicted as pathway A→D→E in Fig. 1. The two other indirect effects are independent of one another: via economic distress (i.e., not mediated by obesity, pathway A→B) and via obesity (i.e., resulting from factors other than economic distress, pathway C→E).

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Earlier work demonstrated that perceived economic distress increased over recent decades among the most disadvantaged Americans, while there was little or no increase in distress for those with high SES (Glei et al., 2018). Similarly, longitudinal analyses showed that Americans with low SES experienced bigger increases in perceived financial strain and work uncertainty between 1995-96 and 2013-14 than their more advantaged counterparts (Glei et al., 2019). Therefore, we hypothesize that growing levels of economic distress, particularly among those with low SES, may be associated with widening SES disparities in pain.

Growing levels of obesity may also fuel rising pain (Institute of Medicine, 2011; Stokes et al., 2020; Zimmer and Zajacova, 2020). Evidence regarding recent trends in the socioeconomic disparity in obesity among US adults is mixed (Ljungvall and Zimmerman, 2012; Ogden et al., 2010; Yu, 2016), although Frederick et al. (2014) reports that obesity has risen faster among adolescents with less educated parents. There are two main mechanisms by which obesity is thought to increase pain: 1) structural damage from the mechanical stresses of excess weight on muscles, bones, and connective tissue (Okifuji and Hare, 2015), and 2) up-regulation of pro-inflammatory cytokines secreted by adipose tissue, such as leptin, which has been linked with osteoarthritis (Walsh et al., 2018; Yan et al., 2018).

It is also possible that weight-based discrimination exacerbates pain via psychosocial mechanisms. The experience of pain is shaped not only by physiological factors, but also by psychological and social influences (Gatchel et al., 2007). Carr and Tsenkova (2018) documented the psychosocial consequences of obesity in terms of discrimination, interpersonal relationships, and emotional well-being. Tomiyama et al. (2018) argued that stigma related to body weight can act as a chronic stressor that harms both physical and mental health. In particular, Ong et al. (2021) showed that exposure to everyday discrimination was associated with increased pain interference. The authors suggested that discrimination may amplify pain via biological processes (e.g., elevated levels of cortisol, inflammatory cytokines, and cardiometabolic markers) or by inhibiting the individual from seeking healthcare. And Sutin et al. (2016) found that perceived weight discrimination was associated with high prevalence of various types of pain (e.g., backaches, joint pain) even after controlling for body mass index. Obesity is also associated with depression and anxiety (Luppino et al., 2010), both of which are associated with pain. While our focus in this analysis is on the effects of economic distress and obesity on the rise in reported pain, these factors are likely to be important in its experience.

In this paper, we evaluate the extent to which subjective measures of economic distress account for widening SES disparities in the reported frequency of pain over recent decades. All types of pain may be influenced by stress exposure, but back and joint pain are likely to be related to obesity, aging, and osteoarthritis, whereas those factors are less likely to be associated with headaches. Therefore, we anticipate that economic distress will be more strongly associated with headaches than with back or joint pain. Finally, we assess whether the link between economic distress and pain is mediated, at least in part, by obesity.

2. Methods

2.1. Data

We used data from two cross-sectional waves of Midlife in the United States (MIDUS), each of which targeted a national probability sample of non-institutionalized, English-speaking adults aged 25–74 in the contiguous United States. In 1995–96, respondents were selected by random digit dialing with oversampling of older people and men (Brim et al., 2016); 3487 respondents completed the phone interview (70% response rate) and 3034 also completed mail-in self-administered questionnaires (SAQs). In 2011–14, a new refresher cohort was drawn from the national population using a sampling frame that included both landlines and cell phones (Palit et al., 2016a); 3577 individuals participated in the phone interview (59% response rate) and 2598 also completed the SAQ. This analysis is restricted to respondents who completed the SAQ (pooled analysis sample: $N = 5632$) because most of the measures used in this analysis come from the SAQ, including measures of pain, economic distress, obesity, income, and wealth.

The MIDUS survey protocols were reviewed and approved by the Education and Social/Behavioral Science Institutional Review Board at University of Wisconsin-Madison. Informed consent was obtained from all human subjects.

2.2. Measures

The key variables were generally measured in the same way in both survey waves unless otherwise specified.

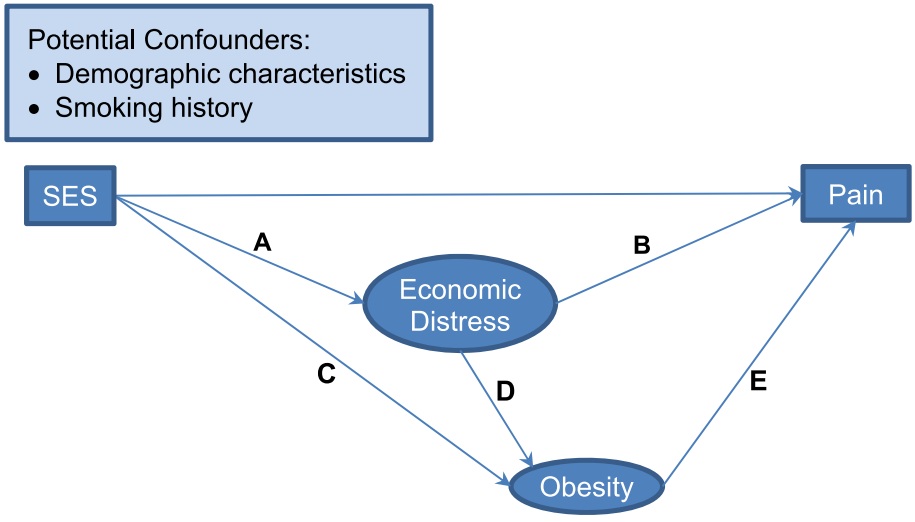

Fig. 1. Conceptual Framework for the Relationships Among SES, Economic Distress, Obesity, and Pain. For simplicity, we do not show the pathways from potential confounders (i.e., age, sex, race, smoking history), all of which are expected to be associated with SES, economic distress, obesity, and pain outcomes. Although not shown here, our model also includes a period effect (1995-96 vs. 2011-14 survey waves) and an interaction between period and SES, which allows the period change to differ by SES (e.g., did the SES disparity in pain widen over time?). The pathway A→B represents the indirect effect via economic distress alone. The pathway A→D→E represents the indirect effect via economic distress further mediated by obesity. The pathway C→E represents the indirect effect via obesity alone.

2.2.1. Reported frequency of pain

Respondents were asked how often, during the past 30 days, they experienced three types of pain: 1) headaches; 2) backaches (Note: In, 1995–96, the question specified "lower back aches", whereas in 2011-14, the question simply specified "backaches" more generally); and 3) aches or stiffness in joints. The six response categories for each of those three questions ranged from "not at all" to "almost every day."

2.2.2. Perceived economic distress

MIDUS includes eight measures related to subjective economic distress. The first question refers to intergenerational financial disadvantage: "When your parents were the age you are now, were they better off or worse off financially than you are now?" There were seven response categories ranging from "a lot better off" to "a lot worse off".

Four other questions pertain to the current financial strain:

1) Financial situation: "Using a scale from 0 to 10 where 0 means 'the worst possible financial situation' and 10 means 'the best possible financial situation,' how would you rate your financial situation these days?"
2) Financial control: "Using a 0 to 10 scale where 0 means 'no control at all' and 10 means 'very much control,' how would you rate the amount of control you have over your financial situation these days?"
3) Financial need: "In general, would you say you (and your family living with you) have more money than you need, just enough for your needs, or not enough to meet your needs?"
4) Financial difficulties: "How difficult is it for you (and your family) to pay your monthly bills?" [response categories: very difficult, somewhat difficult, not very difficult, not at all difficult]

Another question asked the respondent to rate his/her current work situation ("Please think of the work situation you are in now, whether part-time or full-time, paid or unpaid, at home or at a job. Using a scale from 0 to 10 where 0 means 'the worst possible work situation' and 10 means 'the best possible work situation,' how would you rate your work situation these days?").

The final two questions refer to the respondent's future expectations for their financial and work situations:

1) "Looking ahead ten years into the future, what do you expect your financial situation will be like at that time?" [same 0–10 scale as above]
2) "Looking ahead ten years into the future, what do you expect your work situation will be like at that time?" [same 0–10 scale as above]

We reverse-coded the items as needed so that higher values indicate more economic distress. Then, we standardized the items based on the weighted distribution of the pooled sample. Supplemental Digital Content Text 1 provides more detail regarding the sources of the items related to perceived economic distress.

2.2.3. Obesity

We have five measures related to obesity: current BMI, BMI one year prior to the survey, waist circumference, hip circumference, and a subjective question asking the respondent whether s/he considers him/herself overweight where the five response categories range from "very underweight" to "very overweight." BMI was computed based on self-reported height and weight (current and one year ago on a retrospective question). Respondents were also provided with a tape measure and asked to measure their own waist and hip circumference (Supplemental Digital Content Text 2 shows the instructions for self-measurement). We standardized the items based on the weighted pooled distribution.

2.2.4. Control variables

We controlled for age, sex, race (White, Black, Other),[1] smoking history, and a composite measure of relative socioeconomic status (SES), all of which may affect economic distress, obesity, and pain sensitivity. For example, the inverse association between smoking and obesity is well-established, and several studies describe mechanisms by which smoking can influence pain sensitivity (Aamodt et al., 2006; Ditre et al., 2011; Waldie et al., 2008). The direction of causation is less clear for the relationship between smoking and economic distress, but we suspect that distress exacerbates smoking. We also included a dichotomous variable for survey wave to test for a period effect. The SES index was based on education, occupation, income, and wealth, which we converted to a percentile rank representing the individual's position within the distribution at that survey wave (Supplemental Digital Content Text 3 describes how the measure of SES was constructed, and Table S1 provides descriptive statistics for the measures of SES). Supplemental Digital Content Table S2 provides descriptive statistics for other potential confounders.

2.3. Analytic strategy

We used standard practices of multiple imputation for missing data (Supplemental Digital Content Text 4 describes the implementation of multiple imputation). We used post-stratification weights (Brim et al., 2019; Palit et al., 2016b) for the descriptive analyses to ensure that the weighted samples show very similar distributions (in terms of age, sex, race, education and marital status) as the corresponding Current Population Survey.

Structural equation modeling (SEM) allows us incorporate latent variables and estimate the complex system of direct and indirect pathways depicted in Fig. 1. We used the "sem" command in Stata 16.1 (StataCorp, 2019) with the asymptotic distribution free (ADF) method that relaxes the assumption that the errors and latent variables are normally distributed. The regression models were fit using unweighted data because our models controlled for the key covariates that were associated with sample selection. This approach has been shown to yield unbiased and efficient estimates (Solon et al., 2015; Winship and Radbill, 1994).

2.3.1. Measurement models for economic distress and obesity

Our analysis included two latent variables: perceived economic distress and obesity. For economic distress, exploratory factor analysis indicated that all eight items loaded on one factor (eigenvalue = 3.2), although intergenerational disadvantage had a weaker loading (0.32) than the other 7 items (0.49–0.84). Using SEM, the measurement model for economic distress indicated that the two items referring to future expectations did not load well (standardized coefficients were 0.08 and −0.04 versus 0.28–0.90 for the other 6 items). Therefore, we dropped those two items from the model. The modification indexes, which indicate omitted pathways and covariances that are likely to improve model fit (Sörbom, 1989), also suggested there was correlation between the error terms for the questions about financial need and financial difficulties. Although we did not foresee it, that result is not surprising given the similarity of those two questions; to the extent that there is measurement error (e.g., perhaps related to willingness to report financial troubles), we would expect it to affect both of those variables. Allowing for correlation between these error terms slightly improved model fit (e.g., RMSEA = 0.047 vs. 0.049 without those correlations). Supplemental Digital Content Fig. S1 shows the final measurement model for economic distress.

For obesity, exploratory factor analysis indicated that all five items loaded well (≥0.68) on one factor (eigenvalue = 3.6). However, there

[1] We did not include Hispanic ethnicity because the 1995-96 wave of MIDUS did not ask respondents to report their ethnicity.

are well-known sex differences in waist and hip circumference that are independent of obesity. The results also suggested that the subjective evaluation of being overweight differs by sex. Auxiliary analyses showed that men were far less likely to report themselves as being "overweight" than women even after controlling for BMI. Thus, in the SEM, we allowed sex to have a direct influence on those three variables (i.e., waist circumference, hip circumference, subjective evaluation of overweight). In addition, the modification indexes suggested there were correlations between the error terms for current and previous BMI (both of which are a function of current height) as well as between waist and hip circumference (both of which are based on self-measurement). Again, allowing for correlation between these error terms slightly improved model fit (e.g., RMSEA = 0.047 vs. 0.049 without those correlations. Supplemental Digital Content Fig. S2 shows the final measurement model for obesity.

2.3.2. Structural models for economic distress, obesity, and pain

Our SEM regressed the reported frequency of each type of pain on SES, period (2011-14 relative to 1995–96), an interaction between SES and period (which tests whether the SES disparity widened over time), economic distress, and obesity controlling for age, sex, race, and smoking history as potential confounders. The modification indexes indicated correlation between the error terms for the three pain outcomes, which we did not anticipate. However, in retrospect, it seems likely that there are unobserved factors that affect an individual's pain sensitivity and his/her willingness to report pain. All three measures rely on self-report. Thus, any measurement error is likely to affect all three outcomes. Allowing for correlation between the error terms for the three pain outcomes notably improved model fit (e.g., RMSEA = 0.047 vs. 0.056 without those correlations).

The SEM also included equations predicting economic distress and obesity to estimate the mediating pathway via those latent variables. Economic distress was regressed on period, SES, the interaction between period and SES, and the same potential confounders. Obesity was regressed on those same variables plus economic distress, but we used a quadratic specification for age because prior evidence suggests a non-linear relationship between age and obesity (Yang et al., 2021).

Good model fit for a SEM is indicated by a root mean square error of approximation (RMSEA) less than 0.06, comparative fit index (CFI) and Tucker-Lewis index (TLI) values greater than 0.95, and a standardized root mean square residual (SRMR) less than 0.08 (Hu and Bentler, 1999). Our SEM yielded good fit based on the RMSEA (0.047, 90% CI 0.045–0.049) and SRMR (0.039), but CFI (0.827) and TLI (0.748) were subpar, perhaps because our model is complex with many variables, some of which are only weakly correlated with each other. Kenny (2020) pointed out that CFI and TLI depend on the average magnitude of the correlations in the data; if they are low, then CFI/TLI will not be very high. As more variables are added to the model, RMSEA tends to improve, but CFI and TLI generally decline (Kenny and McCoach, 2003).

Using the results from this final SEM, we calculated the predicted latent variables for economic distress and obesity so that we could examine the bivariate relationship between these variables and SES. We plotted the levels of perceived economic distress, obesity, and pain by relative SES for the two time periods (1995-96 vs. 2011–14). The "lpolyci" command in Stata 16.1 was used to perform local mean smoothing—also known as the Nadaraya-Watson estimator (Nadaraya, 1964; Watson, 1964)—across SES percentile for the two survey waves. A locally weighted average is computed for each point in the smoothing grid (in this case, each percentile of SES) using a kernel (in this case, Epanechnikov) as the weighting function.

We use the "nlcom" command in Stata 16.1 to compute the 95% confidence intervals for the total and indirect effects presented in Table 1.

Table 1
Total and indirect effects for the period effects at the (A) bottom and (B) top percentiles of socioeconomic status (SES) and (C) widening of the SES disparity over that period, unweighted analyses.

	Headaches		Backaches		Joint Pain/Stiffness	
	Coefficient (95% CI)	% of TE	Coefficient (95% CI)	% of TE	Coefficient (95% CI)	% of TE
A. Period effect at bottom percentile of SES						
Total effect (TE)	0.05 (−0.06, 0.17)		0.51 (0.38, 0.63)		0.55 (0.43, 0.66)	
Overall indirect effect (IE)	0.11 (0.07, 0.14)	N/A[d]	0.12 (0.09, 0.16)	24%	0.17 (0.14, 0.21)	31%
Individual IEs						
1. Via economic distress alone[a]	0.06 (0.04, 0.09)	N/A[d]	0.07 (0.04, 0.09)	13%	0.07 (0.05, 0.10)	13%
2. Via economic distress to obesity[b]	0.004 (0.002, 0.007)	N/A[d]	0.01 (0.00, 0.01)	1%	0.01 (0.006, 0.015)	2%
3. Via obesity alone[c]	0.04 (0.02, 0.06)	N/A[d]	0.05 (0.03, 0.07)	10%	0.09 (0.07, 0.11)	16%
B. Period effect at top percentile of SES						
TE	0.00 (−0.09, 0.09)		0.20 (0.11, 0.29)		0.27 (0.18, 0.36)	
Overall IE	−0.01 (−0.03, 0.01)	N/A[d]	−0.01 (−0.03, 0.01)	−4%	0.01 (−0.01, 0.03)	3%
Individual IEs						
1. Via economic distress alone[a]	−0.03 (−0.04, −0.01)	N/A[d]	−0.03 (−0.04, −0.01)	−14%	−0.03 (−0.04, −0.01)	−11%
2. Via economic distress to obesity[b]	0.00 (−0.003, 0.00)	N/A[d]	−0.002 (−0.003, −0.001)	−1%	−0.004 (−0.007, −0.002)	−2%
3. Via obesity alone[c]	0.02 (0.01, 0.03)	N/A[d]	0.02 (0.01, 0.03)	12%	0.04 (0.03, 0.06)	16%
C. Widening of the SES disparity						
TE	−0.05 (−0.23, 0.12)	N/A[d]	−0.31 (−0.49, −0.12)		−0.28 (−0.45, −0.11)	
Overall IE	−0.12 (−0.15, −0.08)	N/A[d]	−0.13 (−0.17, −0.09)	42%	−0.16 (−0.21, −0.12)	58%
Individual IEs						
1. Via economic distress alone[a]	−0.09 (−0.12, −0.06)	N/A[d]	−0.10 (−0.13, −0.06)	31%	−0.10 (−0.14, −0.07)	36%
2. Via economic distress to obesity[b]	−0.01 (−0.01, 0.00)	N/A[d]	−0.008 (−0.01, 0.00)	3%	−0.01 (−0.02, −0.01)	5%
3. Via obesity alone[c]	−0.02 (−0.03, 0.00)	N/A[d]	−0.03 (−0.04, −0.01)	8%	−0.05 (−0.08, −0.02)	17%

Notes. The coefficients represent the effects in terms of SD units of pain frequency. The total effect (TE) is the sum of the direct effect (DE) and the overall indirect effect (IE), which is the sum of the individual IEs. The DEs are presented in Table S4.

[a] The IE via economic distress alone is the product of DE (→economic distress) and the DE (economic distress→pain). For example, the IE via economic distress alone→backaches for someone in the bottom percentile of SES (0.07) is the product of DE for period→economic distress (0.405) and the DE of economic distress→backaches (0.168).

[b] The IE via economic distress to obesity is the product of DE (→economic distress), DE (economic distress→obesity), and DE (obesity→pain). For example, the IE via economic distress→obesity→backaches for someone in the bottom percentile of SES (0.01) is the product of DE for period→economic distress (0.405), the DE of economic distress→obesity (0.143), and the DE of obesity→backaches (0.099).

[c] The IE via obesity alone is the product of DE (→obesity) and DE (obesity→pain). For example, the IE via obesity alone→backaches for someone in the bottom percentile of SES (0.05) is the product of DE for period→obesity (0.497) and the DE of obesity→backaches (0.099).

[d] We did not compute the percentages when the TE was not significant.

3. Results

Fig. 2 shows the bivariate association between pain frequency and relative SES in 1995-96 versus 2011–14. There was little difference over time in the reported frequency of headaches, but the frequency of backaches and joint pain was higher in 2011-14 than in 1995-96, particularly at lower levels of the SES spectrum. For example, among those in the bottom quintile of SES, reported backache frequency was about half a SD higher in 2011-14 than in 1995-96, whereas the period difference was around one-fifth of a SD for those in the top quintile of SES (Fig. 2B). See also Table S3, Supplemental Digital Content, which provides means for the measures of pain, economic distress, and obesity by low versus high SES and survey wave.

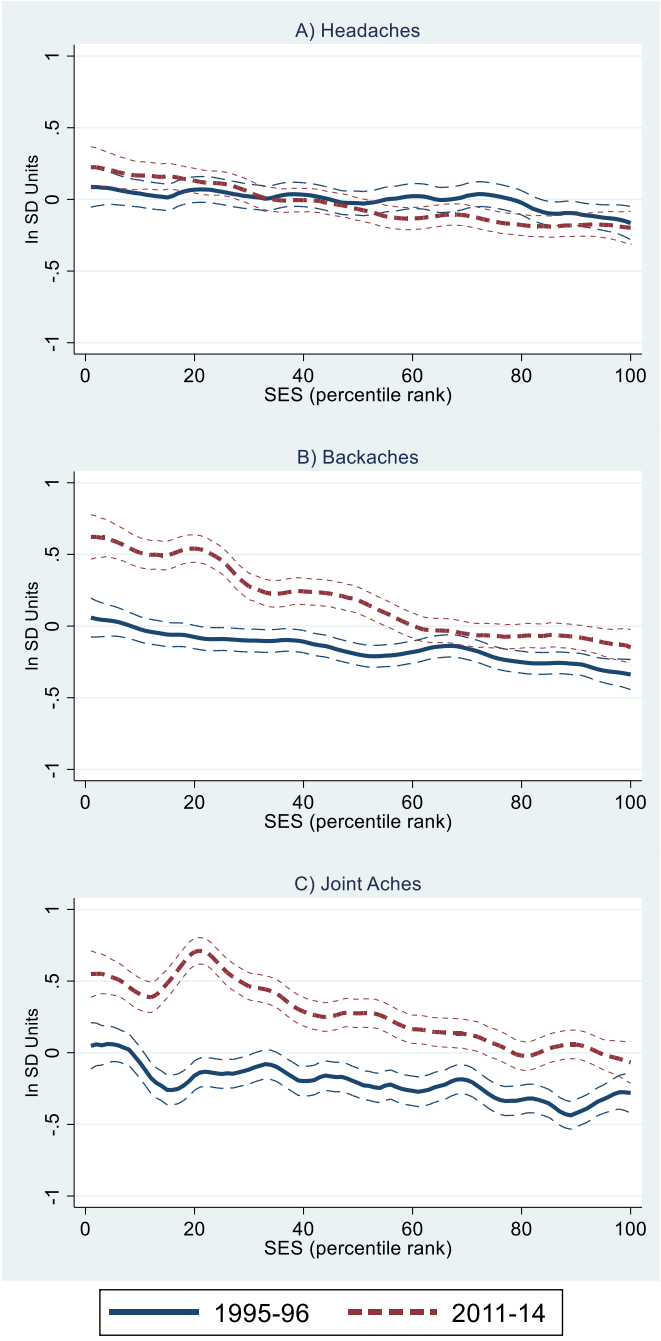

Fig. 2. Smoothed plots of pain measures by relative SES in 1995–96 vs. 2011–14: A) Headaches; B) Backaches; C) Joint pain, weighted analyses. Variables on the y-axis are scaled in terms of standard deviation units, where higher values indicate more frequent pain. For example, a value of zero on the outcome variable indicates that the smoothed mean for individuals at the specified percentile rank of SES is equal to the overall mean for the pooled sample (both waves combined), whereas a value of 0.5 would indicate a level half a standard deviation higher than the overall mean. The error bars represent the 95% confidence intervals.

Fig. 3 presents similar plots for the latent variables: perceived economic distress and obesity. For both of those variables, the period difference was much greater for those with low SES. Among those in the bottom quintile of SES, perceived economic distress was about half a SD higher in 2011-14 than in 1995-96 whereas, if anything, the results suggested an improvement in economic distress over this period for those in the top quintile of SES (Fig. 3A). In contrast, obesity appeared to have grown at all levels of SES (Fig. 3B), but much more for those in the bottom quintile of SES (i.e., more than half a SD higher in 2011-14 than in 1995-96) than the top quintile (i.e., less than one-fifth a SD).

Table 1 presents key results from the SEM, which controlled for demographic characteristics and smoking history (Supplemental Digital Content, Fig. S3 shows selected parameter estimates from the structural models, and Table S4 presents all the direct effects from the full model.).

The total effect (TE) represents the overall period change before adjusting for the changes over time in economic distress and obesity. These models confirm the results suggested by the bivariate plots: there was no significant period change for headaches, even at low levels of SES, but the frequency of backaches and joint pain was substantially

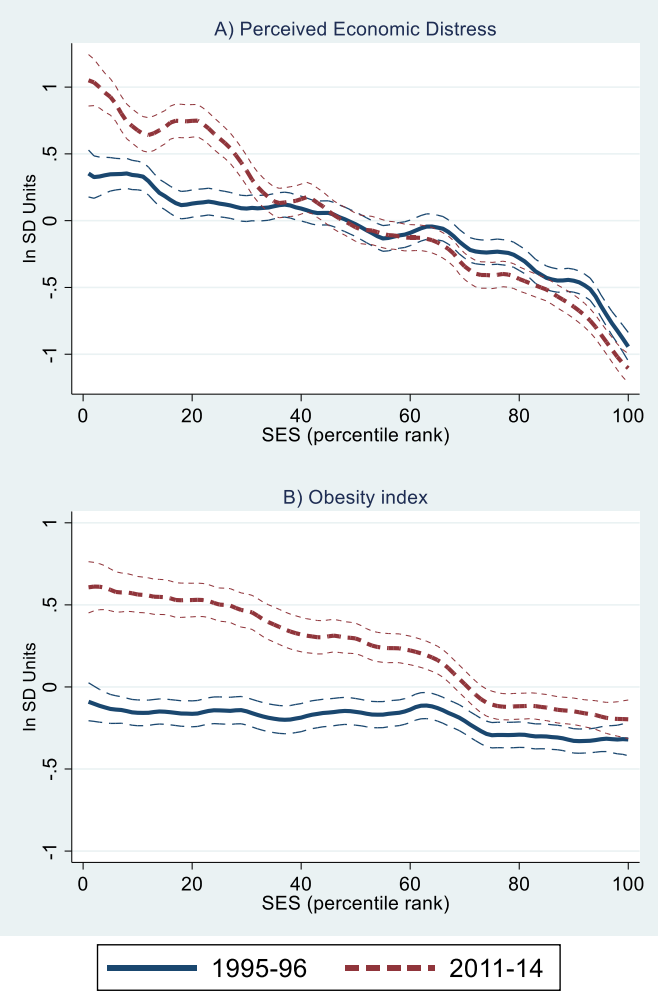

Fig. 3. Smoothed plots of A) perceived economic distress and B) obesity by relative SES in 1995–96 vs. 2011–14, weighted analyses. The error bars represent the 95% confidence intervals.

higher in 2011-14 than in 1995-96, especially for those with low SES (Table 1). For example, the TE for backaches was much larger for someone in the bottom percentile of SES (0.51 SD, Panel A) than for their counterpart in the top percentile of SES (0.20 SD, Panel B). The corresponding TEs for joint pain were 0.55 SD and 0.27 SD, respectively.

In Panel C of Table 1, the TE represents the overall widening of the SES disparity (a negative value indicates that the reduction in pain associated with higher levels of SES was larger in 2011-14 than it was in 1995-96). The SES disparity widened by 0.31 SD for backaches and by 0.28 SD for joint pain, but there was no significant SES widening for headaches.

Economic distress was associated with reports of more frequent pain (Table S4). Contrary to expectations, the coefficient for economic distress was similar for headaches ($\beta = 0.16$, 95% CI 0.12–0.20), backaches ($\beta = 0.17$, 95% CI 0.13–0.21), and joint pain ($\beta = 0.18$, 95% CI 0.14–0.21). Thus, we found no evidence to support the hypothesis that economic distress played a greater role for headaches than for backaches and joint pain.

The indirect effects (IEs) shown in Table 1 indicate the extent to which changes over time in economic distress and obesity account for the period differences in pain frequency. The overall IE is the sum of three individual IEs: 1) via economic distress alone (without being mediated by obesity; pathway A→B in Fig. 1); 2) via economic distress further mediated by obesity (pathway A→D→E in Figs. 1), and 3) via obesity alone (pathway C→E in Fig. 1).

We found little evidence that the relationship between economic distress and pain was mediated by obesity. The IEs via economic distress to obesity were small for both the period changes (Panels A & B) and for SES widening (Panel C). In contrast, there were much larger IEs via economic distress alone.

Combining the two pathways involving economic distress, the results suggest that economic distress contributed to rising pain among those with low SES (Panel A) but had the opposite effect for those with high SES (i.e., in Panel B, the negative IE implies that pain would have increased even more if not for a lessening of economic distress). As a result of these opposing effects at the two ends of the SES spectrum, economic distress accounted for a substantial share of the SES widening in backaches (34% including both pathways involving economic distress) and joint pain (41%, Table 1, Panel C).

In contrast, growing obesity contributed to the rise in pain at all levels of SES—for those with low SES (Table 1, Panel A) as well as for those with high SES (Panel B)—but the relationship was largely independent of economic distress. The IE via obesity alone was much larger than the IE via economic distress to obesity. In terms of the contribution to SES widening (Panel C), obesity played a smaller role than economic distress.

In sum, economic distress and obesity may explain why pain frequency increased more for those with low SES than those with high SES. Together, subjective measures of economic distress and obesity accounted for 42% of SES widening in backaches and 58% of SES widening in joint aches.

4. Discussion

Prior studies have documented huge SES disparities in pain among Americans (Zajacova et al., 2021b) and demonstrated that those disparities have widened in recent decades (Case et al., 2020; Glei and Weinstein, 2021; Zajacova et al., 2021a). Yet, there has been little attention to understanding the underlying factors that explain why the SES disparity widened over time. Our findings support the notion that widening SES disparities in pain may be associated with increasing economic distress among more disadvantaged Americans. Prior research also suggested that obesity contributed to widening SES disparities in pain (Glei et al., 2021), but the current results imply that growing economic distress may have played an even larger role in widening the SES disparity in pain. While obesity also may have contributed, its role appears to be largely independent of economic distress. The main reason economic distress accounted for more of the widening SES disparity in pain than obesity was because those with low SES suffered an increase in distress, whereas those with high SES benefited from a slight reduction. In contrast, obesity grew at all levels of SES, albeit more for those with low SES.

The biopsychosocial approach to pain (Gatchel, 2004; Gatchel et al., 2007) purports to include social factors, but there is little discussion of social context. As Zajacova et al. (2021b, p. 6) note, the model includes social psychological factors (e.g., social support, psychological distress), but ignores "the role of structural inequalities that shape who experiences pain, how they experience it, and how others view their pain." Case et al. (2020) appear to attribute the rise in pain to the erosion of working-class life, but it is not entirely clear whether they are proposing that deteriorating social and economic conditions "caused" the rise in pain or whether they view the rise in pain as a symptom of the malaise among working-class Americans. To our knowledge, this is the first study to explicitly investigate the contribution of economic distress to the rise in pain and the widening SES disparity in pain.

We expected obesity to be a primary path through which economic distress contributes to pain, but found little evidence to support that hypothesis. But if not obesity, then what is the mechanism? We speculate that economic distress may have other physiological consequences that influence pain perception. For example, as noted in the introduction, exposure to stressors can affect the immune system, contribute to atherosclerosis and bone loss, and modify brain function. It is also possible that economic distress induces illness that might cause both weight loss and pain.

We also anticipated that economic distress would have a bigger effect on headaches than on back or joint pain, but found the association to be similar for these three types of pain. Widening SES disparities in pain were evident for backaches and joint pain but not headaches, but the *indirect* effects via economic distress on SES widening of pain were nearly as large for headaches as for backaches and joint pain. In contrast, obesity was more strongly associated with joint pain than with backaches and headaches. Unsurprisingly, the indirect effects via obesity on SES widening of pain were largest for joint pain. Both of the two main mechanisms through which obesity is thought to increase joint pain (i.e., structural damage and inflammation) could contribute. Economic distress contributed to SES widening for all three types of pain, whereas obesity played a bigger role for joint pain than for backaches or headaches.

Although economic distress and obesity accounted for a substantial share of the widening SES disparities in pain, a sizeable portion remained unexplained. What else might have contributed? Link and Phelan (1995) view SES as a fundamental cause of health disparities because it affects access to a variety of resources that influence health and the intervening mechanisms can change over time. Thus, the economic distress portion of SES could be only part of the story. Case et al. (2020) mention social dislocation, which relates to their earlier writings about weakening connections with social institutions such as marriage/family, community, religion, and work (Case and Deaton, 2017). The loss of social structures can generate a sense of anomie (Durkheim, 1897) that may affect health, both physical and mental. The rise in pain may also be related to an increase in psychological distress, but the relationship between pain and mental health is bidirectional (Garland et al., 2013; Zajacova et al., 2021b). It would be difficult, if not impossible, to disentangle the causal effects. Furthermore, the opioid epidemic may have exacerbated reports of pain: prolonged opioid use can increase pain sensitivity (Ballantyne and Mao, 2003). Thus, the increase in pain could be both a cause and a consequence of the opioid epidemic (Case et al., 2020). Finally, we must consider the possibility that the increase in pain reflects a change in reporting or expectations for pain relief (Institute of Medicine, 2011; Zimmer and Zajacova, 2020), but it is not clear why changes in reporting practices would affect those with low SES more than their more advantaged counterparts (Case

et al., 2020; Glei et al., 2021).

Our model assumes that economic distress and obesity affect pain, but it is also possible that pain exacerbates obesity and/or economic distress. For example, pain (and obesity) could limit the ability to work, which may adversely affect financial resources and heighten economic distress. The costs of medical treatments for pain and obesity-related morbidity could also contribute to economic distress. If the widening SES disparity in economic distress is a consequence rather than a cause of the rise in pain, it begs the question: what caused the rise in pain?

4.1. Limitations

The main limitation of this study is potential endogeneity in the relationships among economic distress, obesity, and pain. With cross-sectional data, we cannot establish the direction of causation. If we had valid instruments for economic distress and obesity, then we might be able to estimate a non-recursive model that allows reciprocal relationships between a) economic distress and pain; b) economic distress and obesity; and c) obesity and pain. Unfortunately, we do not have defensible instruments for economic distress or obesity. However, as additional MIDUS rounds are completed, we should be able to at least identify the temporal ordering.

Another limitation is that the measures of obesity are self-reported. Comparisons of self-reported values versus anthropometric measurements among a subset of the MIDUS sample indicate that respondents tend to understate weight and overstate height, and thus BMI tends to be under-estimated (Glei et al., 2021). However, there was no evidence that reporting of BMI or waist circumference varied significantly by SES or frequency of pain (Glei et al., 2021). Thus, measurement error is unlikely to bias our estimates regarding the associations between SES, obesity, and pain.

Third, as with any survey, results are subject to selection biases (e.g., individuals who were younger, male, non-White, and less educated were less likely to complete the SAQ). Fourth, Wave 1 of MIDUS did not include a global measure of pain nor did the questions allow us to distinguish between acute and chronic pain, identify the duration and severity of pain, or determine the extent to which pain interferes with normal activities. Fifth, with only two cross-sectional waves, we can only evaluate period differences between the mid-1990s and early-2010s. More frequent cross-sectional survey waves throughout this period and beyond (to the early 2020s) would have enabled us to better delineate how closely the period trends in economic distress, obesity, and pain track one another. Finally, there were a lot of missing data for income and assets, and such data are often plagued by response errors, owing to lack of knowledge, poor recall, confusion among income categories, and deliberate omission because of sensitivity of the information (Moore et al., 2000).

4.2. Future research

The effects of obesity on health and well-being could stem from the psychosocial effects of discrimination against people who are obese as well as the physiological effects of obesity on the body. Although our model does not distinguish between those two mechanisms, our obesity measures include the respondent's subjective evaluation of their own weight status as well as more standard measurements (e.g., BMI, waist circumference). Future work could evaluate differences in the effects of the subjective evaluation versus the standard measurements, which might provide some clues about psychosocial versus physiological effects of obesity. Researchers could also incorporate measures of weight-based discrimination to help distinguish between these different effects. Our measure of obesity probably does not fully capture the repercussions of weight-based discrimination on pain, but we know that the perception of pain is influenced by psychological as well as physical stimuli. Unfortunately, a more detailed SEM analysis of the physical versus psychosocial components of obesity was beyond the scope of this study.

5. Conclusion

Socioeconomic disparities in backaches and joint pain widened substantially over recent decades, although there was no significant widening for headaches. The growing disparities in back and joint pain may be related to increased economic distress among disadvantaged Americans, and to a less degree, obesity. Growing obesity accompanied an increase in pain at all levels of SES, but made a small contribution to SES widening because obesity increased more for those with low than high SES. In the case of economic distress, an increase was limited to those with low levels of SES; at high SES, there was a slight reduction in economic distress. As Maestas (2020, p. 26560) notes, "If less educated, prime-age Americans are unable to work or are less productive at work because they are in pain, the nation's economic future is at stake … in case educated readers think they are immune to the personal, social, and economic tragedies that lie behind the pain gap, the interconnectedness of our economic lives signals that the plight of less educated Americans is the plight of us all." If SES disparities in pain continue to widen, it bodes poorly for the overall well-being of the US population, labor productivity, and the prospects for these cohorts as they reach older ages where pain may lead to physical limitation, which is a strong predictor of mortality (Glei et al., 2016; Goldman et al., 2016, 2017).

Declaration of competing interest

None.

Data availability

All the data used in this analysis are publicly available from ICPSR (Wave 1: https://www.icpsr.umich.edu/web/ICPSR/studies/2760; Refresher Wave: https://www.icpsr.umich.edu/web/ICPSR/studies/36532).

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2023.116399.

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