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Poverty, Hunger, Education, and Residential Status Impact Survival in HIV

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Abstract

Despite combination antiretroviral therapy (ART), HIV infected people have higher mortality than non-infected. Lower socioeconomic status (SES) predicts higher mortality in many chronic illnesses but data in people with HIV is limited. We evaluated 878 HIV infected individuals followed from 1995 to 2005. Cox proportional hazards for all-cause mortality were estimated for SES measures and other factors. Mixed effects analyses examined how SES impacts factors predicting death. The 200 who died were older, had lower CD4 counts, and higher viral loads (VL). Age, transmission category, education, albumin, CD4 counts, VL, hunger, and poverty predicted death in univariate analyses; age, CD4 counts, albumin, VL, and poverty in the multivariable model. Mixed models showed associations between (1) CD4 counts with education and hunger; (2) albumin with education, homelessness, and poverty; and (3) VL with education and hunger. SES contributes to mortality in HIV infected persons directly and indirectly, and should be a target of health policy in this population.

Keywords

HIV; Socioeconomic status; Mortality

Introduction

Low socioeconomic status (SES) has been associated with increased mortality across many diseases including cardiovascular disease, cancer, and other smoking related conditions [1–3]. After the introduction of highly active antiretroviral therapy (HAART) to industrialized countries in 1995–1996, there were dramatic reductions in mortality for those infected with HIV [4–6], but data on the association between SES and mortality has been inconsistent over the course of the epidemic.

Prior to the introduction of HAART some studies reported no difference in survival based on SES [7–10] and others reported increased mortality or disease progression associated with low SES [11,12]. During the HAART era, improved survival has been consistently linked with higher SES [9,10,13–17] but these studies are limited by the imprecise assessment of SES. For example, SES for an individual was established by taking neighborhood level [9,10,14–16] or county level [13] census derived SES data such as median income [10,13–16] or a combined “SES index” [9] and applying this to the participant's address at the time they entered the study population. In these studies, the timing of the census-block level SES data relative to the timing of enrollment and follow-up varied considerably. For example, in one of the previous studies [16], a subject who entered in 1998 and died in 2004 would have had SES approximated by median neighborhood income from 2001 census data, using the individual's 1998 address.

As acknowledged by previous authors the ecological assessment of SES has multiple possible limitations [9,14–16]. It is possible that area SES data may not reflect individual SES status; that area SES data obtained at the time of census may not accurately reflect area level SES data at the time of enrollment, and that if an individual's residence changes over time the address at enrollment may not accurately reflect SES over time.

The one study in the HAART era that did utilize an individual measure of SES undertook one measurement of SES at baseline and found an association between low SES and mortality [17]. This study was limited by having no biological marker of disease such as viral load data, or repeated measures of CD4 counts. Also, despite being performed in the HAART era, only 22.9% of the subjects were on HAART when the SES measurement was taken.

Therefore, we investigated whether SES predicts mortality in HIV infected persons in the HAART era using prospective and individually collected data while controlling for possible confounding factors. To further understand this relationship we examined whether SES impacted mortality directly or whether its impact was mediated through other known predictors of death.

Methods

Study Population

We examined data from 878 participants enrolled in the Nutrition for Healthy Living (NFHL) Study from 1995 to 2005. This is a prospective cohort study undertaken in the greater Boston and Providence area to investigate the effects of nutritional status on people infected with HIV. Eligible participants were HIV infected adults over 18 years of age. Pregnant women, people with diabetes, thyroid disease or malignancies other than Kaposi sarcoma, and those not fluent in English were excluded from the study. Mortality information was ascertained by quarterly review of the Massachusetts and Rhode Island Registries of Vital Records and Statistics and yearly searches of the National Death Index.

These registries were last reviewed in June 2008 to capture all participant deaths occurring through December 31, 2006, the date of censorship for this study.

Clinical and Laboratory Measurements

Participants were interviewed biannually to obtain information on clinical status, use of recreational drugs, sociodemographic information and use of antiretroviral therapy. HAART was defined as receipt of any of the following regimens: two protease inhibitors, one protease inhibitor and two nucleotide reverse-transcriptase inhibitors (NRTI), or one non-nucleotide reverse-transcriptase inhibitor, and two NRTIs. Individuals on triple NRTI regimens were classified as not being on HAART. Blood was collected at each visit for biochemical and immunologic testing; CD4⁺ cells were counted using flow cytometry, HIV RNA loads were measured by the Amplicor Monitor RT-PCR assay (Roche Molecular Systems) with a lower detection limit of 2.6 log₁₀ copies/ml (400 copies/ml) and albumin was measured by automated colorimetric dye-binding methods (Beckman Coulter) and reported in g/dL. Further details on data collection and subject selection for the NFHL cohort have been previously reported. [18,19].

Definition of SES and other Clinical Parameters

Poverty was defined as total household income below the federal poverty line, which was adjusted annually per the United States census bureau, or a personal annual income less than \$10,000 [20]. Homeless was defined as an individual not having a fixed and regular nighttime residence. This included living in a shelter or welfare hotel, or using public and private places not normally used for sleeping [21] or living in a boarding house. Hunger was defined using questions from the Radimer/Cornell scale that defined individual hunger [22]. The questions used for the current study are also consistent with the validation of the Radimer/Cornell measures performed by Kendall et al. [23]. To meet these criteria a subject had to report going without food due to lack of money plus either; losing weight due to lack of food, or the physical sensation of hunger due to lack of food. If individuals identified themselves as smokers of cigarettes then they were considered active smokers, and if they had used intravenous drugs in the prior 6 months they were identified as active IDU. Average daily alcohol consumption was assessed as part of a 3 days food record. Depression was assessed via an 8-item measure developed by Burnam et al., with scored responses being entered into an algorithm to establish a dichotomous depression variable as per the original study [24].

Statistical Analysis

Individual characteristics were compared based on survival status. Chi-square tests, Student's *t*-tests, and Wilcoxon rank-sum tests were used as appropriate.

Univariate Cox proportional hazards regression for all-cause mortality was used to estimate unadjusted hazard ratios for baseline variables. These were, SES measures (hunger, homelessness, poverty, and education), demographics, mode of HIV transmission, harmful behaviors (intravenous drug use [IDU], smoking, alcohol), viral factors (CD4 counts, HIV viral load), use of HAART, depression, and albumin. Information on demographics, education, and mode of HIV infection were only collected at baseline, unlike all other variables which were collected at each visit. Unadjusted hazard ratios were then estimated using the Andersen-Gill extension to the Cox hazards regression to account for covariates that may have changed over time. We also tested the relationship between SES and mortality, adjusting for potential confounders with a multivariable Cox regression, with all variables significant to the *P* < 0.2 level in univariate analyses being entered into the model. The time varying form of the variable was used, except for variables where only baseline information was collected. Variable selection was by stepwise elimination, with variables

not reaching statistical significance ($P < 0.05$) being deleted from the model. We ran additional analyses to assess whether there were mediating variables between SES and mortality. All variables present in the final multivariable Cox regression, excluding age and any measures of SES itself, were considered to be potential mediators, and were therefore selected as outcome variables for mixed model analyses. These mixed models examined whether baseline SES measures predicted change in the selected outcome. Investigators also confirmed there was a plausible association between SES and the selected outcome variables before proceeding with the mixed models. Also, multivariable Cox regression was performed that did not include outcome variables employed in mixed models, to examine the change in SES parameters between models with and without these potential mediators. For the mixed models, we assumed that missing data were missing at random (that is, unrelated to outcome), based on the typical reasons for missing visits, including difficulty obtaining transport, poor weather, intercurrent illness, forgetting, incarceration, and having to care for dependents. All analysis were conducted using SAS version 9.1 (SAS Institute, Cary, NC) with a 2-tailed P value 0.05 or less indicating a statistically significant association.

Results

Table 1 shows the baseline characteristics of the participants by survival status. There were 200 deaths in the cohort, giving a crude mortality rate of 23%, and median duration of follow up in those that died was 54.8 months (interquartile range 28.9–85.0 months). Mean age of the cohort was 40.2 ± 7.4 years. The participants who died were older (41.5 vs. 39.9 yrs, $P = 0.01$) at their baseline visit. The study population was 56% white and had the following HIV transmission categories: men who have sex with men only (MSM) 47%, only IDU 26%, heterosexual 21%, both MSM and IDU 3%, and transfusion related or undetermined 3%. Interestingly, there was no difference in gender or race by survival status. Gender, ethnicity, and HIV transmission category were all highly reflective of the HIV epidemic in Massachusetts and Rhode Island at the time of the study [25–27]. Individuals who smoked (28% vs. 17%, $P < 0.001$) or used intravenous drugs (40% vs. 21%, $P < 0.001$) had higher mortality rates than those who did not. Individuals who died had lower baseline median CD4 counts (193 vs. 369, $P < 0.001$), and albumin (3.9 vs. 4.1 g/dL, $P < 0.001$); higher HIV log₁₀ viral load (4.5 vs. 3.3, $P < 0.001$).

Unadjusted Cox proportional hazards analyses for baseline and time varying covariates along with the final multivariable analysis are presented in Table 2. There was significant evidence at the $P < 0.001$ level in both baseline and time varying univariate analyses that older age, higher HIV viral load, and lower CD4 counts, and albumin, were associated with increased likelihood of death. An individual's HIV transmission category was a predictor of death in the univariate analysis ($P < 0.001$). When using the category of heterosexual sex only as a referent group there was an increasing likelihood of death with the following categories; MSM only (HR 1.05), history of IDU and MSM (HR 1.26), history of IDU only (1.96) and the highest likelihood with a small group ($n = 20$) with either an undetermined or transfusion related transmission category (HR 3.71). The high hazard ratio for this last category likely reflects patients with conditions such as hemophilia who acquired their infection early in the course of the HIV epidemic and who were possibly co-infected with other blood borne viruses such as Hepatitis C. Consistent with the chi-square analyses in Table 1, active IDU use (HR 2.32, $P < 0.001$) and smoking (HR 1.81, $P < 0.001$) at the baseline visit were predictive of death. The time-varying smoking variable predicted death in the univariate analysis (HR 1.43, $P = 0.01$), unlike active IDU (HR 1.48, $P = 0.23$), but there was not significant evidence in the multivariable model for either of these harmful behavior variables predicting death. In univariate analyses HAART at baseline was not predictive of death (HR 0.89, $P = 0.45$), but as a time-varying variable, HAART did predict

a decreased risk of death (HR 0.73, $P = 0.03$). However, being on HAART did not remain in the final multivariable model. In the univariate analyses of baseline measures of SES, there was significant evidence that homelessness, education level and poverty predicted death, while univariate analysis of time-varying SES variables demonstrated significant evidence of poverty and hunger predicting death. The final multivariable model showed that older age (HR 1.06 per year) at baseline and increasing HIV viral load (HR 1.36 per $\log_{10}$ copies), and decreasing CD4 counts (HR 0.997 per count/ml), and albumin (HR 0.36 per g/dL); increased the likelihood of death (all $P < 0.001$). Notably, one of the SES measures, poverty, continued to significantly predict death (HR 1.50, $P = 0.03$) while controlling for age and the three time varying covariates known to predict mortality in HIV infected individuals [5,6,28–30].

To explore whether measures of SES had an indirect effect on mortality, CD4 counts, albumin, and HIV viral load, were examined as outcomes in a mixed model to see if the markers of SES predicted the baseline level, or change over time, in these strong predictors of death. Table 3 shows the SES measures at baseline that were significant to the $P \leq 0.05$ level in predicting baseline level and change in CD4 counts, albumin, or viral load in the mixed models. Parameter estimates generated by the mixed models and presented in this table are interpreted as follows. Estimates for the intercept indicate the degree to which presence of the risk factor (e.g., hunger) raises or lowers the baseline value of the outcome (e.g., CD4), and estimates for the slope indicate the degree to which the presence of the risk factor (e.g., college education) raises or lowers the change in the outcome per month (e.g., change in CD4 counts per month). In addition, Fig. 1 is a schematic diagram, using the example of CD4 counts, to visually aid the reader in interpreting the mixed model parameter estimates for intercept and slope.

For the mixed model outcome variable CD4, there was significant evidence that; for those who reported hunger at baseline the trajectory of their CD4 counts would be 89 cells lower at each time point compared to those who did not, and for individuals with a college education their CD4 counts would increase by 1.04 cells per month compared to those without this level of education. In regard to albumin, there was significant evidence for multiple measures of SES to predict change in the parameter estimate for the intercept. More specifically, for participants who met the criteria for homelessness or poverty at baseline, albumin measurements over time were 0.21 and 0.09 g/dL lower, respectively, than in those who did not, and for individuals with a college education, albumin was 0.10 g/dL higher. Furthermore there were no baseline measures of SES that significantly predicted change in g/dL of albumin per month, denoting no significant predictors of the slope for albumin. Lastly, for the outcome variable HIV viral load there was significant evidence that individuals who were hungry at baseline had an HIV viral load with a 0.22 $\log_{10}$ copies/ml higher trajectory, compared to those not hungry, and if college educated there was significant evidence the viral load would decrease at 0.005 $\log_{10}$ copies/ml per month compared to those without a college education.

The multivariable Cox regression model that excluded potential mediators (CD4, HIV viral load, albumin) selected age (HR 1.05), transmission category (no HR because defined as class variable), poverty (HR 1.6) and hunger (HR 1.7) as predicting mortality, all $P < 0.05$.

A graphical summary of the models used in the analysis is provided in Fig. 2 to illustrate SES directly predicting death, and also having an indirect impact via CD4 counts, albumin, and HIV viral load.

Discussion

This is the first study to examine the relationship between SES and mortality in HIV infected people in the HAART era using individual and repeated measures of SES. We found that SES had a direct impact on mortality in models that accounted for differences in HIV disease parameters and age. Participants who met the US government definition of poverty in the interval before death were found to be 1.5 times as likely to die. As well as this direct effect, markers of SES such as hunger, education, poverty and homelessness have an impact through factors found to be strong predictors of death in this cohort; CD4 counts, albumin, and HIV viral load. Importantly, these three parameters of HIV disease were also reported as predictors of death in other studies of HIV infected subjects [5,6,28–30], and are routinely used by clinicians for patient monitoring. The magnitude of the observed effects of SES variables was considerable. CD4 counts were 89 cells lower for individuals with hunger at baseline, and for a subject with a college education at baseline CD4 counts increased by one cell per month or 60 cells in 5 years. Conceivably, individuals with low SES are more likely to incur opportunistic infections or other complications of immunosuppression that accompany lower CD4 counts. We found that poverty and hunger independently predicted mortality in multivariable models without CD4, viral load, and albumin, but hunger dropped out of the model when these HIV parameters were added. Thus, part of the association between SES and mortality is mediated through its relationship with CD4 counts, viral load, and albumin.

Importantly, this is the first study in HIV infected individuals to show that SES is associated with impaired CD4, albumin, and viral load over time. These factors in turn strongly predict death. The findings from this study are consistent with other studies that low SES is associated with increased mortality in HIV infected individuals in the era of HAART [9,10,13–17], but differs by applying repeated individual assessments of SES and overcoming acknowledged limitations of ecological assessments [9,14–16]. This work also extends on data from the pre-HAART era where individual baseline measures of SES predicted clinical outcomes [11,12], but considers the influence of SES on mortality during an era when clinical outcomes have improved due to HAART [5,6]. Furthermore, this study is strengthened by having prospectively collected SES measurements allowing us to observe changes in SES over time. This is in comparison to studies using single ecological measures of SES [9,10,13–16] such as median personal income derived from neighborhood level census data collected every five [31] or ten [32,33] years, or single baseline measures of SES [11,12,17]. In addition, studies from the HAART era controlled for severity of disease by using single CD4 counts from the time of diagnosis of HIV [13], AIDS [9,14], when commencing HAART [15,16] or the lowest known value [17] unlike this study which used multiple CD4 measurements. The other benefit to this method of assessing SES is that clinical and SES measures were obtained repeatedly over time, enabling us to examine whether SES impacted mortality through clinical factors that were found to predict death.

The findings that SES directly predicts mortality while controlling for markers of HIV disease severity, and also predicts change in CD4 counts, viral load, and albumin over time are plausible. Individuals with low SES can have re-ordered priorities placing less importance on accessing or maintaining medical care, and adequate nutrition in an effort to deal with financial difficulties or unfulfilled food and shelter needs [34]. Their HIV management or nutritional status may subsequently suffer leading to lower CD4 or albumin, or higher viral load measurements, all three of which are known markers of disease progression and mortality in people with HIV infection [6,29,30,35,36]. Albumin is also considered a marker of chronic illness and nutritional status [37]. Interestingly, markers of low SES such as poverty were found to predict mortality with greater statistical significance than use of HAART in unadjusted and adjusted analyses. This could be explained by

individuals with low SES not accessing HIV care, or even if on HAART not being able to regularly maintain therapy, contributing to increased mortality from HIV associated conditions. Additionally, associations between low SES, mental illness and drug use [38] may lead to increased non-HIV related mortality, regardless of HAART use.

Along with the associations reported in this study of HIV positive subjects, seroprevalence studies conducted in North America have shown higher rates of HIV infection in areas of low SES [39–41]. A North American HIV epidemic more focused in areas of low SES is particularly concerning when combined with findings from this and other studies that SES predicts mortality in HIV positive patients. Additionally these findings may go some way to explaining the unexpected mortality still evident in people infected with HIV who are on effective HAART therapy [42,43]. Thus, interventions aimed at improving SES could have a considerable impact on improving mortality in people infected with HIV. These interventions may take the form of food or transportation vouchers for individuals who fit criteria for markers of low SES such as poverty, employing social workers or case managers to assist patients in obtaining available government subsidies or support to maintain medical and social service appointments. Other strategies to alleviate poor outcomes of low SES include shelter based interventions for those without permanent housing, and harm reduction strategies for individuals taking part in unsafe sex or drug use practices.

A limitation of this study is that it may not be generalizable to all settings due to regional and national differences in the epidemiology of HIV and the way which health care is provided. It should be noted, however, that government funding was available for HIV positive persons in Massachusetts and Rhode Island during the course of the study who did not have medical insurance to receive medical care, ART and other services throughout the duration of the study [44]. Despite this financial support, individuals with low SES may still have difficulty accessing care in this setting or settings of universal health care [45]. Also, we did not use a cause specific mortality measure which may have better explained factors influencing death in different sub-groups. That said, all cause mortality is an accepted outcome measure in a range of clinical fields [46], including HIV clinical research [47], and is not affected by bias associated with incorrect assignment of the cause of death [46,48]. Another possible limitation is that of recall bias to establish total household income for the poverty covariate in this study, although any misclassification from this self report technique is likely to be considerably less compared with other ecological measures of household income such as neighborhood level census derived income data.

Conclusions

Our data suggest that HIV infected individuals with attributes of low SES are more likely to have increased mortality than those who are not living under these adverse conditions. Our data also indicate that SES factors increase the risk of death through adverse effects on other strong predictors of mortality such as CD4 counts, albumin, and HIV viral load. Despite HAART therapy, HIV infected individuals who are poor, homeless, hungry, or have less education, continue to have a higher risk of death. These findings highlight the importance of being aware of and addressing these issues in our patients as we work to maximize chronic HIV care.

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Fig. 1.
Schematic representation of differences in trajectory of CD4 over time as predicted by SES measures

Fig. 2.

Model summary—We studied whether SES and other factors directly predicted death via Cox proportional hazards regression. Mixed models were used to see if SES predicted change in the strongest predictors of death from the Cox models (CD4, albumin, and HIV viral load)

Table 1

NFHL cohort characteristics at baseline

	Dead 200 (22.8)	Alive 678 (77.2)	<i>P</i>-value^a
Mean age in years	41.5 ± 7.8	39.9 ± 7.3	0.01
Gender			0.64
Female	60 (23.8)	192 (76.2)	
Male	140 (22.4)	486 (77.6)	
Race			0.58
White	115 (23.5)	375 (76.5)	
Non white	85 (21.9)	303 (78.1)	
HIV transmission category			<0.001
Heterosexual	34 (18.3)	152 (82.7)	
MSM only	85 (20.4)	331 (79.6)	
MSM and IDU	6 (22.2)	21 (77.8)	
IDU only	67 (29.3)	162 (70.7)	
Other ^b	12 (60.0)	8 (40.0)	
Smoker			<0.001
Active	124 (27.4)	329 (72.6)	
Not Active	69 (17.0)	337 (83.0)	
IDU			<0.001
Active	26 (40.0)	39 (60.0)	
Not Active	169 (21.1)	632 (78.9)	
HAART			0.04
On HAART	79 (19.4)	329 (80.6)	
Not on HAART	102 (25.5)	298 (74.5)	
CD4, counts/ml	193 (65–364)	369 (216–560)	<0.001
Log ₁₀ HIV viral load, copies/ml	4.5 (3.3–5.1)	3.3 (ND ^c –4.3)	<0.001
Serum albumin, g/dL	3.9 ± 0.6	4.1 ± 0.4	<0.001

Note: Sample size varied based on missing data but no more than 3% data missing, except for serum albumin (*n* = 754) and HAART use (*n* = 808). Values represent *n* (% with that characteristic), median (Q1–Q3) or mean ± SD

^a *P*-values chi-square (categorical), Student *t*-test (continuous normal distribution)

^b Transfusion related or indeterminate

^c ND Not Detectable. Lower limit of detection 2.6 log₁₀ (400) copies/ml

Table 2

Unadjusted and Multivariable Cox proportional hazards of mortality

	Unadjusted Hazard ratio for baseline data	P-value	Unadjusted Hazard ratio for time varying data	P-value	Multivariable model Hazard ratio	P-value
Age—increase by 1 year	1.03	0.001			1.06	<0.001
HIV transmission category		<0.001			—	
Heterosexual only	1.0					
MSM only	1.05					
IDU and MSM	1.26					
IDU only	1.96					
Other ^a	3.71					
Active IDU	2.32	<0.001	1.48	0.23	—	
Active Smoker	1.81	<0.001	1.43	0.01	—	
CD4 count—increase by 1 cell/ μ l	0.997	<0.001	0.996	<0.001	0.997 ^b	<0.001
HIV Viral Load—increase by 1 log ₁₀ copies/ml	1.64	<0.001	1.78	<0.001	1.36 ^b	<0.001
Albumin—increase by 1 g/dL	0.38	<0.001	0.26	<0.001	0.36 ^b	<0.001
On HAART	.89	0.45	0.73	0.03	—	
Hunger ^c	1.07	0.81	2.13	<0.001	—	
Homeless ^d	1.78	0.004	1.39	0.12	—	
Poverty ^e	1.62	0.001	1.68	<0.001	1.50	0.03
College education	.68	0.02			—	

Note: Gender, race, alcohol, and depression were not significant to the $P < 0.05$ level in unadjusted analyses^aTransfusion related or indeterminate^bTime-varying^cGoing without food due to lack of money, plus weight loss and/or hunger pangs due to lack of food^dNot having a fixed and regular nighttime residence^eTotal household income below federal poverty line or personal annual income < \$10,000

Table 3

Mixed effects model of CD4, albumin, and viral load over time (months) as predicted by SES

Dependent variable (strong predictor of mortality)	Independent variable (SES measure)	Parameter estimate	P-value
CD4 count (cells/ μ l)			
Intercept	Baseline Hunger ^a	-89.4	0.01
Slope	College educated ^b	1.04	0.01
Albumin (g/dL)			
Intercept	Baseline Homeless ^c	-0.21	<0.001
	Baseline Poverty ^d	-0.09	0.01
	College educated ^b	0.10	0.01
Slope	—		
Viral Load (log ₁₀ copies/ml)			
Intercept	Baseline Hunger ^a	0.22	0.05
Slope	College educated ^b	0.005	0.01

^a Going without food due to lack of money, plus weight loss and/or hunger pangs due to lack of food^b Have at least a college education^c Not having a fixed and regular nighttime residence^d A household below federal poverty line or personal annual income < \$10,000